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CHARLES HILTON FAGGE, M.D., F.R.C.P.

SOMETIME PHYSICIAN TO GUY'S HOSPITAL

COMPLETED AFTER HIS DEATH AND SINCE REVISED OR RE-WRITTEN BY

PHILIP HENRY PYE-SMITH, M.D., F.R.S.

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS; CONSULTING PHYSICIAN TO GUY'S HOSPITAL

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CONTENTS OF THE SECOND VOLUME

	PAGES
PNEUMONIA AND INFLAMMATION OF THE LUNGS	
PNEUMONIA.—Definition and history—Symptoms and clinical course—complications—mode of death—convalescence—Physical signs—Morbid anatomy and histology: stages, locality, events—Ætiology and distribution—Pathology—infective, epidemic, and bacillary pneumonia—Diagnosis—Prognosis—Treatment.	
Secondary pneumonia—Hypostatic pneumonia—Acute pulmonary congestion—Chronic lobar pneumonia.	
BRONCHO-PNEUMONIA, or Pulmonary Catarrh.—Causes in children and in adults—Symptoms and course—Anatomy—Diagnosis—Prognosis and Treatment—Suppurative pneumonia.	
CHRONIC INTERSTITIAL PNEUMONIA, or Cirrhosis of the Lung.—Anatomy—Origin—from the pleura—from the bronchi—Symptoms—Treatment.	
SYPHILITIC INFLAMMATION OF THE LUNGS.—Cases—Diagnosis—Anatomy—Acquired and hereditary forms.	
GANGRENOUS INFLAMMATION OF THE LUNGS.—Anatomy—Origin—Fœtor and other symptoms—Complications and signs—Treatment	1—36
PHTHISIS	
History and definition—unity of phthisis—phthisis always tuberculous—Anatomy—localisation in the apices—Histology: miliary, caseous and infiltrating tubercle—Tuberculous pneumonia and ulceration—vomicae—adhesions—contractions—involution.	
Symptoms: wasting, pallor, pyrexia, cough, sputum—hæmoptysis—phthisis ab hæmoptoe—Physical signs of the three stages and of involution—Diagnosis—Course and duration—mode of death—recovery—Prognosis.	
Phthisis in children—Senile phthisis.	
Ætiology—contagion—hereditary disposition—diathesis and conformation—overcrowding, etc.—Inhalation of dust—pneumoconiosis—damp soil—Infected dwellings—Antagonistic conditions—Age and sex—Distribution.	
Treatment: preventive, curative, and palliative—diet and hygiene—voyages—climate—sanatoria—drugs—Treatment of complications.	
Acute Pulmonary Tuberculosis—Distinctive characters—Anatomy—Physical signs—Clinical symptoms and course—Diagnosis—Ætiology—Prognosis	37—94
ASTHMA	
History—Description of an attack—Diagnosis and relation to structural lesions of the lung—Ætiology—Nature and physiology of the disease—Prognosis and treatment.	
HAY-FEVER.—Onset and course—Ætiology—distribution among persons and places—treatment	95—104

AFFECTIONS OF THE LARYNX

The laryngoscope and its use.

LARYNGEAL PARALYSIS.—Of one or both recurrent nerves—Of the abductor muscles alone—Of the adductors—Aphonia and other disorders of the voice.

LARYNGISMUS STRIDULUS.—Nomenclature—Symptoms and Diagnosis—Pathology—Ætiology—Prognosis and treatment—Spasm of the glottis in adults.

LARYNGITIS.—Acute catarrh of the larynx—Plastic laryngitis—Croup—Chronic catarrh of the larynx—Tuberculous laryngitis—Lupus—Syphilis and leprosy of the larynx—Œdematous submucous laryngitis—Perichondritis.

TUMOURS OF THE LARYNX.—Papilloma—Polypus, etc.—Sarcoma and Carcinoma—Laryngeal malformations. 105—141

OBSTRUCTION OF THE TRACHEA AND BRONCHI

MEDIASTINAL AFFECTIONS, PULMONARY GROWTHS, AND EMBOLISM

Pressure on the trachea from enlarged thyroid, from aneurysm, mediastinal abscess or growth, from enlarged lymph-glands, cancer of the œsophagus, or dilated auricle—Stricture of the trachea—Impaction of trachea or bronchus by foreign bodies—Symptoms of tracheal obstruction—Sequelæ—Diagnosis, duration and treatment.

Inflammation of the mediastinum, acute and chronic.

Sarcoma and cancer of the mediastinum and root of the lung.

New growths in the pulmonary tissues, primary and secondary.

Pulmonary embolism—its causes, symptoms and results 142—162

DISEASES OF THE HEART

FUNCTIONAL DISORDERS

History and general pathology of cardiac diseases—their arrangement—physiological conditions—The pulse—The sphygmograph and other instruments—Bradycardia—Tachycardia and irritable heart—Intermittent and irregular pulse—Allorhythmic pulse—Palpitation—Cardialgia—Treatment of the above conditions—Tension of the pulse—its indications—Asphyxia and syncope—its distinction from epilepsy, collapse and sunstroke—its treatment 163—182

ANGINA PECTORIS

Heberden's original account of the disorder—Onset and symptoms—Recurrence and event—Anatomy—Pathology—Angina vasomotoria—Pseudo-angina—Secondary angina—Cardialgia—Ætiology—Age and sex—Cases—Prognosis—Treatment 183—193

AFFECTIONS OF THE MYOCARDIUM

HYPERTROPHY AND DILATATION.—History—Physiology of the process—its estimation—its anatomy—diagnosis—physical signs—Ætiology—valvular lesions and pericarditis—renal disease and arterial sclerosis—emphysema—exercise and strain—tobacco and beer—Dilatation from specific fevers, from anæmia, and directly from rheumatism—Symptoms—Treatment.

FIBROUS DEGENERATION.—Chronic myocarditis—Anatomy—Origin and pathology—Thrombosis—Syphilis—Cardiac aneurysm—Symptoms—Course and event.	
Acute myocarditis—Its origin, anatomy, and results.	
FATTY DEGENERATION.—Fatty overgrowth and infiltration—Intra-fibrous fatty metamorphosis: its histology, causes, and symptoms.	
Granular or pigmentary, and calcareous degenerations—Rupture of the heart	194—218

PERICARDITIS

Rarity of idiopathic pericarditis—its usual antecedents—Anatomy—fibrinous, serous, hæmorrhagic, and purulent effusion—Physical signs—Symptoms—Diagnosis—Prognosis and events—Adherent pericardium: its effects: its recognition—Hypertrophic pericarditis and pulsus paradoxus—Milk-spots on the heart—Hydropericardium—Hæmopericardium—Treatment of pericarditis and of pericardial effusion—Paracentesis	219—230
--	---------

ENDOCARDITIS AND VALVULAR DISEASE

ENDOCARDITIS.—The Subacute, Simple, or Benign form—Its relation to rheumatism and its ætiology generally—Its anatomy—Its signs.	
The Chronic or Sclerotic form—Its origin and anatomy—Its effects.	
The Ulcerative, Septic, or Malign form—Its origin, anatomy, and results—Its relation to pyæmia and other infective processes—Prognosis—Treatment.	
VALVULAR LESIONS.—Their origin and anatomy—Effects on the heart itself and on other organs—cardiac dropsy—dyspnœa and orthopnœa—hæmoptysis and other symptoms—Physical signs: cardiac murmurs—their rhythm—their locality and conduction—their physical cause.	
Aortic or sigmoid stenosis—Aortic regurgitation—Mitral stenosis—Mitral regurgitation—The anatomy, physical signs, and symptoms of each.	
Lesions of the pulmonary or dextrosigmoid valve and of the tricuspid—Functional murmurs—Combined lesions.	
Relative frequency of valvular lesions—Their general prognosis—Special prognosis as to duration and suddenly fatal termination.	
Treatment of organic diseases of the heart, in general and in particular.	
Congenital cardiac lesions—Disease of the heart in childhood and in old age	231—279

ANEURYSM OF THE AORTA

Aneurysm—Definition of the term—Varieties—Anatomy—Pathology—Origin in strain, in syphilis, and in embolism—Symptoms of two kinds.	
Aneurysm of the ascending aorta—of the arch—of the descending aorta—Diagnosis—event—prognosis—treatment.	
Abdominal aneurysms—Symptoms—origin—diagnosis—treatment.	
Dissecting aneurysms—Statistics of aortic aneurysm	280—295

DISEASES OF THE ALIMENTARY TRACT

AFFECTIONS OF THE MOUTH, THE THROAT, AND THE GULLET

PAGES

Affections of the mouth—Vesicular ulcers of the tongue and lips—Ulcer of the palate—Phagedænic ulcerative stomatitis—Gangrenous stomatitis or cancerum oris—Thrush—Aphthæ—Ptyalism—Xerostomia.

Affections of the fauces and pharynx—Sore throat: catarrhal, ulcerative, and herpetic inflammation—Subacute follicular tonsillitis—Acute tonsillitis or quinsy—Hypertrophy of the tonsils—Granular pharyngitis—Adenoid growths of the naso-pharynx.

Diseases of the œsophagus—Minor disorders—Spasmodic stricture—Simple stricture—Cancerous stricture—its anatomy, symptoms, and event 296—312

DISEASES OF THE STOMACH

Acute catarrhal gastritis—its symptoms, causes, histology, and treatment—Acute paralytic distension—Acute suppurative gastritis.

Atonic dyspepsia and chronic catarrhal gastritis—Symptoms—Anatomy—Causes—Diagnosis and prognosis—Treatment—clinical varieties—drugs and diet—Chronic gastric paresis with dilatation.

Gastralgia—Anorexia—Bulimia—Pica—Flatulence—Eructation and regurgitation—Pyrosis—Vomiting—Hiccough—Hæmatemesis.

GASTRIC ULCER.—Anatomy—Pathology—Ætiology—Symptoms and diagnosis—Latency—Event and duration—Treatment—Duodenal ulcer.

CANCER OF THE STOMACH.—Carcinoma of the pylorus and of the body of the stomach—Sarcoma—Colloid cancer—Other occasional tumours—Symptoms—the tumour—consequent dilatation—Gastro-colic fistula—Diagnosis—Duration—Treatment, palliative and by operation.

Fibrous induration of the stomach—Gastric concretions . 313—365

FUNCTIONAL AND INFLAMMATORY DISEASES OF THE INTESTINES

COLIC.—Onset—causes—diagnosis—prognosis and treatment—Lead-colic—history of its recognition—modes of infection—diagnostic characters—the “blue line”—pathology and treatment—other effects of lead.

CONSTIPATION.—Causes and pathology—effects—treatment—general, and of certain clinical varieties.

DIARRHŒA.—Acute and epidemic form—symptoms—treatment—chronic forms—Tuberculous enteritis—Diarrhœa from lardaceous and malignant disease—Psilosis—Enteritis.

Acute catarrhal colitis—Ulcerative colitis—Enteritis—Inflammation of the small intestines—Membranous colitis—Dilatation of the colon—Intestinal casts.

DYSENTERY.—History—Symptoms—Course—Anatomy—Catarrhal and diphtheritic forms—Sporadic and epidemic dysentery—Ætiology—Diagnosis and event—Prophylaxis—Treatment of acute and of chronic dysentery 366—401

TYPHLITIS

History and name—Frequency—Anatomy—Symptoms and clinical course—of mild cases—recurrent—suppurative with adhesions—sloughing with septic peritonitis—Diagnosis—Sex and age—Pathology—Prognosis, and treatment—Cases 402—408

INTESTINAL OBSTRUCTION

Introduction—classification—frequency and incidence on age and sex of the several kinds of obstruction.

IMPACTION.—Gall-stones—Concretions, etc.—Fæcal masses in the rectum.

COMPRESSION of the gut from without by tumours or other causes.

CONTRACTION AND STRICTURE.—Cicatrices and constrictions—Adhesions and contractions affecting the ileum—Simple and malignant stricture of the colon and rectum—its locality and anatomical structure.

INVAGINATION.—Anatomy—Obstruction and subsequent strangulation—Ætiology and pathology—Symptoms: pain, tumour, hæmorrhage—course—duration of first and second stages—Diagnosis—Prognosis—Treatment.

STRANGULATION.—Bands—Apertures in omentum or mesentery, etc.—Internal hernia—Volvulus—its seats, mechanism, and effects.

Anatomical results of obstruction and strangulation from whatever cause—Origin—General symptoms: constipation, pain, vomiting—Special symptoms and course of chronic obstruction—Special symptoms of acute obstruction—Diagnosis—Prognosis—Treatment: expectant—by colotomy—by abdominal section—by other mechanical methods—Summary—Cases 409—445

WORMS (CESTOID AND NEMATOID ENTOZOA)

Tapeworms—Anatomy and transformations—*Tænia solium*—its distinctive characters and distribution—its relation to *Cysticercus cellulosæ* of pork—*Tænia mediocanellata*—its specific distinctions—its relation to the *cysticercus* of beef—its geographical distribution—*Bothriocephalus latus*—its characters and distribution—its probable origin—Symptoms of the presence of tapeworms generally—Preventive and curative treatment.

Bladderworms—Relation of cystic or hydatid forms to the strobila form—Hydatids infesting the human body: *echinococcus*.

Round-worms—*Ascaris lumbricoides*—its anatomy—natural history—distribution—symptoms, diagnosis, and treatment—Threadworms—anatomy—symptoms and treatment—*Trichocephalus dispar*—*Eustrongylus gigas*.

Sclerostomum duodenale—its discovery—anatomy—habits—Anæmia caused by its presence—Treatment.

Trichina spiralis—its discovery—anatomy, transmigrations, and encapsulation—Trichiniasis—its symptoms, diagnosis, treatment, and prevention.

Filaria sanguinis hominis—its discovery in the blood—its transmigrations—its relations to chyluria and elephantiasis.

Dracunculus—*Distomum*—*Bilharzia hæmatobia*—its habitat—relation to hæmaturia—*Echinorhynchus* 446—476

DISEASES OF THE PERITONEUM

ACUTE SEPTIC PERITONITIS.—Clinical symptoms—Origin: secondary to visceral disease—bacillary—puerperal—uræmic—extreme rarity of idiopathic acute peritonitis—Morbid anatomy—Local and circumscribed peritonitis—Subphrenic suppuration—Diagnosis—Treatment: medical and operative.

CHRONIC PERITONITIS.—Peritoneal adhesions—Thickening, general and local—liver, spleen, omentum—Locular and general effusion—Frequency—Causes.

TUBERCLE OF THE PERITONEUM.—Anatomy—Symptoms—Age and sex—Diagnosis—Prognosis—Treatment.

CANCER OF THE PERITONEUM.—Commonly secondary—Anatomy—Symptoms.	
ASCITES.—Physical signs—diagnosis—Chylous ascites—inflammatory and obstructive ascites—Prognosis—Treatment by drugs and by paracentesis	477—500

DISEASES OF THE LIVER

JAUNDICE AND GALL-STONES

So-called bilious or hepatic dyspepsia—disturbance of secretion or elimination of bile—bilious symptoms—treatment by diet, drugs, and exercise.	
ICTERUS.—Symptoms—tests for bile-pigment and for bile acids in the urine.	
Idiopathic jaundice: its course, pathology, and prognosis—Pruritus—Slow pulse.	
Symptomatic jaundice—cardiac, febrile, toxic, pneumonic, pyæmic—from portal sepsis, suppurating hydatid, and acute tubercle—Jaundice from cirrhosis.	
Epidemic jaundice—Weil's disease—other causes of jaundice.	
Effects of permanent jaundice on the liver and gall-bladder—fatty stools, etc.—Pathological theories of jaundice.	
Treatment of jaundice, primary and symptomatic—of permanent jaundice.	
GALL-STONES.—Their structure and varieties—Symptoms and events of biliary colic—treatment—Suppuration of the ducts and gall-bladder	501—523

INFLAMMATORY DISEASES OF THE LIVER

ACUTE SUPPURATIVE HEPATITIS—ABSCESS OF THE LIVER.—Geographical distribution—Ætiology—Relation to dysenteric ulceration—Statistics—Morbidity—Anatomy—Characters of the pus—Direction of rupture—Symptoms—Physical signs—Diagnosis from acute perihepatitis—from abscess between the liver and diaphragm—from suppurating hydatid or gall-bladder—Prognosis—Treatment of acute hepatitis—Paracentesis of hepatic abscess.	
CHRONIC INTERSTITIAL HEPATITIS—CIRRHOSIS OF THE LIVER.—History—Anatomy—Ætiology—Non-alcoholic cirrhosis in children—Effects upon the portal circulation—Early symptoms—Hæmorrhage—Jaundice—Ascites—Cerebral symptoms—Hypertrophic cirrhosis—Prognosis and treatment.	
Perihepatitis—Hypertrophy of the liver—Simple chronic atrophy—Syphilitic hepatitis and gummata of the liver.	
ACUTE ATROPHIC HEPATITIS—ACUTE YELLOW ATROPHY OF THE LIVER.—History—Symptoms—jaundice—diminished percussion-dulness—the urine—cerebral symptoms—hæmorrhage—Course and event—Anatomy—Ætiology and pathology—Diagnosis—Prognosis—Statistics—Treatment	524—550

NEW GROWTHS AND DEGENERATIONS OF THE LIVER

Carcinoma hepatis—its anatomy and histology—its rarity as a primary disease and its most frequent antecedents—its symptoms, diagnosis, and course—Sarcoma—Cancer of the gall-bladder and bile-ducts.	
Hypertrophy of the liver—diabetic, nutmeg, and obstructed liver.	

Displacement of the liver—Enteroptosis, or Glénard's disease.	
Lymphatic overgrowth—Leuchæmia hepatica.	
The fatty liver—General obesity: its physiology and its treatment.	
Malarial enlargement of the liver—Lardaceous disease—Hepatic tubercle.	
Hydatids of the liver—Other cysts of the liver	551—566

DISEASES OF THE KIDNEYS

FUNCTIONAL DISORDERS

History—Arrangement adopted—Polyuria and oliguria; high and low specific gravity—Baruria and Hydruria—Diabetes insipidus—Renal inadequacy.	
Reaction—over-acid—alkaline—ammoniacal urine.	
Urinary pigments: urochrome—uro-erythrin—hæmatoporphyrin—urobilin and indoxyl—chromogens—Alcaptonuria, etc.	
Lithic (Uric) acid and lithates—Cystine—Oxalate of calcium—Phosphatic deposits—Calcic sulphate—Infected and putrid urine—Bacilluria.	
Hæmoglobinuria—General characters—Toxic form—Infantile form—Paroxysmal form—its relation to malaria—Black-water fever—Raynaud's disease	567—600

BRIGHT'S DISEASE

History—The two chief forms—Additional varieties—Common characters.	
Albuminuria: its detection and estimation, pathology, and significance.	
Casts: their structure, varieties, and diagnostic meaning.	
Renal dropsy: its pathology, its primary and secondary form—Albuminuria without Bright's disease—Dropsy without albuminuria.	
Serous inflammations—Colitis—Dermatitis.	
Retinitis and retinal hæmorrhage—Optic neuritis and atrophy.	
Changes in the heart and arteries: the renal pulse, hæmorrhage.	
Uræmia: eclampsia, coma, amaurosis, vomiting, dyspnœa; theory of uræmia—Anæmia in Bright's disease.	
Parenchymatous or tubular Nephritis, acute and chronic—causes—anatomy—three stages—symptoms—event and prognosis.	
Lardaceous disease generally—its chemistry, causes and distribution—anatomy of the lardaceous kidney—symptoms—diagnosis—treatment.	
Cirrhosis of the Kidney—pathology—causes—anatomy—symptoms and clinical aspects—Consecutive renal cirrhosis—its relation to local disease of the bladder, urethra, and uterus—Hypertrophic cystic degeneration.	
Mutual relation of the several forms described—Geographical and comparative distribution—General treatment and prognosis of Bright's disease, and of its complications	601—671

RENAL CALCULI AND THEIR EFFECTS

Structure and formation of calculi—Lithic or Uric acid and Lithates—Oxalate of lime—Cystine, Xanthine, Indigo, and other rare calculi—Phosphate of lime—Carbonate of lime—Fusible calculus—Ætiology of calculi.	
Symptoms—hæmaturia: its other causes—nephralgia—vomiting—Treatment.	

- Effects—obstruction and suppression of urine—atrophy of one kidney—
Hydronephrosis—anatomy and causes—diagnosis from hydatid and
single cyst—Prognosis and treatment.
Pyelitis and renal abscess, with other causes of pyuria.
Treatment of calculi—solvents of lithic acid—Nephrotomy, nephrectomy,
and nephrolithotomy 672—703

TUBERCLE, CANCER, PARASITES AND ABNORMALITIES OF THE KIDNEYS

- Fatty and lardaceous degeneration—hypertrophy and atrophy of the
kidneys.
Tuberculous pyelitis—Its pathology—Symptoms, causes, and treatment.
Malignant disease of kidney—Sarcoma—Carcinoma—Symptoms—Nephrec-
tomy.
Hydatids of the kidney—Chyluria and *Filaria sanguinis*—Endemic hæma-
turia and *Bilharzia hæmatobia*.
Renal malformations—Floating and moveable kidneys 704—722

ADDISON'S DISEASE WITH OTHER AFFECTIONS OF THE ADRENAL BODIES

- History of the discovery—The anatomical changes in the adrenal capsules
—The symptoms: melanoderma, asthenia, cardiac failure, gastric dis-
turbance—Order of the symptoms—Course, mode of death, and duration
of the disease—Diagnosis—Controversial points—Origin—Relation to
caries and to tubercle—Theory of the symptoms—Prognosis and treat-
ment.
Hæmorrhage, cancer, and other morbid conditions of the adrenal cap-
sules 723—733

DISEASES OF THE PANCREAS AND SPLEEN, ETC.

- Diseases of the Pancreas—Calculi—Abscess—Hæmorrhage—Acute inflam-
mation—Induration—Cysts—Hydatids—Cancer.
Diseases of the Spleen—Abscess—Febrile softening—Induration—Swelling
—Atrophy—Embolism—The spleen in malaria, in rickets, in leuchæmia,
in Hodgkin's disease—The lardaceous spleen—New growths.
Diseases of the Lympharia.
Diseases of the Testes and Ovaries.
Diseases of the Thyroid Body—Exophthalmic Goitre 734—746

DISEASES OF THE BLOOD

ANÆMIA

Blood diseases—Anæmia—Its general characters—Amount of blood—
number of red corpuscles—amount of hæmoglobin—Changes in the
blood-discs and leucocytes—reaction—density—coagulation—Anæmic
murmurs—dyspnœa—Fatty degeneration of the heart—Other symptoms
—Causes and classification—Symptomatic or secondary and idiopathic
or essential anæmia.

LEUCHÆMIA.—History—Definition and characteristic symptoms of leu-
chæmia—The spleen—The bones and the marrow—the lymph-glands—
The blood in leuchæmia—The other organs—Symptoms and course—
Ætiology—Pathology—Treatment—Leuchæmia medullaris—Leuchæmia
lymphatica.

ANÆMIA SPLENICA.—Characters of the blood and spleen—the glands—the liver—Symptoms and course—Diagnosis—Treatment.	
HODGKIN'S DISEASE.—Anæmia lymphatica—History—Ætiology—Morbid anatomy and pathology—Course, prognosis, and treatment.	
IDIOPATHIC ANÆMIA.—Grave, essential or pernicious anæmia—History—Name—Antecedents—Ætiology—Age and sex—Symptoms and diagnosis—Anatomy—Course and prognosis—Treatment.	
CHLOROSIS.—Its relation to sex and age—to menstruation—to imperfect development of the heart and vessels—The blood in chlorosis—Pathology—Symptoms—Prognosis—Treatment	747—787

HÆMORRHAGIC DISEASES

SCURVY.—History—Early symptoms: purpura, spongy gums, hæmorrhagic inflammations—Night-blindness—Course and prognosis—Ætiology—Prophylaxis—Pathology—Diagnosis—Treatment—Infantile scurvy.	
PURPURA.—Symptomatic and primary purpura—Ætiology—Characters and events—Morbid anatomy—Pathology—Diagnosis—Treatment.	
HÆMOPHILIA.—History—Inheritance usually in males through females—Course and symptoms—Pathology—Diagnosis—Prognosis—Treatment	788—807

DISEASES OF THE SKIN

INTRODUCTORY CHAPTER

General pathology and anatomy—Classification: examples—Nomenclature—Historical sketch—The elementary anatomical lesions—The local distribution of cutaneous affections: in depth (bathymetric) and over the surface (regional)—Symmetry—The circumstances: age, occupation, and habits of the patient, season, history of the malady, subjective symptoms, disturbance of other organs, effect of previous treatment—Arrangement followed	808—820
---	---------

ECZEMA AND COMMON SUPERFICIAL DERMATITIS

Definition—Willan's—Hebra's—Its distinction from other forms of dermatitis—Histology—Anatomical lesions—Distribution and local varieties—General symptoms and course—Ætiology—Diagnosis—Prognosis—General treatment—Local applications—Diet and regimen, baths, etc.—Internal remedies—Special treatment of local varieties.	
Impetigo—Its relation to eczema—to pediculi—to contagion—Its treatment	821—848

DERMATITIS PRODUCED BY EPIZOA

SCABIES.—Importance of the disease—Its nature and history—The acarus—The superficial dermatitis it produces—The distribution of the parasite and of the inflammation—Diagnosis—Treatment.	
PHTHIRIASIS.—Pediculi capitis—Pediculi pubis—Pediculi vestimentorum—Their effects on the skin—Treatment of prurigo pedicularis	849—855

PAPULAR FORMS OF CHRONIC SUPERFICIAL DERMATITIS (PIMPLY TETTERS)

Definition—Relation to common dermatitis and to eczema.

LICHEN.—Its traditional species—Lichen circumscriptus—Lichen tropicus—Lichen pilaris—Lichen urticatus, etc.—Strophulus—Lichen scrofulosorum—Lichen planus—Lichen acuminatus—Lichen ruber.

PRURIGO.—Pruritus—Summer and winter prurigo—Prurigo senilis—Idiopathic Prurigo: Hebra's and milder forms—characters and treatment

856—867

PSORIASIS (DRY OR SCALY TETTER)

Frequency—Name—Anatomy and histology—Course—Symptoms—Distribution—Ætiology—Varieties: guttate form, inveterate form—Relation to eczema, to lichen, to pityriasis rubra, and to syphilis—Prognosis—Treatment

868—875

PITYRIASIS RUBRA (UNIVERSAL EXFOLIATIVE DERMATITIS)

Name and traditional species of pityriasis—Pityriasis rosea vel circinata.

Pityriasis rubra—Exfoliative or desquamative dermatitis—History—Accounts by Devergie, Hebra, Wilson, Hutchinson, Baxter—Course and symptoms—Histology—Prognosis—Diagnosis—Treatment

876—880

PEMPHIGUS

Names and definition—Anatomy—Histology—Local distribution—Age and sex—Diagnosis—Ætiology—Prognosis—Pemphigus malignus—Acute form of Pemphigus—*P. foliaceus*—*P. serpiginosus*—*P. vegetans*—Hutchinson's cases—*Cheirpompholyx*—Frequency of pemphigus—Treatment.

Epidermolysis bullosa.

Dermatitis herpetiformis of Duhring—Hydroa æstivalis—Impetigo herpetiformis

881—890

ERYTHEMA AND ITS ALLIES

Definition of the group—Its characters—the anatomical lesions—course—locality—symptoms—ætiology—Symptomatic and traumatic erythema—Varieties: (1) Erythema multiforme—(2) *E. bullosum*—Herpes iris—Herpes gestationis—Hydroa—(3) *E. nodosum*—(4) Urticaria—Urticaria pigmentosa—Treatment of erythematous affections.

Erythematous and other rashes produced by drugs—Copaiba—Bromides and iodides—Belladonna—Opium—Quinine—Salicylates—Arsenic—Mercury—Antitoxic Sera

891—907

DISORDERS OF THE SEBACEOUS GLANDS, THE HAIR-SACS, AND THE SWEAT-GLANDS

ACNE.—Nomenclature—Anatomy and course of local lesions—Distribution—Age and sex—Symptoms—Ætiology—Treatment—Acne tarsi—Acneiform eruptions produced by tar—by bromide—Acne varioliformis.

Comedones without inflammation (acné cornée)—Miliium—Seborrhœa oleosa—Seborrhœa sicca—Seborrhœa corporis—Seborrhœic eczema—Lichen circumscriptus—Xerodermia—Steatoma meliceris and sebaceous cysts.

SYCOSIS.—Name—Anatomy and course—Locality—Diagnosis—Distribution—Treatment—Parasitic sycosis—Sycosis capillitii frambœsiformis.	
FURUNCULI.—Pathology—Anatomy—Course and distribution—Age—Contagion—Treatment—Carbuncle.	
AFFECTIONS OF THE SWEAT-GLANDS.—Anidrosis—Hyperidrosis—Bromidrosis or foetid sweat—Chromidrosis—Hæmatidrosis—Sudamina	908—927

RINGWORM AND ITS ALLIES, WITH OTHER AFFECTIONS OF THE HAIR AND NAILS

RINGWORM.—(1) <i>Tinea tonsurans</i> —The fungus—Anatomy—Course—Events—Histology—Detection of the fungus—Ætiology—Prognosis—Treatment—Parasiticides—Irritants—Mode of application—Epilation—Precautions against contagion—(2) <i>Tinea circinata</i> —Form and locality—Burmese ringworm— <i>Tinea marginata</i> —Treatment of ringworm of the body—(3) Onychomycosis—Rarity and obstinacy—(4) Erythrasma.	
FAVUS.—History—Anatomy—The fungus—Treatment.	
TINEA VERSICOLOR.—Names—Parasitic nature—Appearance—Distribution—Diagnosis—Treatment— <i>Tinea vel Pityriasis rosea</i> .	
ALOPECIA.—(1) Physiological—(2) Febrile and syphilitic—(3) Area—its appearance—locality—spread—question of its parasitic nature—prognosis—diagnosis—(4) Universal alopecia—(5) Congenital alopecia.	
TRICHOCLASIA, or brittleness of the hair—Piedra—Beaded hairs.	
AFFECTIONS OF THE NAILS	928—947

CHRONIC DEEP INFLAMMATIONS WITH CUTANEOUS HYPERTROPHIES AND OTHER CHANGES OF UNCERTAIN NATURE

Deep and chronic dermatitis—its definition—its relation to superficial dermatitis—to hypertrophy—and to new growths.	
GUTTA ROSEA.—Origin in recurrent erythema—development—localities—causes and pathology—relation to dyspepsia—to drink—to ovarian irritation—treatment—Chilblains.	
EPIDERMIC AND PAPILLARY HYPERTROPHIES.—Callosities and corns—Leucoplakia lingualis et buccalis—Warts—Condylomata and mucous patches.	
ICHTHYOSIS.—Anatomy—varieties—xerodermia—Prognosis and treatment—Ichthyosis intra-uterina—Horns—Hyperkeratosis centrifuga.	
SCLERODERMIA.—Scleriosis and morphœa—History—description—distribution—histology—diagnosis—prognosis and treatment—Sclerema neonatorum—Linear atrophy.	
ELEPHANTIASIS.—Nomenclature—anatomy—pathology—relation to chyluria and filaria sanguinis—clinical characters—Dermatolysis.	
XANTHELASMA.—History—course and symptoms—histology—relation to jaundice—Xanthoma diabeticorum	948—968

SYPHILODERMIA

Importance of lues as a cause of cutaneous lesions—their pathological character—The roseolous exanthem—the papular—squamous—pustular—bullous—macular—Alopecia—Tertiary lesions—Cicatrices—Diagnosis of syphilitic dermatoses—Hereditary syphilodermia—Treatment	969—974
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LUPUS AND ITS ALLIES

Definition, history, and nomenclature—Anatomy, histology, and local course—A tuberculous lesion—Distribution—Symptoms—Age, etc.—Diagnosis from cancer, rodent ulcer, and syphilis—Pathology—Relation to tubercle and scrofula, to phthisis and other granulomata—Clinical course and prognosis—Treatment, local, internal, by tuberculin, and by light.

Tuberculous ulcers—Bazin's erythema induratum—Tuberculosis verrucosa.

Lupus erythematosus—Pathology—Locality—Course—Histology—Treatment.

Disseminated erythematous lupus.

Rhinoscleroma 975—990

LEPROSY

History and terminology—Geographical distribution—Anatomical lesions and course—Histology—The *Bacillus lepræ*—Symptoms and event—Ætiology—Treatment—Other exotic diseases—Frambœsia—Acrodynia, etc. 991—996

NEW GROWTHS

Cheloid—Terminology and history—Appearance, course, and symptoms—Histology—Relation to scars—Distribution—Prognosis and treatment.

Multiple fibroma—Anatomy and distribution of the tumours—Their course and treatment—Neuroma—Myoma.

Molluscum contagiosum—Distinction from fibroma and from sebaceous cysts.

Adenoma—Colloid milium—*A. sudoriparum*—*A. sebaceum*.

Angioma or vascular nævus—Elephantiasis teleangiectodes—Lymphangioma—Angioceratoma.

Mycosis fungoides—Anatomy, clinical characters, and course—Precedent dermatitis.

Kaposi's xeroderma maligna—age—primary condition—later course.

Sarcoma, carcinoma, and rodent ulcer of the skin 997—1010

PIGMENTAL AFFECTIONS OF THE SKIN

Albinism—Leucoderma, congenital and acquired—Vitiligo—its relation to leprosy—Canities—Melanoderma, secondary to dermatitis, syphilis, adrenal disease, malaria, etc.—Ephelis—Lentigo—Acanthosis nigricans—Chloasma—Relation of melanoderma to leucoderma 1011—1015

ZONA AND OTHER TROPHONEUROSES OF THE SKIN

ZONA or Herpes Zoster.—Anatomy—distribution—symptoms—sequelæ—Pathology—Traumatic zona—Cases—Treatment.

Trophic nerves, vaso-motor or secretory—characters of neurodermatoses—bullæ, gangrene, distribution, anæsthesia, origin in the ganglia or in the trunks of nerves—Atrophy, pigmentation, and other supposed nervous lesions 1016—1022

REMARKS ON THE PRACTICAL CLASSIFICATION
AND DIAGNOSIS OF CUTANEOUS DISEASES

Diagnosis and nomenclature—Rare and exotic diseases—Factitious and traumatic lesions—Animal and vegetable parasites—Bacteria—Symptomatic rashes—Syphilitic dermatoses—Eczematous, psoriatic, and acneiform dermatitis—Deep dermatitis: ulcers, lupus, and late syphilis—Treatment as the result of diagnosis 1023—1029

INDEX OF SUBJECTS 1031—1041

INDEX OF AUTHORS 1043—1050

ERRATA.

- Vol. ii, p. 346, line 7, *for* Walsh *read* Welch.
 „ p. 384, *for* Dickenson *read* Dickinson.
 „ p. 681, *note, for* Mussen *read* Musser.
 „ p. 729, line 21, *for* 191 *read* 1901.
 „ p. 988, *for* Balzar *read* Balzer.

TEXT-BOOK OF MEDICINE

VOL. II

PNEUMONIA

AND INFLAMMATION OF THE LUNGS

“Vehemens et acutus morbus quem περιπνευμονικὸν Græci vocant.”—CELSUS.

PNEUMONIA.*—*Definition and history—Symptoms and clinical course—complications—mode of death—convalescence—Physical signs—Morbid anatomy and histology: stages, locality, events—Ætiology and distribution—Pathology—infective, epidemic and bacillary pneumonia—Diagnosis—Prognosis—Treatment.*

Secondary pneumonia—Hypostatic pneumonia—Acute pulmonary congestion—Chronic lobar pneumonia.

BRONCHO-PNEUMONIA, or Pulmonary Catarrh.—*Causes in children and in adults—Symptoms and course—Anatomy—Diagnosis—Prognosis and treatment—Suppurative pneumonia.*

CHRONIC INTERSTITIAL PNEUMONIA, or Cirrhosis of the Lung.—*Anatomy—Origin—from the pleura—from the bronchi—Symptoms—Treatment.*

SYPHILITIC INFLAMMATION OF THE LUNGS.—*Cases—Diagnosis—Anatomy—Acquired and hereditary forms.*

GANGRENOUS INFLAMMATION OF THE LUNGS.—*Anatomy—Origin—Factor and other symptoms—Complications and signs—Treatment.*

By *pneumonia* (the peripneumony of older writers), which in Greek medicine denoted “disease of the lungs,” is now understood an inflammation of the texture of the lungs, and when the term is used without

* *Synonyms.*—Acute pneumonia—Pleuro-pneumonia—Peripneumony—Fibrinous, Plastic, or Croupous pneumonia—Sthenic pneumonia—Lobar pneumonia—Massive pneumonia—Acute inflammation of the lungs—Pneumonic or pulmonary fever.

Περιπνευμονία (or in the Attic form περιπλευμονία), from πνεύμων or πλεύμων (whence *pulmo*), the lung, occurs in Hippocrates and Plato. It did not originally carry with it the notion of an inflammatory or febrile disorder. The prefix *peri-* was only dropped in the nineteenth century.

Many German writers have adopted the term “croupous,” because the exudation mainly consists of a fibrinous material like that of croup. In fact, “croupous” has been misused to mean “fibrinous” or “plastic.” The adjective is on every ground objectionable. It suggests some connection with the malady known as croup, whereas there is none; and it confounds a clinical with an anatomical condition.

qualification, one that is specific, acute, and primary. It corresponds to the anatomical condition known as hepatisation, and due to exudation into the alveoli.

It may be defined as a specific febrile disease running a short course, with hepatisation of one or both lungs and pleurisy.

With qualifications the word is applied much more widely, to any form of pulmonary inflammation. As here defined, it excludes (1) that form of lobular inflammation of the lungs which arises by extension from the bronchial tubes, and has been called catarrhal or lobular or *broncho-pneumonia*; (2) the suppurative or *pyæmic lobular pneumonia* which occurs as the result of infective embolism; (3) *traumatic pneumonia* from wounds of the lung, injuries of the chest, or the penetration of foreign bodies into the air-passages. (4) The congestive or *hypostatic pneumonia* which often forms the immediate cause of death in persons suffering from almost any chronic or acute disease, particularly enteric fever. (5) The disseminated *caseous pneumonia* which destroys the lung in phthisis. (6) Lastly, chronic fibrous *interstitial pneumonia*, a distinct process both clinically and pathologically, which arises by extension from the peribronchial connective tissue or the pleura.

Each of the so-called forms or varieties of pneumonia requires carefully chosen adjectives to discriminate it, and it would be well if each had a distinctive and substantive name. But when the term pneumonia is used without a qualifying epithet, it is generally understood (except perhaps in the case of children) to refer to the classical peripneumony.

For the sake of distinction, we may describe the pneumonia now to be discussed as *idiopathic* and *microbic* in its origin, *acute* in its course, *lobar* in its extent, *basal* in its usual distribution, and *plastic* or *fibrinous* in the character of its exudation.

It is true that lobular and pyæmic inflammations of the lung are often acute, that idiopathic is at best a negative phrase, that the disease often exceeds and sometimes falls short of occupying a complete lobe, and that it may affect the apex of the lung; hence no adjective is quite satisfactory. Nevertheless we shall see that in its natural history and its clinical features, as well as in its anatomy, pneumonia in the restricted sense of the term is one of the most peculiar and distinctive of diseases.

The following passage from the Latin version of Artæus, the Cappadocian, who wrote about 70 A.D., shows how little the symptoms of the disease have changed.

“Spiritus gravis est ac fervidus. Facies rubet, et in ea præsertim malæ. Quod in oculis album est humidissimum et pingue apparet: acumen nasi simum fit; venæ in temporibus et cervice elatæ sunt; cibi fastidium. Pulsus in initio mali magnus, vacuus, creberrimus, quasi compressus. Calor in exterioribus partibus obscurus et humidior; in interioribus autem aridus atque ferventissimus; ex quo spiritus incalescit, sitis fit, et linguæ siccitas, et frigidi aeris desiderium, et animi molestia. Accedit etiam tussis, quæ plerumque sicca est, vel si quidquam excreatur pituita prodit, aut squamans, aut bile saturata, vel cruenta et coloris floridissimi.”—ARTEÆUS.

Under the title Peripneumony it was well known in Greek medicine. Morgagni identified the characteristic clinical symptoms with the equally characteristic solidification of the lung, and Laennec discovered its physical signs, and described them very much as they are now described. The two most important advances made since 1826 have been the abandonment of the so-called heroic method of treatment, and the discovery of the specific pneumococcus.

Symptoms and course.—An attack of pneumonia often sets in suddenly with a well-marked rigor. This occurred in 241 out of 280 of the writer's cases, and again in 782 out of 975 cases ('Collective Investigation Record'). In mild cases there may be only a feeling of chilliness, which almost escapes notice. In the case of children, the disease is frequently ushered in by convulsions or by vomiting. When, however, pneumonia supervenes as a secondary complication of other diseases, its onset is insidious, so that no symptoms mark its onset, and we first discover its presence by its characteristic physical signs.

Pyrexia develops very rapidly, so that the temperature may reach 104° within three or four hours from the rigor. At the same time the pulse becomes frequent, full, and "bounding," *i.e.* at once large and resistent—not soft as in typhus and enterica. As the rigor passes off, the cheeks acquire a crimson flush. A point on which Addison used to lay great stress is that, as tested by the hand, there is in pneumonia a pungent dry heat of skin hardly observed in any other affection. We saw how this has since been confirmed by the observations of Schülein (vol. i, p. 36); and it agrees with the description of Aretæus: *calor aridus atque ferventissimus*.

The thirst, restlessness, headache, are like those of any febrile disorder. Indeed, before the art of auscultation was introduced, a case of pneumonia was often called "typhus" or "continued fever;" or if, as sometimes happens, there was violent delirium from the first, and the patient died comatose, the disease was set down as "meningitis" or "phrenitis." But even before the physical signs of the disease were known, experienced physicians seldom failed to recognise pneumonia; and at the present day the aspect often shows us the nature of the case before we see the rusty sputa or use the stethoscope.

There is a marked change in the ratio of the pulse to the respiration. In all fevers, both pulse and respiration are accelerated; but in pneumonia the increased frequency of the respiration is out of proportion to that of the pulse, the ratio being as one to three, or one to two, instead of 1 to 4 as in health; or the patient may breathe sixty or eighty times a minute. Walshe in one case observed the number of respirations in the minute to be actually greater than that of the heart-beats, and Jürgensen recorded similar observations in the case of old people with slowly-acting hearts and atheromatous vessels. It is curious that the distress produced by such rapid breathing varies extremely in different cases; some patients, breathing thirty or forty times a minute, seem to be unconscious of dyspnoea.

As the respiration is quickened, the nostrils expand during inspiration, while the sterno-mastoidei and other muscles of forced breathing begin to work, and the cheeks and lips acquire a purplish tint.

In marked cases the flushed face, erect posture, anxious look, bright eye, and rapid breathing, with the herpes of the lips, the burning skin, and the short, repressed cough, give a *facies* which is very characteristic. But even with careful scrutiny, there is sometimes complete absence of physical signs for the first twenty-four hours, or even longer.

Pneumonia is accompanied by pleurisy, and this causes *pain*, usually seated near the nipple or towards the axilla. That this *point de côté* is really due to the coincident pleurisy was taught by Addison long ago. It is sometimes the first symptom which the patient notices; but often the pain does not appear until the second day, and it may occasionally be entirely absent. When the pleuritic pain is severe, the patient tries to fix

the ribs by pressure with his hand, and curves his spine towards that side, so as to bring the ribs as close together as possible.*

The *cough* is interrupted and restrained by the pain; and this distress is increased by the fact that the *expectoration* in pneumonia, though scanty, is tenacious and viscid; it clings to the lips and to the spittoon, even when inverted. Pneumonic sputum has a peculiar colour, due to the albuminous and mucous secretion being intimately mixed with air and blood. The most characteristic tint, and also the most usual, is bright orange, tawny, or like the rust of iron, so that it is commonly called "rusty" expectoration. But sometimes the tint is a paler apple-yellow, and sometimes it is the bright scarlet of unaltered blood. Spitting of pure blood enough to be called hæmoptysis is not common; but it may occur in cases uncomplicated by tubercle, purpura, or cardiac disease, and the result may be as good and speedy a recovery as usual.

In some cases the sputum is thin, watery, and of a brownish-red colour, so that it is compared with prune-juice or liquorice-water. Such sputum is seen chiefly at advanced stages of the disease, and when it is taking an unfavourable course; but it may be present at an early period, and in cases that ultimately do well.

Remak showed, many years ago, that in pneumonic sputum there can sometimes be detected with the microscope branching fibrinous casts of the smallest bronchial tubes.

Micrococci are present and, with few exceptions, are encapsulated diplococci (p. 14).

Some patients have no cough, and therefore no expectoration, throughout the whole course of pneumonia; but this is rare except in the case of children and aged persons. As in other affections, although a cough brings up sputum into the pharynx, it is swallowed again by children and old men. The writer has seen this curious inability to expectorate in school-boys as old as fourteen, and once or twice in young adults; while on the other hand children of three and four will sometimes expectorate well.

In 290 cases observed by the writer, there was no expectoration in 130, of whom 113 were children; of the remaining 160 cases, there was no blood in the sputum in 24, free hæmoptysis in 13, a rusty colour in 116, and a greenish tint in 7.

Course.—After the acute onset, pneumonia runs on for some days with but little change. As more of the pulmonary tissue undergoes hepatisation, the physical signs become more marked; but there is often no corresponding increase in the severity of the symptoms.

The type of the *pyrexia* is, as a rule, continuous, with but slight diurnal oscillations.†

The *pulse* does not rise so much as the temperature and respiration. It often becomes more frequent from day to day, but sometimes remains for several days at 90 or 100, and becomes gradually smaller and softer. In adults there is always ground for alarm if it rises above 120, but in

* So Celsus: "*Id genus morbi (quod περιπνευμονικόν Græci vocant) plus periculi quam doloris habet*" (iv, 7).

† Wunderlich states that it is not uncommon for an irregular and sudden elevation of temperature to occur, which is followed by a no less sudden fall through as many as 7° or even 9° F. to the normal point or below it, and that again in a few hours by a fresh elevation. I have observed more than one case at Guy's Hospital in which similar strange deviations in the regular course of the fever took place again and again, and, in fact, made up the greater part of the temperature chart, without obvious cause.—C. H. F.

children it may reach 130 to 140 without imminent danger; in old people it is commonly much slower.

The *tongue* is plastered with white thick fur, but in severe cases becomes after three or four days dry, brown, and covered with sordes. The bowels are generally constipated, but sometimes there is diarrhœa.

A symptom which usually appears between the second and the fifth day is an eruption of *herpes* upon the lips or face—a crop of clear painless vesicles occurring in a group on a red patch of skin. It has been noted in from one third to one half of all cases of pneumonia, and is said to be a good sign. This traditional opinion is confirmed by the observations of Geissler, who found ('Arch. d. Heilkunde,' 1861) that out of 431 cases the mortality without herpes was 29·3 per cent., whereas with herpes it was only 9·3 per cent. The writer found herpes in 53 out of 434, *i. e.* in only an eighth, but this is certainly below the mark. In 25 fatal cases seen in consultation, not one had herpes. In some cases the vesicles are few, and may be easily overlooked, particularly when the upper lip is covered by a beard. Herpes is much less frequent in secondary than primary pneumonia, a fact which may partly account for its good omen.

After the first few days the *skin* in many cases becomes moist, and there is occasionally profuse sweating. An icteric tinge of the conjunctivæ is not uncommon, and sometimes there is well-marked *jaundice*. Formerly it was taught that this occurred only when the base of the right lung was the seat of the disease, and that it was due to the extension of "the disease" through the diaphragm to the liver—an unscientific conception. The truth is that icterus may appear when pneumonia affects the left lung or the upper lobe of the right.

The *urine* in pneumonia is scanty, of high specific gravity, deeply-coloured, and strongly acid, depositing lithates in abundance. The amount of *chlorides* excreted by the kidneys undergoes a great diminution; they may even be altogether absent. This is a point of some diagnostic value, although a less degree of the same thing occurs more or less in other febrile diseases. The explanation is apparently that the salts are retained in the hepatised lung, for, as Cohnheim argued, the diminished supply of them in the food is insufficient to account for so great a difference. During convalescence they reappear, probably in increased quantity.

Not infrequently the urine contains *albumen*, perhaps in more than half the cases. It is not of ill prognosis, and never leads to Bright's disease.

The *nervous system* is, as a rule, less affected in pneumonia than in other maladies with equally high temperatures. Many patients retain their consciousness throughout the illness, or merely wander at night during their broken sleep; but in other cases *delirium* appears as the fever increases, and it may be of a furious maniacal type.* Pneumonia of the apex is particularly apt to cause delirium. When the patient's habits are intemperate, the symptoms of delirium tremens frequently appear.

The *blood* in pneumonia is usually altered by a decidedly increased number of leucocytes being present. This leucocytosis begins early and disappears with the crisis, but more gradually than the fall of temperature. It is absent in very slight cases and in severe cases with slight reaction,

* An inherited neurotic tendency seems to lead to the occurrence of severe cerebral symptoms in pneumonia. In the case of a man whose last hours were passed in a paroxysm of raging madness, I was told that hardly one of the brothers and sisters of the patient had escaped some form of neurosis.—C. H. F.

but in the latter is of bad omen. After the crisis there is a slight increase of eosinophil leucocytes.

These facts are established by Ehrlich, von Jaksch, and Türck in Germany, and by the numerous observations made in the Massachusetts General Hospital at Boston.*

Defervescence.—When pneumonia ends in recovery, the subsidence of the pyrexia and of other symptoms is usually abrupt, by *crisis*. The improvement usually begins in the evening or night. The fall of the temperature is rapid, being often completed in twelve or sixteen hours, and sometimes in five or six. For the next day or two it often stands below normal.

Less frequently the defervescence in pneumonia takes place gradually, by *lysis*, and occupies two or three days, or even more.

In cases which end by crisis, it is surprising how much better the patient feels as soon as the pyrexia has subsided. His appetite quickly returns, he sleeps well, and his skin perspires comfortably. Both the pulse and the respiration decline step by step with the temperature. Yet the physical signs at first show no improvement in the affected lung, and several days may pass before the consolidation clears up.

Modes of death.—In unfavourable cases, the pulse is not only unusually frequent, but also irregular and weak. The breathing becomes more and more rapid, but shallow and ineffectual, the patient being no longer conscious of the want of air. He sinks down in bed; the face is pale and livid in hue, leaden or slate-coloured; the skin is covered with a cold sweat; the pulse becomes weaker, and finally imperceptible. Occasionally death is sudden, from syncope, perhaps when the patient raises himself in bed. Now and then the occurrence of the crisis is followed by prostration and collapse, from which the patient never rallies. Secondary pneumonia, septic pneumonia, and pneumonia potatorum have their own peculiar symptoms before death.

Duration.—The pyrexia most often lasts six, seven, or eight days (169 out of 316 of the writer's cases), next often either four or five (51), or else nine or ten (48). The short and abortive cases, under five days, are most often seen in children, and seldom after twenty-five. The shortest observed was in a girl of seven, who had fever just under forty-eight hours. The long cases, over ten days, either recover by lysis or are retarded by complications.†

Complications.—Bronchitis and pleurisy are constantly present. The former is a serious condition when chronic, and coupled with emphysema, and may determine a fatal issue of unilateral pneumonia in elderly patients. The latter is painful rather than dangerous, and does not often lead to hydrothorax.

The *sequelæ* of pneumonia are few. Not infrequently empyema supervenes, and when dulness and pyrexia continue after ten or twelve days, its presence is almost certain. It depends sometimes on the presence of pneu-

* Ehrlich's "Anämie," in Nothnagel's 'Spec. Pathologie u. Therapie,' vol. viii; also separately translated by W. Myers as 'Histology of the Blood,' 1900; Cabot's 'Clinical Examination of the Blood,' 1897.

† In about 65 per cent. of cases, in which the exact duration of the disease can be reckoned from an initial rigor, or convulsion, or vomiting, the crisis is found to occur between the fifth and the eighth days; next often on the sixth, or the seventh. In some cases it is earlier; in others it is delayed until the second week. It is rare for fever to continue longer than this; and when defervescence fails to occur by the fourteenth day there is ground for suspicion either that the case is not one of true pneumonia, or else that some complication is present.—C. H. F.

mococci alone, sometimes on that of strepto- or staphylococci, alone, or together, or with pneumococci; and occasionally tuberculous bacilli may be found. Dr Hale White found 26 cases of empyema among 890 of pneumonia at Guy's Hospital ('Reports,' vol. li), but there were only six among 708 cases at St Thomas's ('Reports,' vol. xix). The writer met with 24 in 430 cases.

True pneumonia (acute, lobar, and fibrinous) appears never to result in phthisis, or to be closely followed by it. Gangrene is a dangerous, but a very rare, complication or sequel.

Pericarditis is a dangerous complication; and with double pneumonia it is usually fatal. Happily it is not common (14 cases in the above-mentioned 430). Septic endocarditis may be combined with inflammation of the pericardium, or it may occur independently (7 cases in 430, one of them in company with pericarditis and three with meningitis).

Inflammation of the meninges as a complication of pneumonia was often assumed to account for delirium or mania, but without warrant. It is now, however, known that true specific lepto-meningitis does occur as an occasional complication of pneumonia, and the specific pneumococci are found in the exudation. There were 5 cases in the writer's 430.

Jaundice has been mentioned as an occasional and, so far as is known, an insignificant symptom. Inflammation of the larynx, the tonsils, or the middle ear, are probably accidental complications, and the same may be said of epistaxis.

Diarrhoea from catarrhal or ulcerative colitis has been recorded in fatal cases by Bristowe and by some other physicians.

Recurrence.—A person who has once suffered from pneumonia is afterwards more likely to be attacked by the disease. Instances have been recorded in which it has recurred eight, ten, and even more times. Sometimes the same part of the lung is affected on successive occasions, sometimes not. Relapses are not frequent, if by this term we understand the repetition of the morbid process before the patient has completely recovered; but the spread of the disease to the other lung not infrequently comes on in the form of a relapse.

Physical signs of pneumonia.—The above account of the symptoms and course of pneumonia is almost such as might have been written a century ago. The signs discovered by Laennec and his successors form a consecutive series, which should be studied along with the anatomical change of which it is the indication.

The earliest sign of commencing hepatisation of the lung is, according to Stokes, a peculiar harshness of the respiratory murmur; but a less doubtful one is the presence in inspiration of a very characteristic sound known as pneumonic, or "fine (*i. e.* small) crepitation."* It is like that produced by rubbing a lock of hair between the fingers close to one's ear, a comparison of C. J. B. Williams. It is much "smaller" than any other râle, and would not be recognised as due to bursting of bubbles, as would gurgling or mucous rattles. It is consonating, musical in character, and high-pitched in note. Laennec, who discovered it, calls it "*une espèce de crépitation ou de râle léger, dont le bruit peut être comparé à celui du sel que l'on fait décrépiter en le chauffant dans une bassine.*" He supposed it to be pathognomonic of pneumonia. A sound very like it may be occasionally heard in cases of œdema of the lung in Bright's disease, but this may be due

* *Fr.* Râle crépitant.—*Germ.* Das knisternde Rasseln.

to lobular catarrh. An identical or closely similar crepitation may often be heard in cases of lobular pneumonia (pulmonary catarrh) in children. When a person who has been lying on the back with some febrile disease is made to sit up and breathe deeply, so as to fill those parts which have been for some time disused, a sound like the crepitation of pneumonia may sometimes be heard at the base of the lungs. In the last case there can be no doubt that the cause of the sound is the opening up of portions of the tissue that had become collapsed; and most authorities agree that in pneumonia it has a similar origin, and is due to the inspiratory separation of the walls of bronchioles which, being swollen, had come into contact in expiration. It is heard only during inspiration, and sometimes only just at the end of deep inspiration, as after coughing.

If this is the case, it differs in origin from other râles, whether crepitant or nonconsonating, and does not depend on the bursting of bubbles, but on the admission of air through a narrow and adhesive passage. Whatever its mechanism, true pneumonic crepitation is to the ear quite distinct from any other rôle.

One must search carefully for this important sign before concluding that it is not present. In some cases of pneumonia crepitation is never discovered, but it is transitory, and so may have passed off before an examination of the chest is made. Sometimes, however, it remains audible during the whole course of the disease, not at the same spot, for where there is complete consolidation it disappears; but in one spot after another, as they are successively attacked by the disease.

As inflammatory "engorgement" or "congestion" passes on to consolidation (red hepatisation), there are developed other signs—dulness on percussion, bronchial breathing, and bronchophony with increased tactile vocal fremitus.

The degree of *dulness* varies much in different cases. It is not so absolute as in cases of fluid effusion into the pleura; nor is the sense of resistance to the finger so great. In exceptional cases the percussion-sound undergoes modifications, the explanation of which is obscure. Thus a cracked-pot sound is sometimes elicited either by unskilful percussion, or by the chest walls being very yielding, as is frequent in children. Occasionally the sound, while still short and high-pitched, acquires tone; but more often this tympanitic dulness (vol. i, p. 1025) is heard not directly over the solid lung, but in its neighbourhood. Thus hepatisation of the upper lobe of the lung behind may cause a Skodaic percussion-note beneath on the same side.

The *bronchial breathing* that accompanies the second stage of pneumonia is very characteristic. Usually it is a typical in-and-out, whiffing, tubular sound, of equal length in expiration and inspiration, and in quality not unlike a to-and-fro bellows murmur of the heart. Occasionally no sounds are audible, and the explanation given, and sometimes verified after death, is that the tubes passing to the consolidated part happen to be filled with fibrinous plugs.

Bronchophony generally goes with bronchial breathing, and presents like differences of degree. Dr Gee remarks that in infants with pneumonia a bronchophonic cry is often the only auscultatory sign that can be obtained. This increase of vocal resonance is much less remarkable in women than in men, and is best heard with a bass voice and slow counting.

Pectoriloquy is sometimes heard over the solid lung, either with the laryngeal or the whispered voice.

Increase of vocal fremitus is less variable and is also best heard in the case of men; but even this may be absent, and we must remember that on the right side fremitus is often greater in healthy persons than on the left.

Lastly, when pneumonia affects only the central part of the lung, or reaches no part of the surface except that which is in contact with the diaphragm, physical signs are absent, until it has spread to the surface.

During the stage of *resolution* the peculiar auscultatory signs of hepatisation gradually disappear. The breath-sounds lose their tubular blowing quality, and are replaced by râles. Sometimes a crepitation is now heard, which may be scarcely larger or less musical than that of the early period of the disease; this is called *crepitation redux*. In other cases the sounds are so "large," and at the same time so consonating, that one might imagine the patient to be in an advanced stage of phthisis, with cavitation. A considerable time generally passes before the vesicular murmur becomes as pure and distinct as it should be. The percussion-resonance also may long remain deficient, even when there has been no pleuritic effusion.

Anatomy.—The pneumonic process, which is revealed by the signs just described, consists in rapid conversion of the spongy pulmonary tissue into a solid mass.

The first is the stage of "active congestion" or *engorgement*, a stage added by Bayle to the three described by Laennec. The affected part of the lung is heavy, and dark red in colour. It pits under the finger, and a reddish frothy serum oozes from its cut surface; if the pressure is increased, its substance breaks down. Microscopically, the most obvious appearance is the dilated and tortuous state of the capillaries of the alveolar walls; minute punctiform hæmorrhages are also to be seen in the connective tissue between the lobules and beneath the pleura. This is a short and somewhat hypothetical stage.

The first to be usually recognised during life or after death is the stage of *red hepatisation*, when the lung has become like liver. It now sinks in water, it does not crackle when pressed, it is easily broken, and little or no fluid can be squeezed from it. Its cut surface has a dull, lustreless appearance, and is distinctly granular. The granules are composed of a solid inflammatory exudation, which completely fills up the alveoli and infundibula; by scraping the cut surface, we obtain fibrinous casts of the spaces in which they were moulded. The seat of the plastic exudation in lobar pneumonia is therefore, as Addison first stated, not the interstitial spaces of the lungs between the vesicles, but the alveoli and infundibula themselves, as also the intra-lobular air-passages and smallest bronchioles. The red colour of the lung at this period is probably due chiefly to the fact that great numbers of red discs are extravasated and mingled with coagulated fibrin and exuded leucocytes.

The next stage is that of *grey hepatisation*. This is marked, first by a change in the colour of the hepatised lung from red through reddish grey to grey or pale yellow; and secondly, by the diseased tissue becoming even softer than before, by its being less markedly granular on section, and by its beginning to exude on pressure a turbid fluid, more or less opaque, white, and puriform. The most advanced stage of grey hepatisation has been described by some pathologists as a fourth stage, which they term

purulent infiltration. Histologically there is a wide difference between the characters of red and those of grey hepatisation. In the latter no fibrinous coagula are visible; the substance which fills the alveoli is now a mass of crowded leucocytes. The extravasated red discs are no longer to be seen; probably they are taken up by phagocytes, or they break up and are absorbed by the lymphatics. The change of the tissue in colour may be also partly due to the increased exudation compressing the pulmonary capillaries and driving the blood out of them.

The most important histological distinction between the two kinds of hepatisation is afforded by the state of the alveolar walls. In the "red" stage they are unaltered, except that their capillaries are distended with blood-discs; in the "grey" stage they are infiltrated with leucocytes, which fill up every interstice between the air-vesicles. The iron-grey tint is due only to the presence of carbon from inspired air; for a hepatised lung from a child or from an animal which died in the country is yellowish white, not grey. The friability of a hepatised lung depends partly on softening of the tissue, but chiefly on its being no longer elastic and yielding.

At all stages of hepatisation the characteristic encapsuled diplococci are to be seen after staining a section or a scraping, and sometimes streptococci appear in addition.

Distribution.—Pneumonia never attacks the whole of the lungs at once; it begins at some one spot, from which it rapidly spreads.

All physicians are agreed that the right lung is more often the seat of pneumonia than the left, the proportion being about as five to four. Sometimes both lungs are attacked together or, more often, in succession. On either side the lower lobe is affected far more frequently than any other part; Jürgensen says in three cases out of four, and among 450 cases at Guy's Hospital the proportion was three out of four and a half. As a rule hepatisation begins at the extreme base, and extends gradually upwards from day to day; but exceptionally it may spread upwards and downwards from the middle of the lower lobe, or backwards from the anterior border. If it begins in the upper lobe it usually passes from the apex downwards. Sometimes its distribution remains strictly limited by the lobar septa: sometimes its spreading edge forms a horizontal line, ignoring them altogether. The most common seat of pneumonia is the right base, next the left base, next both bases. At the apex, left pneumonia is more rare than right. In 434 patients of all ages, the writer found the hepatisation to be in 291 basal (right and left nearly equal), in 80 apical (60 right and 20 left), and in 8 central, beside 55 cases in which both bases or a base and apex were affected. In only one case were the two apices alone affected.

In children, Henoch found right apex pneumonia in 21, and left apex pneumonia in only 4 cases out of 74 of lobar pneumonia. Dr Goodhart's numbers are 18 right and 17 left apex pneumonia out of 120 cases of lobar pneumonia in children.

Anatomical events.—The results of pneumonia are unlike those of ordinary inflammation; for either death occurs, or the whole affected tissue quickly clears up and returns to its former condition. The formation of an *abscess* in the lung, as the result of true pneumonia, is admitted by all writers to be very rare, and some, including the present writer, doubt whether it ever occurs.* Cases have, indeed, been recorded; but the ques-

* Laennec admitted "Il n'y a pas de lésion plus rare qu'une véritable collection de pus dans le tissu pulmonaire."

tion is whether a more accurate pathology might not have led to a different interpretation. They may have been circumscribed empyema, or suppurating bronchial sacs or hydatids; or true pulmonary abscesses, but not of pneumonic origin—septic or pyæmic. The termination of pneumonia in *gangrene*, though also very infrequent, undoubtedly occurs under special circumstances to be afterwards discussed (*infra*, p. 33).

When pneumonia ends by *resolution*, it is after clear evidence from physical signs that the disease had reached the stage of red hepatisation; the only doubt is whether the supervention of the third stage of grey hepatisation is compatible with recovery. In their work on Pathological Anatomy, Wilks and Moxon express a decided opinion that in most cases the disease has not advanced far beyond the “red” stage when resolution begins, and most writers agree with this view.

There are two ways in which the exudation that fills the pulmonary alveoli may be got rid of: one is by its escaping into the air-passages and being expectorated; the other by its being reabsorbed into the blood. Rindfleisch maintained against Jürgensen that the greater portion takes the former course. This seems evident from the solidification of the lung continuing for days after the expectoration has ceased. Probably the chief channel whereby the heavy mass of exudation is removed is that of the lymphatics and veins—the salts in solution, the leucocytes after breaking up, and the bacilli by phagocytosis.

After the subsidence of pneumonia, if the patient should die at no long interval from some other cause, the lung is found to have nearly regained its healthy appearance, but to be slightly redder and tougher than natural.

It is a very important question whether, instead of subsiding, true pneumonia ever leads to permanent changes in the lung, and particularly to development of fibrous tissue, *i. e.* to cirrhosis or chronic pneumonia. Although such a termination of pneumonia is quite the exception, we shall see reason to believe that it may occur in very rare cases, either as a uniform induration, or as a circumscribed interstitial transformation into fibrous tissue (cf. *infra*, p. 22, 28). As for a termination of pneumonia in phthisis, there is every reason to believe that the cases that have been so interpreted were phthisical from the beginning.

Concomitant post-mortem lesions.—*Pleurisy* of the dry kind is a constant part of the disease. Wherever the hepatisation reaches the surface of the lung, the corresponding part of the pleura becomes covered with lymph. But the term “pleuro-pneumonia” is unnecessary, and, indeed, misleading, since after death exudation on the pleura is always found, whether pleurisy was recognised during life or not.

Pericarditis is much less common, and peritonitis scarcely ever seen; but Dr Fagge stated that very rarely he had found the upper part of the peritoneum coated with lymph. Sometimes the mediastinal tissues are infiltrated with a gelatinous exudation. *Endocarditis* is a somewhat rare but undoubted pneumonic complication, and pneumococci are found in the valvular tissue.

The bronchial lymph-glands are constantly found reddish grey, swollen, and soft; moreover the subpleural lymphatics corresponding with the seat of the pneumonia may sometimes be seen distended with white or yellow lymph, so as to give a marbled appearance to the surface.

A rare lesion in an autopsy on pneumonia is acute *meningitis*. Of four cases recorded by the writer, two were cerebro-spinal.

Somewhat less infrequent is ulceration of the larynx; the ulcers are usually found over the arytaenoid cartilages, where they occur from many other causes.

Probably it is to the pyrexia which accompanies pneumonia that are due other slight but almost constant morbid changes that are met with after death—a moderate degree of enlargement with softening of the spleen, slight catarrh of the intestine, and cloudy swelling of the kidneys.

Ætiology.—Pneumonia is met with at all ages, though rare in children under three years old; and in both sexes, though commoner in men than in women. Among 434 patients the writer found 320 men and 114 women. The difference between the sexes is much greater in adult life than in childhood, and it is remarkable that it disappears in the statistics of prisoners confined in gaol. We may therefore plausibly ascribe it to difference of occupation and habits.

It used to be thought that children were seldom the subjects of lobar pneumonia, but though broncho-pneumonia is more common under ten years of age, it is far from being the constant form of pulmonary inflammation. The writer has met with it in three infants under two years old. Among 434 cases no less than 95 occurred under ten, 93 between ten and twenty, 87 between twenty and thirty, 62 between thirty and forty, 54 between forty and fifty, 20 between fifty and sixty, 18 between sixty and seventy, and 5 between seventy and seventy-five.

Lobar pneumonia is met with in every race of the human family. Among the lower animals it is a frequent cause of death in monkeys, horses, and among cattle; it frequently assumes an epizootic form, and decimates both oxen and sheep.

It has been questioned whether pneumonia is more apt to occur in those who are strong and healthy, or in those who are weak and delicate. There is no difficulty in finding instances in support of either view, and no one can help being impressed by the frequent case of a vigorous young man carried suddenly off by this disease. Yet these are exceptions.

If we take the cases of hepatisation of the lung as they occur in the deadhouse, the majority are found to be secondary to cardiac or renal disease, to enteric or other specific fevers, or to wasting maladies like cancer, paraplegia, and more particularly diabetes. On the other hand, it is not frequent for acute pneumonia to be associated with phthisis, with bronchitis, or with primary pleurisy.

Even if these cases be excluded, and we examine the records of apparently primary idiopathic pneumonia, we find, as Wilks long ago taught, that in many cases the patient is old and feeble, or half starved, or habitually intemperate.

The principal exciting cause of pneumonia—as of pleurisy and bronchitis—is commonly supposed to be “catching cold.” There is no physician who cannot recall to his memory instances in which patients were attacked immediately after getting wet through, or after lying on damp grass, or after some other very definite exposure to cold. But when we tabulate our cases, we can only admit a chill to be a cause of the disease, in comparatively small proportion.* Moreover, sailors and others whose occu-

* Among 205 cases collected by Grisolle, 45 were supposed to be due to cold; but among 186 cases of Ziemssen's only 10, and among 212 cases of Griesinger's only 4. These figures are taken from Jürgensen's article in ‘Ziemssen's Handbuch.’

pations expose them to bad weather do not seem especially prone to pneumonia.

With respect to the various seasons of the year, it has been shown that whereas in Vienna the prevalence of bronchitis reaches its maximum in March, and then gradually declines through the rest of the spring and summer, the prevalence of pneumonia increases steadily from February to May, after which it falls rapidly. This last part of the statement must not be taken as applying to other places besides Vienna, still less to Europe generally; for Jürgensen finds that there is a broad difference between continental and insular climates as regards the months in which pneumonia is most apt to occur; in the former it is between March and May, in the latter between December and February. But if the year be divided into two halves, the one from December to May, the other from June to November, then it is found that, throughout Europe, two thirds of the cases of pneumonia fall into the first half, one third into the second.

The geographical distribution of pneumonia is as different from bronchitis as its seasonal prevalence, for the latter is most frequent in cold, damp climates, in England, northern Europe, and the United States; and it is rare in equable climates like those of the Mediterranean coasts, the Atlantic and Pacific islands, and in India and China.

Pneumonia, however, though common in the north and south temperate zones—in Melbourne, and New Zealand, and Buenos Ayres, as well as in Europe and the United States of America—is also found frequently in Italy, in northern China, in Beloochistan and other high districts of India, in the Transvaal and in Peru. In all these places there is rapid fall of temperature and cold nights after very hot days.

There seems, therefore, reason to believe that a sudden fall of temperature after the heat and sweat and fatigue of the day, particularly with dry air and cold east or north-east winds, is really an important contributing cause of acute lobar pneumonia.

True pneumonia is not set up by local *injuries* to the chest, wounds of the lungs, foreign bodies in the bronchi, or any like causes. Traumatic inflammation of the pulmonary tissue is chronic and interstitial, or may be acute and caseous, but it does not cause true hepatisation. It is impossible to induce the affection experimentally in animals by local irritation.

Infection.—Occasionally pneumonia assumes an *epidemic* character among men, as among cattle. This is seldom seen now, but there is reason to believe that, as with measles, syphilis, influenza, and perhaps the sweating sickness, this was once a more frequent type, and that its present sporadic occurrence has not been, and possibly may not be, always the rule.

In Sturges' and Coupland's monograph there are accounts of epidemic and very fatal pneumonia in Flanders during 1557, in the garrison of Philipsbourg in 1688, and in Iceland in 1863. They also refer to an epidemic in the 22nd Regiment, when stationed in New Brunswick, recorded by Dr Walshe in the 'Army Medical Reports' for 1867, and to one in the Mediterranean Fleet in 1860, described by Dr Bryson in the 'Lancet' (Jan. 9th, 1864). Jürgensen recorded the prevalence of epidemic pneumonia from 1873 to 1881 in the village of Lustnau, near Tübingen, and Dr Ballard reported to the Local Government Board an outbreak of apparently infectious pneumonia at Middlesborough in 1888.*

* See the bibliography by Dr Coupland in the Report on Acute Pneumonia ('Collective Investigation Record,' vol. ii, p. 10); also four instances, *ibid.*, vol. i, p. 104, and an account of an epidemic of pneumonia in the Punjab by Surgeon-Major Maunsell, vol. ii, p. 77.

The historical aspect of epidemic pneumonia was well treated in Dr Wilson Fox's posthumous work on diseases of the lungs. (See also Dr Whitelegge's article in the first volume of Allbutt's 'System of Medicine'.)

Cases have been reported of pneumonia spreading in families. One striking instance of five adult brothers and sisters, living in the same house, and successively carried off by the disease, was described by Dr Patchett in the 'Lancet' for 1882.

During the last ten years the frequency of pneumonia as a sequel to *influenza* has been forced upon the attention of physicians in this country, on the continent of Europe, and in America. Some of the epidemics of pneumonia, particularly those in a single town or village or in a single house and family, are no doubt to be explained as pneumonia following epidemics of influenza, just as bronchitis and pulmonary catarrh appear epidemically after an epidemic of measles, or albuminuria after an epidemic of scarlet fever. These cases of pneumonia following influenza form a link between the ordinary primary sporadic cases, and epidemics like those above quoted.

Pneumonia frequently occurs as a complication of other diseases, particularly typhus, relapsing and enteric fever, variola, puerperal fever, and other septic conditions. Less frequently it follows erysipelas, scarlatina, and diphtheria and rheumatism. Of chronic diseases the most often complicated by pneumonia are diabetes, alcoholic poisoning, Bright's disease, acute and chronic cancer, and many lingering maladies of the nervous system. Indeed, we may almost say with Arbuthnot, if we use pneumonia in the wider sense of the term in which he employed it, that a peripneumony is the end of most diseases.

Specific microbes.—In the pneumonic lung, the discovery of microphytes has rewarded the skill of modern histologists. Friedländer described in 1882 a micrococcus, usually occurring in pairs or chains, and surrounded by an envelope; this he called *pneumococcus*. It is frequently present in the hepatised lung, and has been found in the rusty sputum. Klebs had (1877) previously described what was probably the same organism, and afterwards Dr Giles found them in India ('Brit. Med. Journ.,' July 7th, 1883). They formed a characteristic nail-shaped cultivation in agar-agar, and were believed to produce pneumonia in mice; but they may also occur in lobular and other kinds of inflammation of the lung. Moreover, other microphytes, spherical and rod-shaped, have been found in cases of pneumonia.

A better claim to be the specific microbe of pneumonia is that of another micrococcus or short, lancet-shaped bacillus, which also frequently occurs in pairs and is surrounded by a capsule. It was brought forward as a rival to Friedländer's microbe by Fränkel in 1884, but had been previously discovered in hepatised lung by Talamon, and inoculated by Dr Sternberg in America. In fact, it is probably identical with the organism which occurs in healthy saliva, and has been variously named *Bacillus salivarius*, *B. sputigenus*, and *Streptococcus lanceolatus Pasteuri*. This was discovered by Weichselbaum in 94 out of 129 cases of pneumonia (unfortunately not all lobar fibrinous pneumonia), while Friedländer's was found in only 9. *Streptococcus pyogenes* in 21, and *Staphylococcus aureus* in 5. A distinct rod-shaped microbe was discovered by Klein in the Middlesborough epidemic (Report to the Local Government Board, 1889).

Most pathologists now admit the organism described by Talamon, Gamaleia, Sternberg, and Fränkel to be entitled to the distinctive title of *Pneumococcus lanceolatus*. We certainly find either it, or something undistinguishable from it, in stained specimens of rusty sputum, in hepatised lungs, in pleural effusion, endocardial membrane, and meningeal lymph, when these occur as complications of pneumonia.

As with other specific microbes, we should expect that its chief effect would depend on the chemical poisons it secretes; and toxines have been prepared from it which produce severe symptoms in rodents, but not, it appears, the lesions of pneumonia.

The pneumococcus does not fulfil all the criteria (vol. i, p. 8) necessary to establish its place as the sole and necessary pathogenic antecedent to pneumonia.

Pathology.—The traditional view is that pneumonia is an acute inflammation of the lung, and that the pyrexia and other symptoms are secondary to the local lesion. Long ago, however, doubts were expressed by Skoda and Oppolzer, of this being the true pathology of the disease; and many pathologists now regard pneumonia as a general and specific disease to which the hepatised lung bears the same relation as the intestinal ulcers to enteric fever, or the exanthem to scarlatina. The following considerations bear upon this question, which is far from having only a speculative interest.

Are we right in assuming hepatisation to be an inflammatory process at all? We have seen that it cannot be caused by injury or irritants, and it is doubtful whether it is the direct result of exposure to cold. It does not lead to suppuration on the one hand, or to fibrous induration on the other. The exudation is peculiar, and unlike that of undoubted inflammation which has extended from the bronchial tubes to the air-vesicles; the distribution is remarkable, and the disease never or scarcely ever assumes a chronic course.

On the other hand, the inflammatory nature of the process seems proved by the constantly concomitant pleurisy and occasional pericarditis; as also by the exudation of fibrin and leucocytes along with blood-discs. Moreover, we are familiar with other cases of non-traumatic inflammations which are more or less peculiar to the organs they affect, and which, as they do not originate from ordinary irritants, so fail to produce the ordinary results of inflammation. Such are the tubal nephritis of Bright's disease, the membranous laryngitis of diphtheria, and the synovitis of rheumatism.

If the characteristic lesion of pneumonia may be accepted as a special form of inflammation, it is not to the catarrhal inflammation of mucous, but to the plastic inflammation of serous membranes that it should be compared. The pulmonary alveoli are lined by epithelium provided with lymphatic stomata, not with glands and a vascular mucosa.

It has been asked why the general symptoms are so independent of the severity and extent of the local lesion. But the severity is probably the same, or nearly so, in every case, and the extent is certainly not without influence; double pneumonia is a more serious disease than single, and consolidation of three fourths of a lung than of half its lower lobe.

If, however, the other symptoms of pneumonia are secondary to the pulmonary lesion, why is the temperature so much higher than in other

inflammations, whether serous, mucous, or visceral—higher than in any other disease except specific fevers, tuberculosis, or pyæmia? Why is there albuminuria with cloudy swelling of the renal epithelium, which disappears with the pyrexia, and never leads to permanent local changes? Why are the chlorides so remarkably deficient in the urine? Why do the general symptoms sometimes precede evidence of the local lesion, and often cease a day or two before the local changes disappear.

Again, the course seems too typical for that of a local inflammation. Why does crisis often occur at the end of a week? and why is convalescence so rapid and complete?

Pneumonia, though certainly not infectious in the ordinary sense of the term, sometimes occurs, as we have seen, in epidemics; yet it does not protect from future attacks.

Lastly, the presence of a microphyte, even if not yet certainly pathogenic, suggests a specific origin and character.

At present it appears that, beside the local inflammation, which may be compared with acute nephritis, acute myelitis, and acute yellow atrophy of the liver, pneumonia is also a specific infection of the whole organism; that, however, the same bacillus is not invariably present, so that possibly microbes of different morphology may secrete the same toxins; and that this bacillus being present in the mouth of many healthy persons, may very likely, under the depressing effects of cold, or of influenza or other fevers, acquire new and dangerous powers, and may invade the lung, pleura, meninges, or endocardium. The termination of pneumonia by crises may be ascribed to the gradual development of antitoxines.

Diagnosis.—Acute lobar pneumonia is so well-marked a disease in its symptoms and course, and auscultation gives us such clear and precise evidence of its presence, that once suspected, it can always be discovered; and the disease is too common not to be thought of in any case of pyrexia.

Secondary pneumonia may be overlooked without care and watchfulness; but in primary cases the difficulties lie in its distinction from bronchopneumonia in children, and from acute pleurisy with effusion in either children or adults. In children there is usually no rusty sputum, and often no vocal fremitus; and in adults, as well as in children, tubular breathing may be heard with pleuritic effusion. The symptoms when the pleurisy is due to pneumococcal infection are those of pneumonia, and therefore we have chiefly physical signs to trust to. One help is Wilks' observation, that in passing from the bronchial breathing of hepatisation to the normal sounds of unaffected lung, we traverse a belt of consonating râles, whereas this border is absent in pleuritic effusion. A large effusion would displace the heart, but such a case would be otherwise unmistakable.

Prognosis.—Uncomplicated pneumonia is far less dangerous than its severe symptoms seem to threaten. The average death rate cannot be stated with accuracy; for, in the first place, it varies according to the proportion of primary cases to those which are secondary to some other disease; many of the latter are obviously hopeless from the first. Moreover, the danger of pneumonia is widely different at different ages—children, with rare exceptions, recover from acute lobar pneumonia; and in young healthy adults, of temperate habits, the prognosis is almost as good. But to old people, *i. e.* to all above sixty—or to those who, though not old, are worn out by misery, dissipation, or drink—the disease is

exceedingly fatal.* Nevertheless, from time to time one sees a patient recover even when the circumstances have appeared most adverse. It was probably accidental good fortune by which, of 5 patients above seventy, 3 recovered, and 2 of these were eighty; but perhaps we may regard any life which by reason of strength attains fourscore years as to some extent a selected life.

Among 238 cases collected by the writer of children under ten, only one died to 65 who recovered, and between ten and fifteen only 2 to 43 who recovered, while between fifteen and twenty there were 5 deaths to 43 recoveries. The proportion of deaths increased steadily from twenty years upwards, thus:

Between 20 and 30 there were 19 deaths out of 83 cases.

„ 30 and 40	„ 21	„ 62	„
„ 40 and 50	„ 31	„ 50	„
„ 50 and 60	„ 14	„ 20	„
„ 60 and 70	„ 9	„ 18	„
Above 70	„ 2	„ 5	„

The most important consideration as to the prognosis in a case of primary pneumonia—apart from the age of the patient (which is as important as in enteric fever), and his habits—is the presence of renal disease; this complication makes even a limited consolidation of a single lung in a young and temperate person of the gravest significance. Disease of the heart is, in the writer's experience, far less serious.

Lastly, even in a young and healthy subject, danger is indicated if the local lesions are extensive, *i. e.* if the whole of one lung or if both lungs are hepatised. Experience has taught that it is never safe to speak confidently of the recovery of a patient with pneumonia, however favourable its course may be during the first few days. For what was single may soon become double pneumonia; a change for the worst is apt to occur suddenly; the pulse, though of moderate frequency hitherto, runs up quickly, respiration becomes more frequent and shallow, the strength fails with terrible rapidity, and in a few hours the end may come. Probably the cause of the fatal issue in such cases is the supervention of inflammatory œdema or “acute congestion” in parts of the lungs previously unaffected. Other cases go steadily downwards from the very commencement.

In children above infancy, primary lobar pneumonia is of good prognosis. The less apparently severe cases which appear in rachitic children, or after measles and whooping-cough, are often really catarrhal pneumonia affecting a number of lobules close together, and are more dangerous. Of 120 cases of pneumonia in children Dr Goodhart lost 25.

The most grave complications are pericarditis, and the less familiar endocarditis or meningitis; next general bronchitis, and delirium. Very high temperature is sometimes fatal; in 6 of the writer's 434 cases it was over 106°, and four of these were fatal. Symptomatic albuminuria, herpes, hæmoptysis, icterus, or sweating are not of unfavourable import.

Of 362 hospital patients of all ages 86 died (nearly a fourth); of 73 private cases 25 (more than a third); but the latter were all seen in consultation, and therefore were mostly severe. The mortality among 1060

* Of 37 cases of pneumonia in persons reported as intemperate, 15 died, or about 2 in 5; of 228 reported “temperate,” 42 died, or less than 1 in 5; and of 80 total abstainers, 9 died, or little over 1 in 10 (‘Collective Investigation Record,’ vol. i, p. 95). The third class would include a large proportion of children, and the first many elderly people.

cases published in the 'Collective Investigation Report' was less than a fifth (191 in 1060), in Stockholm 281 in 2618 (Huss), and at the Middlesex Hospital 192 in 1010 (Coupland). Of 55 cases of double pneumonia observed by the writer 30 were fatal and 25 recovered; and here it is observable that only one patient above forty recovered.

To sum up:—secondary pneumonia is much more dangerous than primary; with primary pneumonia the danger increases with the patient's age and with the extent of lung involved; and in all cases intemperance adds greatly to the danger.

Treatment.—Pneumonia has been the battle-field of therapeutics as well as of pathology since it was described clinically by Laennec and anatomically by Rokitansky.

From 1820 to 1840 antiphlogistic treatment was vigorously applied to this, the typically sthenic inflammation in a young and healthy subject. In France especially *la saignée coup sur coup* was practised in the hope and belief of "jugulating" the disease. In England twenty ounces of blood and more were often abstracted from the arm, time after time, or the patient was bled in the recumbent posture, so as to obtain a larger flow before syncope occurred. Any failure was ascribed to want of early and bold venesection.

Mercury, usually in the form of calomel, and combined with opium, was given as a necessary adjunct to venesection, particularly in England. Antimony was often conjoined with mercurials, and in Italy it was given in large and nauseating doses as a specific remedy for pneumonia.

A great revulsion of practice occurred in this country in the sixth decade of the last century against "antiphlogistic" and in favour of "corroborant" treatment of inflammations, and of acute diseases generally. Wine and brandy, often in "heroic" doses, were prescribed in pneumonia under the influence of the late Dr Todd.

In Germany Skoda, then at the head of the Viennese school, had learned to distrust large and systematic depletion, although he still bled in the earlier stages of pneumonia, and believed in the power of drugs to defibrinate the blood and cut short the disease. There was a remarkable contrast between the treatment of pneumonia in his wards, as witnessed by the writer in the year 1865, and in those of Bouilland during the preceding winter, where repeated bleeding was still the rule. At the same time cases of pneumonia were being "cured" at the Homœopathic Hospital in Vienna by infinitesimal doses of useless drugs.

Meantime some physicians, among whom Hughes Bennett of Edinburgh and Austin Flint of Philadelphia deserve the foremost place, had been observing the natural history of pneumonia, and had found that, at least in young and temperate patients, its danger had been much over-estimated, that it tended to recovery after a week's duration, and that neither bleeding, nor mercury, nor antimony, nor alcohol was necessary in favourable cases, or could be depended on to save life in unfavourable ones.

For many years past the treatment of pneumonia in England has been on the same plan as that of typhus, enteric fever, or scarlatina—expectant, not in the sense of doing nothing for the patient, but of putting him in the best circumstances for recovery when the malady has run its course, and watching meantime for any unfavourable symptom, so as to meet it when it appears.

Of late, however, it has been asserted by some German physicians that better results can be obtained by vigorous antipyretic treatment, like that which has been described in the chapter on enteric fever. At Basle cold baths have been used whenever the temperature rose a little above 102° Fahr. Jürgensen advised that 104° should be the point at which baths should be systematically employed; while for patients whose temperature ranges at from 101° to 103° he merely ordered a tepid bath in the morning, so as to increase the normal remission during the early part of the day. He also administered quinine in doses of thirty grains at intervals of forty-eight hours. The theoretical basis of this practice is that the great danger of pneumonia is failure of the heart, as the result of the combined action of pyrexia and disturbance of the pulmonary circulation. In proof of its efficacy Jürgensen adduced a tabulated statement of the fatal cases that occurred in his practice, showing that scarcely any of his patients died except such as had some dangerous complication. He prescribed wine as a stimulant to the heart before each cold bath; and when signs of syncope appeared, he gave alcohol freely, as well as camphor and musk.

Few English physicians, however, believe that pneumonia can be jugulated by cold baths any more than by bleeding, or that moderate pyrexia is so injurious that it must be persistently attacked. Even if cold baths are harmless, there is reason to believe that antipyretic drugs are as liable to do mischief as the antiphlogistic treatment which they now supersede. A careful trial of antipyrin in a series of cases of acute pneumonia led the late Dr Botkin, of St Petersburg, to the candid avowal that it was doubtful if it ever did good, and certain that it often did harm.

The present writer has more than once employed venesection in what seemed a suitable case without preventing death; has used cold baths to check pyrexia without doing any harm, but with doubtful effect on the course of the disease; has given aconite from the beginning without in the least altering the rise of temperature or averting serious complications, and has seen quinine, antimony, wine, and brandy, all in turn prove useless to check the progress of the disease.

The rationally expectant treatment of pneumonia, as generally carried on in hospitals and in private practice in London, is somewhat as follows. On the first rigor and rise in temperature the patient is put to bed, kept undisturbed, and his strength husbanded. The worst cases are those in which the patient has kept up during the first day of illness, or when some unhappy street outcast has walked about with the disease upon him until brought exhausted to a hospital.

The room must be kept comfortably warm. The patient must not talk, and must not sit up in bed. A single calomel purge does no harm, and has the advantage of preventing the exhibition of physic afterwards, when the patient is less able to bear its effects.

The diet should consist of beef-tea, milk, and arrowroot or gruel, with tea, fruit, jelly or ice, if desired, and a free supply of water, effervescing drinks, or any harmless form of diluent. A cup of tea is harmless and refreshing, and strong coffee is sometimes a useful stimulant. Nitre, citrate of potash, or acetate of ammonia are usually prescribed, and probably are of real service as diuretics, and by promoting the solution and excretion of inflammatory products towards the close of the disease; but they need not be insisted on if (even when suitably disguised) they are refused by a child.

When pleuritic pain is present, it may often be relieved by poultices, a mustard plaster, or, according to some authorities, by a blister; but in really severe pain nothing gives so rapid and complete relief as a few leeches. In aged or feeble patients we must trust to hot applications and morphia injections. When cough and expectoration are difficult, ipecacuanha with paregoric and squills is indicated. If there is continued want of sleep, ten grains of Dover's powder is probably the most efficient remedy, and mere symptomatic albuminuria does not counter-indicate it. If, however, there is any doubt as to the efficiency of the kidneys, it is safer to give chloral hydrate or hyoscyamine.

If the temperature rises over 104° in the case of an adult, cold sponging should be employed; and if this is ineffectual, and delirium is present, Leiter's coils, or the wet pack, or ice-bags under the armpits are probably safer than the cold bath. Nevertheless, if the heart is sound and the patient young, a bath may be used. Dr Lees published some good results of treatment with ice-bags in the 'Lancet,' vol. ii, for 1889, and Dr Goodhart recorded his own experience of the treatment of pneumonia by cold applications in the 'Guy's Hospital Reports' for the same year.

If cyanosis appears, with signs of dilatation of the right side of the heart—a small, frequent, and feeble pulse, epigastric pulsation, distended and pulsating jugular veins—the abstraction of six, eight, or even ten ounces of blood from the arm will relieve the pressing symptoms and perhaps do permanent good. Wet or dry cupping between the shoulders is generally less effectual.

In the case of children, writes Dr Eustace Smith, where there is great dyspnœa and threatened cardiac failure from over-distension of the right side of the heart, life may often be saved by taking one, two, or more ounces of blood from the arm, according to the age of the patient. "I can look back," he continues, "to some fatal cases which I now believe might have been saved had I had the courage to relieve the labouring heart by judicious removal of blood."

The greatest danger in pneumonia is not from the fever nor from the pulmonary obstruction—it is from failure of the heart. To meet this, digitalis may be given with advantage, but it is less effectual than in cases of valvular disease, partly from the pyrexia, and partly from the degeneration of the heart muscle. Digitalis in large doses has been strongly recommended by Dr Pétresco of Bucharest, and the slowing of the pulse showed that the drug was genuine. His results are remarkable, and the practice deserves further trial. Ammonia, with or without senega, ether, and Hoffmann's anodyne (Sp. *Ætheris co.*) are all valuable drugs. But the most important means of meeting either an obstructed pulmonary circulation or a directly failing left ventricle are, first, the subcutaneous injection of strychnia; and secondly, brandy in measured doses, but increased if necessary up to ten or twelve ounces in the day. Children and young adults seldom need it, but there are few patients above forty, and probably none above fifty, who do not require stimulants in larger or smaller quantities from very early in the disease. Sometimes port wine or champagne suits better than brandy; the kind of stimulant may be decided by the patient's feelings, but the quantity and frequency must depend on the state of the pulse and the first sound of the heart.

After the temperature has fallen, unless empyema follows, the convalescence of a pneumonic patient is uninterrupted. He may be allowed to

eat as his appetite returns, and to go abroad when he feels able to. There is no danger of phthisis or other chronic affections following. In this respect it resembles typhus, and differs widely from enterica, as also from pleurisy and from broncho-pneumonia.

If, as most pathologists believe, pneumonia is a specific fever due to the *Pneumococcus lanceolatus* above described (p. 15), it is natural to hope for effectual specific treatment either by inoculation with an attenuated cultivation of this microbe, as in the case of variola and hydrophobia, or more probably by injection of antipneumonic serum from animals rendered gradually immune by previous injection, as in the case of diphtheria. Drs G. and F. Klemperer have prepared such serum, and used it in Germany, and Dr Washbourn has done the same in England. The writer has used this plan of treatment in several cases, but with inconclusive results. When the attack has been slight, the patient's recovery may have been independent of the remedy; when it has been severe, his death may have been inevitable under any treatment. It does not appear as yet that others have more decisive results to report on a large number of cases. My colleague, Dr Pitt, has recently tried a serum prepared by Dr Pané, of Naples, who has had some success with it in that city; but its use in Guy's Hospital has not been followed by decisive results at present.

Secondary forms of pneumonia.—We have seen how the course of pneumonia differs according to the age and habits of the patient, and according as the heart and the kidneys are healthy or the reverse. Another important practical distinction is between primary pneumonia, with or without complications, and pneumonia secondary to other diseases.

The anatomy of secondary pneumonia is the same as that above described, but its symptoms are much more obscure; so that for its recognition we must depend on a moderate rise of temperature, increased rapidity of breathing, slightly blood-stained sputa, and physical signs—which must be sought for. Its prognosis is much more unfavourable than that of primary cases, and it usually brings about a fatal issue in the fevers and chronic diseases in which it occurs.

In *rheumatic fever* pneumonia is not common, but when it occurs it is a very grave complication.

In the course of *Bright's disease*, pneumonia is less common than inflammatory oedema, but when it occurs it is very apt to be fatal.

In *disease of the heart* pneumonia may follow pulmonary hæmorrhage—a condition in which the air-vesicles are filled with blood, and a patch of lung becomes solid, and dark red on section. Hepatisation often takes place around the wedge-shaped mass of solid lung, but it does not spread far, and the symptoms are not severe.

In *diabetes*, pneumonia is a very fatal complication. It often comes on in the latter stages of the disease, and passes rapidly into grey hepatisation, with a lower temperature than usual.

Alcoholic pneumonia, or that which is found associated with delirium tremens, is peculiarly prone to a rapid and fatal issue.

In typhus, enterica, and other *fevers* ordinary lobar pneumonia may occur; but far more frequently a condition of the lungs is recognised during life, and verified after death, which has been called "typhoid pneumonia," or, more properly, *hypostatic broncho-pneumonia with congestion*. It is found occupying not the bases, but the backs of the lungs—the lowest part

as the patient lies in bed ; it almost always affects both lungs more or less ; it is not continuous, but consists of patches of airless granular tissue, including several lobules, and surrounded by congested and œdematous but crepitant lung (cf. vol. i, p. 120). The pneumonia is of the catarrhal kind, not massive hepatisation. It is not accompanied by the high temperature of primary pneumonia nor by its other striking characters, and yields imperfect resonance rather than dulness, toneless or subcrepitant râles rather than true pneumonic crepitation. When recognised, it shows prostration in general, and weakness of the cardiac muscle in particular, and calls for ammonia and for brandy.*

Inflammatory œdema and acute pulmonary congestion.—Dropsy of the lung is not infrequent in the course of Bright's disease ; and like œdema of the glottis, it is probably always in some degree inflammatory. But apart from these cases there is an œdematous inflammation of the lungs which comes on suddenly, either in the course of acute nephritis or pneumonia, or occasionally without previous disease. It has been described as acute pulmonary congestion, and may or may not be identical with the first stage of hepatisation (p. 9). A case is narrated by Hertz in 'Ziemssen's Handbuch,' and another by Dr Leuf in the 'American Journal of Medical Science,' January, 1885. Such marked and severe cases are rare, but many intelligent observers believe that "active congestion" of the lungs, distinct from pneumonia and from tuberculosis, is a real and separate disease. In France it is known as "maladie de Woillez."†

Chronic lobar pneumonia.—We have seen that one peculiarity of acute pneumonia is that, when the patient recovers, the hepatised lung soon clears up completely, and the tissue retains no trace of the process it has passed through. To this, as to every pathological rule, there are occasional exceptions:—abscess following pneumonia is so rare that its very existence is doubtful ; gangrene only ensues under special conditions, which will be discussed further on ; and phthisis does not originate in acute lobar pneumonia. But Addison described certain very rare cases in which, after an illness of a few weeks, with evidence of pneumonic consolidation, the patient dies, and the lung is found uniformly "albuminised." The section is not soft, lacerable, and granular, as in acute grey hepatisation, but smooth, solid, and tough. The alveoli are full of fibrillated lymph, and the exudation cells may have begun to be granular and fatty. It is a condition of the lung very liable to break down, and thus lead to rapid phthisis ; on the other hand, it is a condition from which recovery may take place, and the lung be completely restored.

Wilks and Moxon, after quoting this account, continue, "That there is a chronic pneumonia of such a kind can scarcely be denied when it is remembered for how long a time all the signs of consolidation may endure, and then a complete restoration take place. We must therefore believe that there is a true chronic pneumonia, whose origin is an ordinary inflammation and exudation into the alveoli, and whose appearance is best denoted by the term 'uniform albuminous induration.'"[‡]

* It is in this sense of hypostatic congestion that Arbuthnot's dictum applies: "A peripneumony is the last fatal symptom of every disease, for nobody dies without a stagnation of the blood in the lungs" ('Of Diet,' chap. iii).

† See on this subject Hodgkin's remarks ('Lectures on Morbid Anatomy,' vol. ii, p. 129).

‡ 'Pathological Anatomy,' p. 338. Addison's original description appeared in the

They add, however, that such chronic pneumonia may be of the lobular kind, and Addison himself said that this condition may be limited to one or a few lobules only; so that it may perhaps be doubted whether this "least frequent of the permanent pneumonic indurations of the lung" is not, after all, an unusually extensive catarrhal pneumonia, which may clear up or go on to cirrhosis, or if tuberculous in origin, end in phthisis.

BRONCHO-PNEUMONIA.*—In young children, acute bronchitis affecting the smaller bronchial tubes is often accompanied by inflammation of the pulmonary alveoli, usually of both lungs. Ziemssen, out of ninety-eight cases, found that sixty-seven—and Steffen, out of seventy-two cases, found that fifty-two—occurred in patients under three years old.

Ætiology.—In many instances broncho-pneumonia is secondary to some infective disease. Measles and whooping-cough furnish by far the largest number of cases; and it may follow diphtheria, influenza, smallpox, or scarlet fever. Liability to broncho-pneumonia, as a complication of bronchitis, is greatly increased by the impure air of close rooms; and we may trace the greater frequency of the affection in winter to defect of ventilation rather than directly to inclemency of season. It is probable that rickets renders a child more apt to become affected with broncho-pneumonia if it is attacked with bronchitis. In the 'Guy's Hosp. Rep.' for 1860, Wilks showed that broncho-pneumonia is a common cause of death in children suffering from severe burns.

As to the origin of the exceptional cases when broncho-pneumonia like that of infants occurs in adults, very little is known. The following cases recorded by Dr Fagge seem to have been septic in origin.

A woman, aged thirty, having miscarried eight days before, was attacked with shivering and headache, and died after an illness that lasted ten days.

A man, aged twenty-five, of dissipated habits, who was said to have been under a course of mercurial treatment for syphilis, received a blow on the nose while he was drunken; this was followed by epistaxis, which continued until the nares were plugged. The mucous membrane then suppurated profusely, the discharge being very foetid; and he sank and died at the end of a week.

A third case was that of a man aged twenty-eight, a singer at a music-hall. He had a fall from a cart, and this led to an illness which proved fatal in three weeks. At first he tried to go on with his singing, but he was soon obliged to give it up. When admitted, shortly before his death, he was already comatose.

In this instance some of the patches showed central sloughs, but in the reports of the two other cases it is expressly stated that the appearances were identical with those that are commonly seen in children. It seems probable that in the second case the inflammation was set up by the inhalation of foetid pus from the nasal cavities into the air-passages.

We must of course exclude cases of broncho-pneumonia dependent on the micrococci of suppuration (pyæmic), or the bacillus of tubercle, or on the entrance of food or other putrescible material leading to gangrene.

Breathing irritant gases, especially chlorine, sometimes sets up intense broncho-pneumonia. It is not improbable that the immediate cause of the ordinary broncho-pneumonia of children is the entrance into the alveoli of inflammatory products formed in the smaller tubes, as the result of violent inspiratory efforts.

Broncho-pneumonia is not infrequent in extreme old age, and it often ends the life of those who have long laboured under senile bronchial catarrh.

'Guy's Hospital Reports' for 1843, and will be found in the volume of his collected papers published by the New Sydenham Society, p. 27.

* *Synonyms.*—Catarrhal pneumonia—Lobular pneumonia—Pulmonary catarrh.

Indeed it is probably identical with much of what is still called "capillary bronchitis."

Clinical course.—The presence of pulmonary catarrh, in addition to the acute bronchitis which precedes and gives origin to it, is usually rather inferred than proved. Physical examination of the chest often helps but little. If several lobules side by side beneath the pleura are consolidated, there may be impairment of resonance (or even dullness) on percussion, bronchial breathing and bronchophony, the latter being especially obvious when the patient, if a child, is crying; moreover the bronchitic râles may become consonating in quality. Sometimes a crepitating râle is heard, like that of acute pneumonia, but coarser, and audible during expiration as well as inspiration.

Nor, again, is broncho-pneumonia attended by very marked symptoms. A sudden rise of temperature may indicate its supervention in the course of acute bronchitis, if the pyrexia has previously been moderate. But the thermometer may indicate 104° or even higher in a child affected with bronchitis, independently of any complication (vol. i, p. 1053).

Jürgensen says that broncho-pneumonia—as, for example, after measles—may be accompanied with a temperature of 105.8° , with scarcely any remissions. In fatal cases the temperature sometimes rises before death, and may reach 107° or 108° ; but sometimes it falls below normal. The pulse is often extremely rapid, so that it cannot be counted at the wrist; but in children a pulse rate of 150 to 200 is not incompatible with recovery. It is surprising, too, how hurried the breathing may be in cases which ultimately do well. What is really alarming is for the pulse to be feeble and "running." When the disease ends in recovery, the pyrexia and the other symptoms gradually subside (*lysis*); there is never a sudden fall of temperature (*crisis*).

Herpes on the lips seldom accompanies pulmonary catarrh. Albuminuria and diarrhoea are not infrequent. Often with the supervention of broncho-pneumonia the cough from which the patient had been suffering ceases, or becomes less loud and shorter. Usually no expectoration makes its appearance. There is extreme restlessness, the child tossing about in bed, and asking to be taken up by its nurse and shifted in position every few minutes. The breathing is often shallow, and the face and lips are apt to become pale, with perhaps a livid blush upon the cheeks. There is on the whole more dyspnoea and distress than in cases of lobar pneumonia in children.

Anatomy.—When pneumonic hepatisation has arisen by extension from the bronchial tubes it is not found in large continuous areas, but in scattered patches throughout both lungs; it is very seldom confined to one. These patches are more or less rounded in form; they are usually of about the size of peas, but sometimes may be larger. They are reddish brown in colour, or more or less grey, or greyish yellow, according to the stage to which the inflammation has advanced. As seen upon the cut surface of the lung, they appear slightly raised above the rest of the parenchyma. They have a dull, lustreless appearance, and are granular on section, but the granulations are less obvious than in the red hepatisation of lobar pneumonia. The substance of the patches is soft and friable, and when squeezed they emit a more or less opaque fluid. They are sometimes so closely collected together in part of a lung that a continuous area may seem to be consolidated; but even then the lobular markings can be distinguished, and

the cut surface has not that even and uniform appearance which characterises true lobar hepatitis.

Many lobules are found collapsed, smooth, airless, and depressed below the surface without any granular appearance. This lobular carnification was formerly confounded with lobular hepatitis. The conditions are distinct in origin and nature, but they often exist together (cf. vol. i, p. 1056).

Vesicular pneumonia was a name used by Stokes, and applies to the first stage of pulmonary catarrh. In all probability it soon becomes lobular, but occasionally it may be seen after death as a series of minute dots over the cut surface of a lung, too small for lobules, though too large for single vesicles, and these are found to contain catarrhal products. The same appearance to the naked eye may, however, be produced by cross-section of small bronchial tubes, each surrounded with peribronchial inflammation.

Pleurisy is often associated with pulmonary catarrh, though not so constantly as with pneumonia. A thin layer of lymph may be seen over any patches that happen to lie just beneath the pleura. Indeed, both at the bedside and in the deadhouse it is a fact that, altogether apart from the presence of pneumonia, lobular or lobar, pleurisy is a far from infrequent complication of bronchitis; in adults affected with bronchial inflammation a pleuritic friction-sound may often be detected if it is listened for.

Histologically, the morbid process in broncho-pneumonia is a *catarrhal* inflammation; that is to say, the cells which fill the affected alveoli are many of them epithelial in character, large, of irregular shapes, with bold nuclei. With them are found leucocytes and mucus, but neither blood-discs nor fibrin. Probably the seat of the disease is the intra-lobular air-passages, and the contents of the vesicles have been carried thence.

In the air-vesicles and smallest bronchioles may be found various microbes, streptococci, staphylococci, and sometimes an encapsuled diplococcus. But none of them are constant, and probably none are specific.

In some cases the inflammation appears to spread to the connective tissue by which the pulmonary lobules are united together. Jürgensen speaks of "thick whitish-grey bands, which are seen crossing one another upon the cut surface of the organ." In 1878 a well-marked instance occurred in a child aged three, who died in Guy's Hospital after an illness of five weeks, which perhaps began in whooping-cough. The left lung contained many patches of broncho-pneumonia, and the right was almost entirely consolidated. Its tissue, however, felt hard, and the fibrous tissue had obviously undergone a great increase. Such cases seem to afford an explanation of an appearance not infrequently seen in autopsies, marbling of a part or the whole of a lung by fibrous bands which intersect one another and divide it up into irregular areas. This condition in an adult is often regarded as early cirrhosis; but it is probably sometimes the residue of a former attack of pulmonary catarrh.

Diagnosis.—Broncho-pneumonia sometimes gives rise to cerebral symptoms like those of *tuberculous meningitis*. Delirium, coma, retraction of the occiput, vomiting, strabismus, convulsions, may each appear in these cases.

Another difficulty is to distinguish pulmonary catarrh from *acute tuberculosis* of the lung; or, rather, to determine whether a condition of broncho-pneumonia is idiopathic or produced by the invasion of the tubercle bacillus.

Broncho-pneumonia in children may be mistaken for *enteric fever*. Rose

spots are not constant in children; diarrhœa is frequent in broncho-pneumonia, and often absent in enterica; but a full abdomen and a swollen spleen point to the latter, and the curve of temperature, with its relation to the respiration, will generally decide the question.

Perhaps the most frequent difficulty is to decide between pleuritic effusion—that is, as a rule, in a child, between *empyema*—and pulmonary catarrh. In both cases there is cough, dyspnœa, dusky face, clubbed fingers, and pyrexia. In both cases râles and rhonchi may be heard, and more or less extensive dulness on percussion. Over the dull space tubular breathing may be heard in each case, the condition of tactile fremitus may be difficult to obtain, and the vocal resonance may be absent. In this more than in any other state of the lungs is it allowable to depend on the aspirating needle to decide a point of great practical importance, where the utmost care and experience in physical diagnosis often fails to decide the question.

Prognosis.—The ordinary duration of broncho-pneumonia is about a fortnight; but sometimes it runs on for three or four weeks or even longer. It may destroy life very rapidly—within a few days, or even in twenty-four hours; but sometimes death occurs with extreme wasting, after all acute symptoms have passed off. The disease is said to be fatal in from half to two thirds of the cases; but when consecutive to measles the risk is smaller than this. Convulsions are of bad omen. Very young infants are far more likely to die than older children; and the prognosis is also more serious in those who are ill-fed or rachitic. In children it is far more dangerous a disease than lobar pneumonia. Of forty-five cases of acute broncho-pneumonia in children, recorded by Dr Goodhart, twenty were fatal.

Treatment.—Emetics do good service in the first few days; afterwards they are too depressing. Expectorants are useful, but opium must be avoided. The ammonia and senega mixture is almost always valuable; and sweetened with glycerine or treacle or syrup of tolu it will be taken by children as well as other medicines. Dr Eustace Smith recommends dry cupping of the back in bad cases. Diarrhœa should be checked by chalk powder. Stimulants are almost always necessary—sweetened brandy and water, or the egg-and-brandy mixture in half-drachm doses.

During convalescence, which is often long and tedious—contrasting with that of lobar pneumonia—quinine and iron are indicated, with cod-liver oil. It is desirable to remove the child to the seaside as soon as practicable, and to have it as much as possible in the open air.

When catarrhal broncho-pneumonia becomes chronic, the lung is very liable to tuberculous infection. This is the ordinary beginning of phthisis, and will be discussed in a future chapter.

Septic pneumonia.—The term “lobular pneumonia” has been applied to the suppurative process which attacks the lungs in pyæmia. It is lobular in distribution, but is neither catarrhal, caseous, nor fibrinous, but purulent; and it never occurs except as part of a general process of embolism and septicæmia. The modern doctrine of pyæmia was to a large extent worked out by Virchow and Cohnheim in the case of the pulmonary circulation.

When pulmonary embolism is non-infective, it produces hæmorrhage by the same mechanism as in the case of the brain; but there is no reason to suppose that this is the only cause of so-called pulmonary “apoplexy.” Under extreme congestion from obstruction on the left side of the heart the capillaries of the alveoli may give way; or extravasation may occur from

changes in the vessels, or in the blood itself, as in cases of purpura hæmorrhagica. When the embolus is infective it produces intense congestion, and soon after suppurative inflammation. The abscesses (or hæmorrhagic congested patches) are multiple, and are most numerous in the back part of the lung, and generally near the surface.

The symptoms of this form of lobular pneumonia are merged in those of the fatal disease of which it is a part, but its presence may be suspected when a case of pyæmia is complicated by pain and other signs of pleurisy, which is caused by an abscess reaching the surface of the lung.

CIRRHOSIS OF THE LUNG.*—This term was first applied in 1838 by Sir Dominic Corrigan to an affection of the lung, in which its alveolar structure is replaced by a fibrous material resembling that of cirrhosis of the liver ('Dublin Journ. of Med. Sci.,' vol. xiii, p. 266). He described the dilated bronchi, the dark grey colour, the contraction, which he compared to that of a scar after a burn, and the displacement of the heart and liver; and he drew a clear distinction between this disease and phthisis.

The mere excess of fibrous tissue in the lung is not characteristic of any one morbid process. The condition which has been termed "fibroid phthisis" is nothing but a regressive stage, or a very chronic form, of a really tubercular process. The subject will again come before us when we deal with phthisis and pneumoconiosis.

There remain cases which are not tubercular, and never have been. In most of these, while only one lung is diseased and shrunk into a hard grey mass, the other one is often perfectly free; whereas in phthisis, long before one lung is destroyed, the other becomes involved in the disease.

Again, in Corrigan's disease, when one part of a lung is affected before the rest, it is usually the lower lobe. In each of five cases of double cirrhosis observed at Guy's Hospital the bases of both lungs were found to have undergone cirrhosis; in none was there any indication of the presence of tubercle. These cases are usually complicated by pleuritic adhesions on the affected side, and often by bronchiectasis.

Anatomy.—The process begins, according to some pathologists, in peribronchitis, which spreads from the smaller tubes to the elastic tissue surrounding the alveoli; according to others, in a chronic inflammation of the subpleural and interlobular connective tissue. Both origins, however, are denied by others, and Wilks and Moxon state decidedly that the process starts in the walls of the alveoli. Of cirrhosis affecting one lung only, with thickened pleura and dilated tubes, the same authors say that sections of the lung show not only fibrous tissue surrounding the bronchial tubes and blood-vessels, but also thickening of the air-sacs. This is well shown in a drawing given by the late Dr Coats, of Glasgow (fig. 231), in which the alveoli are represented as lined with large nucleated epithelioid cells, quite unlike those of the normal lung—a condition also represented in a drawing by Heschl, reproduced in the late Dr Wilson Fox's article in 'Reynolds' System,' p. 765. When the "fibroid" (or fibrous, for it is true connective tissue) degeneration has reached its full development, the lung on section exhibits large tracts of iron-grey, slate-coloured, black, or marbled tissue, which is completely airless, firm, and sometimes of cartilaginous hardness. The microscope shows only dense fibrous tissue, with no trace remaining of the pulmonary structures which it has displaced.

* *Synonyms.*—Chronic interstitial Pneumonia—Iron-grey consolidation of Addison—Induration ardoisée of Cruveilhier—Induration grise et mélanique of Andral—Fibroid lung.

Origin.—This chronic fibroid induration is an important conservative process in phthisis, few cases of which are entirely without some cicatricial induration; but as a process independent of tubercle its pathology is still a matter of uncertainty; possibly it has more than one mode of origin.

(1) Now and again it would seem to have begun in an attack of acute lobar *pneumonia*. This question has been already touched upon (p. 22). No doubt, as a rule, acute pneumonia, if it does not prove fatal, undergoes resolution, and does not lead to chronic induration of the lung. Still, the evidence on which this opinion rests can only be the observation of many cases, in none of which such a result was noted. In hospital practice it rarely happens that a patient comes under the eye of the same physician during an acute illness, and again, months or years afterwards, when a chronic affection of the lung proves fatal. Moreover medical literature contains some carefully recorded cases in which the origin of cirrhosis in pneumonia appears clear.

The two following are taken from Dr Bastian's collection of thirty cases, tabulated in the second volume of 'Reynolds' System.' The first was taken from Charcot's 'Thèse de Paris.' The patient, a man aged sixty-one, was admitted on March 30th, 1850. He had been attacked five days before with rigors and pain in the side, and had rusty sputa. There were all the signs of pneumonia affecting the whole of the right lung. These continued with but little alteration until his death on July 19th. At the autopsy the right lung was of a greyish-blue colour on section, as hard as cartilage, shrunken to two thirds its natural size, and enveloped in an immensely thickened fibrous mass.

The other case was recorded by Dr Mayne ('Dublin Hosp. Gaz.,' May, 1857). It is that of a man aged fifty-four, who in July, 1855, after a hard day's work, was seized with rigors and all the symptoms of pleuro-pneumonia. The acute disease subsided, but he never afterwards regained his health and strength. In October, 1856, he was attacked with fresh febrile symptoms, and he died at the end of the year. The lung after death was found affected with well-marked cirrhosis.

Dürr has recorded, in a volume published by Jürgensen, two cases which occurred in children. In each of these the primary attack appeared to be one of lobar, not catarrhal, pneumonia; but probably the "pneumonia" which leads to cirrhosis is, as a rule, lobular and catarrhal, and in children it is often secondary to measles or whooping-cough.

To this origin in alveolar catarrh Dr Fagge was disposed to refer one variety of the affection, in which, instead of part of the lung being all converted into a fibrous substance, it is intersected by bands crossing one another, so as to split it up into areas of various sizes and shapes. This appearance has been supposed to represent an early stage of cirrhosis, such as might, if the patient had lived, have involved the whole lung. But may it not be rather a residue or relic of a former acute process? Many pathologists who hesitate to admit the origin of pulmonary cirrhosis in true lobar pneumonia would readily allow the probability of so much more chronic, more irregular, and, so to speak, more irritative a process as lobular or alveolar catarrh being the antecedent of iron-grey cirrhotic induration.

(2) A frequent starting-point of cirrhosis of the lung is *pleurisy*. In most cases the pleura over the affected part of the lung is adherent to the parietal layer, and the two together form a dense white mass, of cartilaginous hardness, and from a quarter of an inch to an inch in thickness. There is also a similar thickening of the pleural layers separating the different lobes. This looks as if the lung substance were invaded from the surface by extension along the interlobular septa.

A case showing how easily erroneous conclusions may be drawn, even from *post-mortem* observations, occurred at Guy's Hospital in 1877. A man aged twenty-seven died of dis-

ease of the left side of the chest, with a history of having had inflammation of the lungs at the age of fifteen, and some thoracic affection even before that, in early childhood. There was a localised empyema in front, and the pleura elsewhere was thickened in places to the extent of an inch. The lung was very small, but its tissue was generally healthy, except that it was intersected by fibrous bands. It would have been natural to infer that the organ had been invaded from without. But part of the other lung was affected in a precisely similar way, although the pleura covering it was in a normal state.—C. H. F.

(3) *Dilatation of the bronchial tubes* in the affected lung or part of a lung is present in most cases of cirrhosis. In fact, Jürgensen and Grainger Stewart discussed the two conditions together. But various forms of bronchiectasis occur without there being any change in the pulmonary tissue, unless it be emphysema. And in six of Dr Bastian's thirty collected cases of cirrhosis it is expressly stated that the tubes were of their natural size. As to the relation between the bronchial dilatation and the cirrhosis, when they co-exist, there are differences of opinion. Some think that a chronic inflammatory process may start from the smaller tubes, and lead to a gradual fibrous thickening of the alveolar walls, with obliteration of their cavities. But the very definite localisation of the morbid process, the completeness of the destruction of the pulmonary texture, and the fact that the pleura becomes so greatly thickened, are arguments against this view. Corrigan suggested that the bronchiectasis was secondary to the cirrhosis; he supposed that the contraction of the adventitious fibrous material in the lung dragged upon the walls of the tubes, so as to widen their channels. Wilson Fox, however, believed that if the dilatation of the tubes really follows the cirrhosis, it is caused by the expiratory force of coughing (vol. i, p. 1063).

On the whole, it appears likely that some cases of cirrhosis begin with catarrh of the pulmonary alveoli, which may sometimes have arisen from an illness that occurred in childhood and had been forgotten. In others the process may have begun in peribronchitis, in others in the pleura, and in some it may perhaps be a true primary chronic inflammation of the lung.

Incidence.—Cirrhosis is most apt to occur in adult men. In Sutton's 34 cases ('Med.-Chir. Trans.,' vol. xlviii, 1865) only one occurred in a woman; in Wilson Fox's collection 22 men to 16 women. In Bastian's list (collected for 'Reynolds' System') there were 24 male and 6 female patients; and of these 2 were only seven years old, 3 were between sixteen and twenty, 21 between twenty-four and fifty-seven, and 4 between sixty-one and seventy-one.

In 1887 the writer had a typical example in Mary Ward, in a boy seven years old, and during 1897-8 two of unilateral cirrhosis in boys of ten and fourteen; but with these exceptions all his patients have been adult, and almost all between thirty and sixty. Among 112 cases collected, 11 occurred in patients under twenty years of age, 18 between twenty and thirty, 24 between thirty and forty, 13 between forty and fifty, 24 between fifty and sixty, 8 between sixty and seventy, and 4 above seventy.

Symptoms.—Clinically, cirrhosis varies according to its extent. If it affects a portion of one lung, the patient gradually becomes a chronic invalid, with cough, dyspnoea, and wasting. The expectoration is purulent, sometimes dirty grey, and apt to be offensive; the fingers become clubbed, and there is more or less marked emaciation. The physical signs are those of partial consolidation of the lungs, with the addition of more or less abundant râles. Such cases are not uncommon in hospital practice. After staying a certain number of weeks in the ward, the patient is discharged much as he came in, or at best with some relief to his symptoms. In five

cases of cirrhosis of the bases of both lungs, the cause of death was either unconnected with the pulmonary affection, or it was an attack of acute pneumonia, or lardaceous disease of the kidneys, the result of the protracted suppuration.

If cirrhosis involves the whole of one lung, the affected side of the chest falls in, so that it measures in each direction much less than the other side. The opposite lung becomes emphysematous, and the heart is much displaced toward the affected side. In such cases there is often difficulty in distinguishing the disease from chronic pleurisy, or empyema with retraction after paracentesis. According to Walshe, there is in cirrhosis less twisting of the ribs on their axes; nor is the shoulder lowered so much, nor is the inferior angle of the scapula tilted so far outwards, as in pleurisy.

From a malignant tumour cirrhosis is distinguished by the state of the mediastinum. This is pushed away by the growth of a tumour, and dragged over by the progress of cirrhosis.

If the right lung is cirrlosed, the heart is seen beating at the right nipple; if the left, its visible pulsation may extend upward nearly to the second space. In either case the opposite lung enlarges, so that the whole region, and even the space beneath the costal cartilages on the affected side, becomes resonant on percussion, and transmits to the ear a loud vesicular murmur. After a time, however, this emphysematous and perhaps hypertrophied lung fails to carry on respiration efficiently. The right side of the heart becomes dilated, a tricuspid regurgitant murmur is heard at the ensiform cartilage, the patient suffers from permanent orthopnoea, and ascites and oedema set in. The case, in fact, assumes all the characters of chronic valvular disease of the heart, or of the most protracted cases of "fibroid" phthisis, and terminates in the same way.

In the absence of a history or of the scar of an incision, the same symptoms might be due to an empyema which had opened into the lung. In that case the presence of large rattles and of abundant discharge of offensive pus would not exclude the diagnosis of cirrhosis, as these symptoms would if there had been paracentesis and collapse of the lung.

The *treatment* must be symptomatic, and the symptoms to be relieved are those of phthisis, of bronchitis, and of bronchiectasis. Probably the most important treatment is the removal of the patient from town to country and from cold to warm places. The south-west counties of England and Ireland, the Riviera, and islands like Madeira or Teneriffe, or those of the Pacific Ocean, are probably the best for cases of cirrhosis.

SYPHILITIC DISEASE OF THE LUNGS.—Only within living memory has it been recognised that other viscera than the testes, and other regions than those accessible to the surgeon's touch, may be seats of syphilitic lesions. The brain, the liver, the heart, beside the larynx and trachea, are now known to be liable to the formation of internal nodes or gummata.

Moreover there is a form of chronic interstitial pneumonia, or cirrhosis of the lungs, characterised by deep scarring and contraction of the tissue, by peribronchitis and tracheitis, often with ulceration and deformity, and by the presence of typical gummata. The process affects the roots or bases rather than the apices; it often starts from previous ulceration of the air-passages; and it is apt to lead to gangrene.

Its nearest anatomical ally is chronic interstitial hepatitis with gum-

mata, which clinically resembles, but pathologically differs from, alcoholic cirrhosis.

The disease is a rare one, and would be little but a pathological curiosity were it not for the importance of recognising the true nature of these cases in order to treat them successfully.

Lancereaux described cases of this kind in France ('*Traité de la Syphilis*,' 1866); Dittrich and Virchow in Germany ('*Kr. Geschw.*,' ii, 463); and Wilks in this country ('*Path. Trans.*,' vol. ix, 1858, with plate, and '*Guy's Hosp. Reports*' for 1863, 3rd series, vol. ix, p. 33, two cases).

In a series of twenty-two cases of visceral syphilis brought before the Pathological Society in 1877, Dr Greenfield described three of presumably syphilitic affection of the lungs ('*Path. Soc. Trans.*,' vol. xxviii, p. 258). In the same volume is the description of a specimen shown by Dr Sutton of chronic syphilitic pneumonia, from a patient under Dr Gull and Mr Durham in Guy's Hospital (p. 304); three of "fibroid phthisis" in syphilitic patients, by Dr Goodhart, with a histological drawing in which peribronchitis is clearly shown (p. 313, and abstracts of nineteen cases from Guy's Hospital, p. 322); one apparently of gummata coalesced into a large mass in one lung, by Dr Gowers, with a histological drawing (p. 330); one by Dr T. H. Green of a similar large mass in one lung of a man with "undoubtedly syphilitic lesions in his liver;" two by the late Dr Mahomed—both in women with undoubted syphilitic disease, but the pulmonary lesion less certainly of the same origin and perhaps in an early stage; lastly, three cases of gummata in the lung from the museum at Netley by Dr Aitken.

The following examples have come under the writer's notice:

(1) A woman of about forty, who was under treatment in 1863 at the Hôpital Beaujon in Paris. She was wasted, with cough, purulent expectoration, and hæmoptysis; and from other proofs of lues was treated with perchloride of mercury to her great benefit.

(2) A groom of about thirty who, together with signs of chronic pneumonia and hæmoptysis, had tibial nodes and amygdaloid lymph-glands. He was an out-patient under the writer's care in 1868-70, and improved greatly under iodide of potassium and mercury, with which, however, it is right to add, cod-liver oil was given; and when last seen he had gained weight, was free from pain, and able to resume work. This case closely resembled one recorded by Walshe on p. 513 of his 4th edition.

(3) A strong and muscular seafaring man about forty, who had symptoms and physical signs resembling phthisis, but was well nourished and had a good appetite, an out-patient in 1869. A gumma was discovered near the hip-joint. He was put on iodide of potassium, and under this treatment not only did the node disappear, but his cough and other pulmonary symptoms ceased, and he was to all appearance cured.

(4) A patient seen with Dr Miller, of Norwood, about 1875, who had certainly suffered from syphilis, and in whom there were physical signs of very local consolidation in one lung, together with laryngeal ulceration and hæmoptysis. He was treated with mercury, but died from stenosis of the air-passages before much good could be effected. An autopsy was obtained, and beside deformity and contraction of the trachea and bronchi, there were several gummata in the right lung, with scarring and fibrous induration.

(5) A patient in Guy's Hospital, aged forty-two, with signs of chronic disease of the lungs, which had been called consumption, and with no history or signs of past syphilitic lesions (1876). The nature of the case was not recognised until after the man's death from eclampsia. Previous to this severe caries of one ulna had led to such extensive suppuration that the arm was amputated. At the autopsy were found caries of the frontal bone with pachymeningitis, fibroid testes, and two small fibroid patches in the left ventricle. The two primary bronchi were contracted and deformed. The right lung contained a single fibroid nodule in the lower lobe; the left was solid in patches, firm and grey, with dilated tubes. There was nothing that could be called tubercular in either lung; the larynx and ileum were healthy, and there were no miliary tubercles anywhere to be found ('*Path. Trans.*,' 1877). Microscopical examination of the indurated tissue in the lung showed it to consist of a fibro-nuclear growth with numerous vessels.

(6) A sailor, aged forty-four, in Guy's Hospital, February, 1876. There were signs of chronic phthisis with hæmoptysis; a history of chancre, but none of secondary lesions, and

intense dyspnoea, evidently from obstruction to entrance of air. The larynx was perfectly normal, and there were no signs of aneurism or thoracic tumour pressing on trachea. Tracheotomy was therefore not performed, and the patient died six days after admission. There was found after death ulceration with stenosis of the trachea, "fibroid phthisis" with one old vomica and much puckering and cicatrisation. There were scars on the surface of liver, but no actual gumma. The liver and kidneys were lardaceous.

Between *sypilis pulmonum* and chronic interstitial pneumonia, or cirrhosis of the lungs, the chief anatomical distinction is the presence of gummata, and the fact that the process does not invade lobule by lobule, but divides the lung into sections by thick fibrous septa with deep cicatricial contraction. We have two unfailing criteria of its origin, one in the associated changes in other viscera, the other in reaction to treatment.

Syphilis is no protection from true phthisis, and what has been called syphilitic phthisis is in most cases nothing more than true tubercular phthisis in a syphilitic subject, which runs its course uninfluenced by the latter disease. But there is also a form of chronic pneumonia, with fibroid induration and bronchiectasis, with irregular local distribution, with no tubercle, and little or no caseation, which starts either from gummata, or from thickened patches of pleura, or from a chronic contracting peribronchitis. This peribronchitis is associated with an ulcerative inflammation of the trachea or bronchi, or both, closely related to ordinary syphilitic laryngitis. The symptoms during life are indistinguishable from those of ordinary phthisis, though the physical signs point to a more chronic and fibroid, less acute and caseous, form of disease. If the physical signs are confined to one lung and absent from the apices, one may suspect the true nature of the case; but it is only by concomitant lesions of other organs and by the effect of treatment that we can establish the diagnosis during life. Hæmoptysis is often a striking feature. Dr W. H. Porter ('New York Medical Journal,' July, 1885, with plate) mentions tenderness of the tibiæ and sternum on pressure as a symptom of value. The absence of the *Bacillus tuberculosis* is a most important negative diagnostic sign during life (Fournier, 'Gazette Hebdomadaire,' Dec., 1875).*

Hereditary syphilis of the lungs.—It is quite possible that some of the cases of gummata with cicatrices and chronic induration just described may be due not to acquired, but to congenital syphilis.

But there is another form of pulmonary disease which appears to occur only in children who show signs of hereditary lues. It is uniform, without gummata or cicatrices, and with no primary lesion of the trachea and bronchi, or of the pleura. Lungs in this condition have been described by Wagner, Virchow, and Heller in Germany, by Robin in France,† and by Wilks and Moxon in this country, as a diffuse form of consolidation, firm, dense, or even tough in texture, white in colour, and airless. Both lungs are sometimes affected, more often one, either throughout a lobe or in smaller circumscribed masses. The more universal cases are, as might be supposed, found in stillborn children, or in those who only survive birth a few days. The condition seems to be a chronic thickening of the alveolar walls and of the interlobular septa, whereby the alveoli are encroached

* Beside the papers quoted above, the following may be mentioned:—Dr Moxon, 'Guy's Hosp. Rep.,' 3rd series, vol. xiii, p. 374; Dr Goodhart, 'Path. Trans.,' vol. xxv, p. 31; Dr Rolleston (ibid., vol. xlii, p. 50), and a report by Dr Perry in the same volume (1891, p. 53); Dr Farquharson ('Clin. Trans.,' vol. viii, p. 71); the late Dr Wilson Fox's article in 'Reynolds' System;' Dr Bäumler's in 'Ziemssen's Handbuch;' and Dr J. K. Fowler's in 'Allbutt's System' (vol. v, p. 311).

† Lorain and Robin called it "epithelioma" of the lungs, Virchow "white hepatisation."

upon, and the spongy tissue rendered heavy, bulky, and more or less completely solid. On certain points there is conflicting evidence. Some writers describe the diseased patches or lobes as quite exsanguine with obliterated capillaries; others speak of free production of new vessels, so that the new growth is very vascular. Dr Greenfield described the lung of a presumably syphilitic child, twelve months old, in the 'Pathological Transactions' for 1876 (xxvii, p. 43). It was tough, yellowish white in colour, with a smooth, not granular, section, and exuded very scanty fluid. Histological drawings are given, which show bands of fibrous tissue obliterating the alveoli, of which the walls are remarkably thickened, but the endothelium is unaltered. There was no positive proof of syphilis in this case; but Dr Goodhart mentions a specimen ('Diseases of Children,' chap. xxii) in which similar *post-mortem* appearances were found in a child three months old, together with undoubted syphilis of the liver. Microscopically it showed all the features exactly as described by Dr. Greenfield—excessive fibro-nucleated growth, extreme vascularity, and collapse of the air-vesicles.

The symptoms observed during life appear to be inconspicuous.

In two cases of acquired syphilitic disease of the lungs resembling phthisis in adults, the writer found very marked benefit result from treatment by mercury and iodides.

GANGRENE OF THE LUNG.—This is a rare condition, and is rather a complication due to accidents in the course of pneumonia or other pulmonary disorders than a disease in itself.

Anatomy.—Of pulmonary gangrene without antecedent pneumonia it would be difficult to find unequivocal examples. But in the *post-mortem* room cases are sometimes seen in which there has been rapid and extensive sloughing of a portion of a lung, and in which no zone of hepatised tissue separates the gangrenous part from that which is healthy or merely oedematous. In such cases the fact of there having been inflammation is unproven. As a rule, however, the sloughing mass lies within an area of consolidated lung-substance, of which it had evidently at one time formed a part. Should the disease have proved fatal at an early stage, one condition may gradually merge into the other; should it have reached a more advanced stage, there may be a well-marked line of demarcation, or the dead tissue may have been cast off with the formation of a cavity. Experience in the deadhouse does not support the distinction between two separate forms of pulmonary gangrene, the one "circumscribed," the other "diffuse," though the distinction has been taught since the time of Laennec, who first recognised gangrene of the lungs.

The diseased part is of a dirty greenish-brown or black colour, and so soft as to be sometimes almost diffuent. It is often horribly foetid, but occasionally the odour has been little marked, as Cruveilhier long ago noticed. Microscopically the pulmonary structure is hardly to be recognised, the alveolar walls having broken down, and little remaining but elastic tissue.

Origin.—Gangrene is only a rare complication of pneumonia (*i. e.* the acute disease which causes lobar hepatisation), except in aged and feeble persons, in drunkards, or in those who are exhausted by some other malady—for example, by diabetes. But one or two instances have occurred in Guy's Hospital in which an ordinary attack of pneumonia in a healthy subject was followed, when the acute stage subsided, by symptoms which

seemed to indicate sloughing. Walshe records just such a case—that of a man who was slowly recovering from an attack of pneumonia of the right lower lobe, when hæmoptysis set in, and was followed by the copious expectoration of a frothy, intensely foetid sputum, while at the same time physical signs like those which indicate the formation of a cavity made their appearance. Ultimately this patient left the hospital free from all physical signs except dulness, and in tolerable health.

In this, as in other cases which have recovered, the proof of gangrene is obviously impossible; its probability will depend on the pathological experience and belief of the observer, and on the question whether the case occurs under his own eyes and nose, or whether he only reads the description of the symptoms.

The writer saw repeatedly during 1885, at Brighton, a strong and previously healthy man of about fifty with pneumonia, in whom, after the disease had run its ordinary course, local signs of dulness and large consonating râles at one spot on the affected side continued for three weeks, and were combined with foetid and blood-stained, but not rusty, expectoration, and a gangrenous odour of the breath and of the room in which he lay. The pyrexia was moderate; there was much sweating and loss of flesh; but the patient took food well, and at last recovered completely. Two years later I saw him again, when he was in robust health, and there was no physical trace whatever of the past disease in his lung.

Another disease in the course of which gangrene may occur is phthisis. Walshe speaks of having seen some six cases in which the special fœtor appeared incidentally in connection with tuberculous vomicæ already formed. Moreover, pulmonary gangrene sometimes complicates cirrhosis, and, more rarely, foetid bronchiectasis.

Again, the pneumonia which frequently forms the last stage in cases of diabetes, is particularly apt to end in gangrene; this occurred in four out of ten cases of diabetic pneumonia at Guy's Hospital.

In most cases, however, gangrene of the lung arises from direct putrid infection. There are various ways in which it may be set up. Sometimes it is by direct extension from neighbouring parts, as when an ulcerating cancerous growth in the œsophagus eats its way into the lung, or when perforation occurs from a suppurating hydatid or from simple abscess of the liver, or from suppuration spreading from an ulcer of the stomach, or from a putrescent empyema. The gangrenous pneumonia which Buzzard observed among our troops in the Crimea was associated with scurvy and a foetid condition of the gums.

Sometimes the infection is brought by the blood-vessels. Thus emboli may become lodged in branches of the pulmonary artery, either derived from a cerebral sinus, which was inflamed after otorrhœa, or from a systemic vein in the neighbourhood of gangrene, or necrosis.

Again, putrid materials that pass into the air-passages may be originally derived from the mouth, as in cases of gangrene of the cheek or of the tonsils, of diphtheria of the fauces, or of sloughing cancer of the tongue. There are cases, also, in which necrotic pneumonia is probably due, not to the entrance into the bronchi of matters from without, but to the decomposition of retained secretion, as in Dr Pitt's case of gangrene from bronchiectasis ('Path. Trans.,' 1891).

In yet other cases the disease starts from a foreign body, as a piece of bone, impacted in one of the bronchi, and there setting up sloughing.

The most frequent, however, of all the causes of pulmonary gangrene is the entrance of food into the air-passages, as in patients who have chronic laryngeal disease, or in those who are comatose from apoplexy, or in lunatics who have to be fed by force. A like result may be produced by particles of food from the stomach being drawn into the lungs during the act of vomiting, especially during narcosis by anæsthetics; we have seen instances of death brought about in this way in cases of hernia or of intestinal obstruction in which there had been a copious discharge upwards of the contents of the small intestine.

In all these cases the sloughing is due, not to mere purulent infection, but to decomposing particles of food or of tissue; not to pyogenic micrococci, but to necrotic saprophytes.

Sputum.—In foetid bronchitis, as we have seen (vol. i, p. 1072), the patient's breath and his expectoration may have either the true odour of gangrene, dependent upon the decomposition of dead tissues, or a peculiar nauseous acrid odour, which is sometimes not unlike that of fæcal matter, and which appears to be due to the presence of free fatty acids.

In the former case the sputum does not separate into three layers, nor are the "plugs" described in vol. i, p. 1072, to be found. The odour in these cases is simply that of gangrene—an indescribable fœtor, but one which is always of the same character, though it varies greatly in intensity, being sometimes only just perceptible, sometimes so strong as to poison a whole room. As a rule, the patient's breath has the same smell as the expectoration, especially after coughing; or the breath may be characteristically offensive for some days, while the sputum remains odourless. Moreover there are cases in which during life no fœtor is discoverable, either in the breath or in the expectoration, so that the presence of gangrene may not be suspected until it is seen at the autopsy.

The foetid sputum of gangrene of the lung is commonly of a dirty grey or greenish colour; sometimes it is brown or almost black, from the presence of altered blood; sometimes thin, only slightly tinged with blood, and less offensive to the sight than to the smell. The microscope does not often lead to the detection of recognisable fragments of pulmonary tissue, although we shall see that such fragments are often to be found in cases of phthisis. Obvious hæmoptysis is said to occur more often in children than in adults. Fatal hæmorrhage, due to the erosion of a large vessel, is extremely rare.

Complications.—When the surface of the lung is affected, the pleura usually gives way, leading to the formation of pneumothorax, which is quickly followed by septic pleurisy. If there should happen to be local closure of the serous cavity by adhesions, it is said that a subcutaneous emphysema may develop itself, or that an abscess may form, which may open externally after burrowing to a distance. Another occasional effect of the presence of a patch of gangrene in the lung is said to be the dropping of putrid matters into tubes belonging to other parts of the organ, so as to set up sloughing in them also. In this way, according to Hertz, the diffuse form of gangrene may arise out of the circumscribed.

Symptoms.—The only physical signs of gangrene of the lung are such as serve to indicate the formation of a cavity in the organ at a spot where the tissue previously was either healthy or simply consolidated. But it can be only in very rare cases that such signs are to be definitely made out. They would include amphoric breathing, large consonating râles, and bron-

chophony. When there is a possibility of the presence of phthisis, the mere detection of a cavity proves nothing as to the exact seat of the sloughing process, unless it is known that no vomica existed at the same spot before the foetid expectoration began.

The general symptoms that accompany gangrene of the lung are often severe, but they do not point to its presence so definitely as might be supposed. It is said, for example, that the pulse is small, feeble, and very frequent, and that the pyrexia quickly passes into an adynamic form, with prostration. That absorption of putrid matters into the blood from the lung should produce such results is, indeed, to be expected; but when the characteristic foetor is absent, it surely is not possible for anyone, from the intensity of the general symptoms alone, to suspect that sloughing of the lung is taking place. In ordinary pneumonia the patient often falls into a similar condition before death; and the same may be said of many other diseases that sometimes lead to pulmonary gangrene.

Prognosis.—It is only when the gangrene is limited to a small part of the lung that recovery is possible. How minute a slough may cause foetor is well shown by one of the cases of phthisis complicated with gangrene which were recorded by Walshe; in that instance the expectoration of a pea-like mass brought the foetor to an end.

Treatment.—It is an important point to diminish the foetor as far as possible. As in cases of putrid bronchitis (*cf.* vol. i, p. 1076), the most effectual means of attaining the object aimed at is by inhalations of oil of turpentine, carbolic acid, creasote, or eucalyptol. Turpentine inhalations were used by Skoda forty years ago. His plan was to pour a teaspoonful or two of oil of turpentine upon the surface of some boiling water, and to let the patient draw the vapour into the lungs. The *oleum cardinum*, or the eucalyptol, may be directly inspired from a sponge placed in an “orinasal respirator.” The effect of such inhalations is sometimes very striking in cases of foetid expectoration. Or we may use a Siegel’s spray apparatus, so as to atomise a liquid containing from five to two parts of carbolic acid in 100 parts of water. The inhalations may be repeated two or three times a day. Care must be taken that there is not enough absorbed to set up headache or giddiness, or to give the urine a dark colour.

It is needless to say that the patient must be frequently fed, though not more frequently than every two or three hours, and that the administration of stimulants in large doses is sometimes necessary. Ammonia, camphor, ether, quinine, musk, and the tincture of perchloride of iron, may each do good service. Oil of turpentine may also be administered by the mouth in doses of twenty or thirty drops, either beaten up with the yolk of an egg, or made into an emulsion with tragacanth or tincture of quillaia.

PHTHISIS

‘While meagre Phthisis gives a silent blow,
Her strokes are sure, but her advances slow ;
No loud alarms nor fierce assaults are shown ;
She starves the fortress first, then takes the town.”
GARTH.

History and definition—unity of phthisis—phthisis always tuberculous—Anatomy—localisation in the apices—Histology: miliary, caseous and infiltrating tubercle—Tuberculous pneumonia and ulceration—vomice—adhesions—contractions—involution.

Symptoms: wasting, pallor, pyrexia, cough, sputum—hæmoptysis—phthisis ab hæmoptoë—Physical signs of the three stages and of involution—Diagnosis—Course and duration—mode of death—recovery—Prognosis.

Phthisis in children—Senile phthisis.

Ætiology—contagion—hereditary disposition—diathesis and conformation—overcrowding, etc.—Inhalation of dust—damp soil—Infected dwellings—Antagonistic conditions—Age and sex—Distribution.

Treatment: preventive, curative, and palliative—diet and hygiene—voyages—climate—sanatoria—drugs—Treatment of complications.

Acute Pulmonary Tuberculosis—Distinctive characters—Anatomy—Physical signs—Clinical symptoms and course—Diagnosis—Ætiology—Prognosis.

OF all diseases which attack adults, Phthisis* or pulmonary Consumption is in this country, and throughout the temperate regions of Europe and America, by far the most fatal. It is estimated that a third of the deaths between fifteen and forty-five in England is due to this terrible disease.

The early Greek physicians knew that progressive loss of flesh often accompanies cough, spitting of pus and blood, and other signs of disease of the lungs. This was distinguished as *phthisis pulmonum*.

The word *phthisis* (wasting, *tabes*, *consumptio*) occurs frequently in the Hippocratic writings, and there, as well as in those of Celsus and of Aretæus, means pulmonary phthisis. The ancient physicians distinguished between Empyema, as suppuration outside, and Phthisis, as suppuration inside the lung. Celsus divides *Tabes* into Atrophy, Cachexia, and Phthisis. The following quotation shows how good a clinical knowledge of the disease he had:

* *Synonyms.* — Phthisis pulmonalis — Tabes pulmonum — Consumption — Decline — Chronic tuberculosis of the lungs. — *German.* Lungenschwindsucht. — *Fr.* La phthisie.

"The third kind of decline (*tubes*) and by far the most dangerous is what the Greeks have named *φθίσις*. It usually arises from a cold in the head, whence it settles on the lungs, and there causes ulceration. There follows a slight feverish movement, which remits and comes again. There is a constant cough, raising of yellow matter, and sometimes of blood."

There is a curious secondary meaning of the word. Even before it was shown that all cases of phthisis are tuberculous, such expressions as "renal phthisis," "abdominal phthisis," "laryngeal phthisis," were introduced, to imply that the kidneys, the bowels, or the larynx present tuberculous lesions. But to make the clinical term phthisis synonymous with the anatomical term tuberculosis only leads to confusion.

It would be impossible, if desirable, to abolish so ancient and universally accepted a term as phthisis, and all that is needful is to define the disease known to Hippocrates and Galen, to Sydenham, Morton, and Laennec, to be a complex pathological process dependent, like Enteric fever or Diphtheria, on a single cause, infection by a specific microbe.

Phthisis one disease.—The appearances presented by the diseased lungs in cases of phthisis differ exceedingly; and its clinical symptoms and course are subject to no less wide variations. One cannot be surprised, therefore, that both pathologists and physicians have endeavoured to divide it into several species, forms, or varieties. Addison led the way in this direction by insisting that much of what was commonly regarded as tubercular disease in the lungs was in reality pneumonia. In his essay, read before the Guy's Physical Society in 1845, he described first a "pneumonic" and then a "tuberculo-pneumonic phthisis;" and his final sentence is, that "in every form of phthisis, inflammation constitutes the great instrument of destruction." After his time other pathologists, particularly Niemeyer, asserted that "catarrhal" or "caseous" pneumonia is the essential morbid change in many, if not in most cases of phthisis; while many in England declared the so-called "fibroid phthisis" to be non-tuberculous. But the general opinion remained true to the belief that all forms of phthisis are tuberculous.* The long-continued controversy was finally closed by the discovery of the specific microbe which is present in all tuberculous lesions, and which is never absent in any case of what is clinically recognised as phthisis. Thus the doctrine of Laennec is now universally accepted, that phthisis is only one disease, and that it is always tuberculous, or, to use his own words in 1819, "the existence of tubercles in the lungs is the cause, and constitutes the true anatomical character of consumption."

The tubercle bacillus (described above, vol. i, p. 368) is present in all cases of phthisis of whatever form or clinical aspect. In his earliest communication on the subject, Koch stated that he had found bacilli in twelve cases of caseous pneumonia with bronchitis; and these appear to have been all the examples of that variety of phthisis he had then examined. In fibrous phthisis one must look for the characteristic bacilli in the parts of the lungs most recently affected. In the caseous form, also, they are

* Dr Fagge's opinion was thus expressed in the first edition of the present work:—"For my own part, I believe that all ordinary cases of phthisis are essentially of the same nature. The varied appearances which may be found in the lungs after death seem to me to depend mainly upon whether the tubercles and the tuberculous infiltration become caseous or undergo fibrous changes. This, to a great extent, rests upon the degree of rapidity with which the disease has advanced during life. Thus pneumonic phthisis is, I think, generally equivalent to a phthisis which has progressed quickly: fibroid phthisis to one which has been slow in its course."—C. H. F.

generally limited to the edge of the infiltrated tissue. Sometimes nests of bacilli occur in the midst of inflammatory consolidation. In tuberculous vomicæ they are often present in great numbers. Caseous fragments, which are so often found in the cavities of phthisis, consist, according to Koch, almost entirely of masses of bacilli.

The varied appearances which may be found in the lungs after death depend mainly upon whether the tubercles and the inflammatory consolidation they cause become caseous and soften, or undergo fibrous changes and harden. This chiefly depends on the rapidity with which the disease has advanced during life; so that pneumonic phthisis generally means cases which have advanced quickly; fibroid phthisis, those which are very chronic. Scarcely any case of phthisis is without bronchitis and pleurisy, and few are without tuberculous lesions elsewhere; but none are without the essential lesions of tubercle, catarrhal pneumonia, caseous degeneration, and more or less attempt at fibrous cicatrisation.

Anatomy: locality.—Phthisis affects both lungs. Clinically we constantly meet with early cases of consumption, in which the physical signs are confined to one side of the chest; but, as the disease advances, the opposite side becomes also involved. The evidence of autopsies proves that it is the rarest event for a patient to die of phthisis with only one lung affected. Nevertheless the disease is not perfectly symmetrical; it is almost always earlier and more advanced in one lung than in the other. It is much more symmetrical than pneumonia or pulmonary cirrhosis or pleurisy, more so than tuberculous disease of the testes or adrenals, and less symmetrical than chronic tubal nephritis or than psoriasis.

It has long been known, both to physicians and to pathologists, that the upper parts of the lungs are almost invariably affected with phthisis, in whatever form, before the lower parts; and that in all but the most exceptional instances the disease spreads downwards from apex to base. Moreover the same is true when the lower lobe is affected—the earliest lesions are in its apex, and the disease spreads downwards.

The general rule of the proclivity of the apex is liable to certain exceptions. In some cases the tubercles appear a little lower down, leaving the extreme summit of the upper lobe free, and often the seat of emphysema. But the tuberculous process seems never to spread upwards from the base of a lung into and through the upper lobe. Most cases of “basal phthisis” are probably not tuberculosis, but a form of “chronic pneumonia” or cirrhosis of the lung (*supra*, p. 27).

A satisfactory explanation of this remarkable distribution is not easy to find; and the difficulty is increased by the fact that the same distribution is true of miliary tuberculosis. Dr Hamilton believed that the apices are the driest parts of the lungs, so that caseation of catarrhal products is more apt to take place there than elsewhere; and also that the apices expand less during breathing, and so catarrhal products are more likely to accumulate there than in other parts of the lungs. Rindfleisch insisted that the upright position of the body in man and in the *Quadrupana* causes the weight of the shoulders and arms to fall upon the upper ribs, and so interferes with their play, and leads to a deficiency in the movement of air in the apices as compared with the lower lobes. Thus catarrhal secretions are less easily removed thence. Strümpel also thinks that the bacilli lodge in the apices because less movement takes place there; and Geigler believes

that the circulation in the apices is less active than in the rest of the lungs. On the other hand, the proclivity of the apices to phthisis is no greater in men than in women, who use those parts of the lungs far more than men do, and according to Moxon the anterior edges are the earliest seats of tubercle in persons who are confined to bed; a fact which he explained by supposing that in a bedridden patient these parts are the most, not the least, active in respiration.

Dr H. J. Campbell, in the 'Guy's Hospital Reports' for 1891 (vol. xlviii, p. 33), observes that the monkeys in the Zoological Gardens at Regent's Park die more frequently from pulmonary tuberculosis than from any other malady (20 cases out of 38 deaths), but that the apices of the lungs are generally free, or less affected than other parts. He finds the explanation of this difference from human pathology in the fact that owing to the length of the first rib and the backward direction of the clavicle in *Quadrupana*, the apex of the lung, even when distended, does not rise out of the thorax as it does in man: it thus escapes the full atmospheric pressure which in human beings makes it difficult to expand the apices by inspiration, and to empty them by expiration. Thus not only are the air-vesicles above the clavicles less aerated than those within the thorax, but mucus and other contents are less readily expelled. Indeed, forcible expiration, as in a fit of coughing, may drive mucus or bacilli from the intra-thoracic into the cervical portion of the lungs.

Often the upper lobe presents a dense fibrous mass (perhaps containing cavities), while in the middle of the organ there are cheesy patches, and in the lower lobe grey tubercles, scattered or in groups. Or, again, the affection in one lung may be fibrous or caseous throughout; while in the opposite lung, in which the disease is of more recent origin, are only clusters of tubercles. Lastly, the pulmonary lesion may appear to be fibrous, not a single miliary tubercle being discoverable, grey or caseating; yet grey or yellow tubercles will be found in the ileum, or liver, or spleen.

Miliary stage and consolidation.—Rindfleisch maintained that the process in phthisis begins where the bronchioles open into the alveoli. Such a lesion at the extremities of several adjacent tubes, and extending along the walls of the tubes, would account for the "racemose" distribution of pulmonary tubercles which gave rise to the phrase "Carswell's grapes." But we seldom see a "peribronchial" distribution of clustered tubercles; and Hamilton described tubercle in the lung as generally beginning in a projection on one side of an alveolus. At first it pushes before it the epithelium and the alveolar capillaries; but it soon breaks through and destroys the alveolar wall, so that a uniform rounded nodule results, in which the outlines of the original air-vesicles are barely recognisable. The cells of the primary grey tubercle may be derived either from the connective-tissue corpuscles of the alveolar wall, or from the endothelium of its capillaries. Sometimes a tubercle sprouts from the inner coat of a branch of the pulmonary artery. Other tubercles lie in the course of the pulmonary lymphatic vessels contained in the periarterial and peribronchial sheaths, the interlobular septa, and the deep layer of the pleura.

In some instances tubercles, scattered or in clusters, spread slowly through the lung, with little or no change in the intervening spongy tissue. But, as a rule, this undergoes early consolidation, so that the tubercles come to be embedded in the products of secondary inflammation. Sometimes the lung is involved uniformly from the apex downwards, the edge of

a consolidated area having a festooned outline, not unlike the border of a malignant growth. More frequently, even when part of the upper lobe is uniformly solidified, there are separate nodules of various sizes lower down; and below these again, scattered tubercles may generally be seen as harbingers of the disease.

The solid area may be soft, translucent, and pinkish, from catarrhal obliteration of the vesicles, or yellow and friable from caseous degeneration. In very chronic cases it is firm, dark, and tough, presenting one form of Addison's iron-grey induration; or it may have a "marbled" aspect, crossed by bands and seams of fibrous tissue, and pigmented in all degrees of depth. There are very few cases in which the caseation may not be found in some part of the lungs.

Excavation.—With rare exception the process of consolidation is followed more or less quickly by one of *ulceration*, leading to the formation of cavities technically called *vomicæ*.^{*} The tubercles no doubt soften in their centres, as they do elsewhere, and a vomica may result from the breaking down of caseous material derived from tubercles alone; but the formation of cavities always involves destruction of infiltrated lung-substance as well.

One characteristic appearance is the presence of a definite caseous zone between a vomica and the surrounding healthy tissue; this is an indication that the disease was still spreading at the time of death. As the vomicæ enlarge, cavities are apt to open into one another, and thus a single cavern of irregular form may be produced. Sometimes the destructive process remains limited by the lobar septum; sometimes this also is ulcerated through, so that the whole of the upper and a part of the lower lobe may form one huge sac.

Sooner or later, if the patient should survive long enough, the further extension of a vomica is arrested by a healing process. The indication that this change has begun is that the interior of the cavity, instead of being rough and shaggy with adherent cheesy débris, has a smooth appearance, like that of a mucous membrane, and is surrounded by a fibrous wall. Such smooth-walled vomicæ are often crossed by fibrous bands or *trabeculæ*, consisting of condensed pulmonary substance, with fibrous tissue that perhaps originally belonged to interlobular septa. In all probability some trabeculæ are remains of partitions that at one time separated vomicæ since coalesced; others contain obliterated branches of the pulmonary artery; and sometimes several can be seen converging to a point of the cavity nearest the root of the lung. The formation of the last is probably due to the resistance offered by the arterial walls to the process of ulceration. Ultimately the trabeculæ themselves have given way, and their loose ends may be seen hanging into the interior of the vomicæ. In large cavities a bundle of such ruptured trabeculæ may sometimes be seen, and their relation to the pulmonary artery is at once shown if a probe is passed into that vessel from the heart. Sometimes a pervious channel persists for some little distance in a trabecula, a fact which we shall see to be of clinical importance.

It has been asserted that some of the smooth-walled cavities in a phthisical lung are dilatations of bronchial tubes, and not formed by ulceration. The notion that some vomicæ are bronchiectases seems to have originated with Laennec, and was suggested by the difficulty of a cavity formed by ulceration having a mucous membrane. But now we know how readily

^{*} *Vomica*, a foul sore, an internal abscess: an old Latin word ('Liv. Hist.,' xxv, 72) applied by Celsus (lib. iv, cap. 8) to abscess of the liver as well as of the lung.

"Et phthisis et vomicæ putres."—Juv., xiii, 95.

epithelium can grow over a raw surface from an edge of skin or mucous membrane, as in the case of a rectal fistula. Moreover, it is doubtful whether smooth-walled pulmonary cavities ever have a continuous epithelial lining. Dr Ewart, in his *Gulstonian Lectures* for 1882, says that this is wanting, except where there are "scattered islets of mucous membrane," the remains of "outlying bronchi intersected by the cavity wall."*

The contents of vomicæ vary widely. When recent they often show caseous masses, which would no doubt crumble into fragments in the course of time, and be expectorated. Cavities of old date usually have pus in their interior. This implies that there is no very free communication with the bronchial tubes, and the fact is that in old vomicæ there is a tendency for the orifices of the tubes to contract until they become very narrow. Often a bronchus of considerable size, into which a large catheter might be passed, has an opening into a cavity that will but just admit a probe. In such a case, swelling of its lining membrane might easily block it altogether.

It is remarkable how seldom the contents of phthisical cavities putrefy; they often have a faint, sickly odour, but they very rarely become foetid, nor do they often undergo that peculiar acid fermentation which is apt to arise in cases of chronic bronchitis and bronchiectasis (vol. i, p. 1071). When there is free escape of pus from vomicæ, their lining membrane may continually pour it out and furnish large daily expectoration. But sometimes at an autopsy the walls are found perfectly dry, and the interior empty; in such cases there may have been no expectoration during life.

Involution.—To complete the morbid anatomy of phthisis we have still to discuss the way in which the tuberculous lesions in the lungs become obsolete, so that they cease to threaten the patient's life, or even to impair his health; for the disease is not incurable.

The proof of this important statement rests not only on clinical observation of persons who have suffered in youth from hæmoptysis, and appeared to be going into a decline, yet have recovered and lived to a good age—of these we shall give examples further on,—but we have the unmistakable evidence of the deadhouse. Here relics of former mischief are frequently discovered in the lungs of those who have died at various periods of life and of every kind of disease or injury. In May, 1880, Dr Heitler, of Vienna, brought before the Medical Society of that city an analysis of all the cases of this kind that had been met with in a series of 16,562 autopsies between the years 1867 and 1879. Excluding cases in which death was due to phthisis, he found that there were no fewer than 780 (or almost exactly 5 per cent.) in which obsolete tubercle was present. In no fewer than 651 cases *both* lungs showed signs of past disease, though generally to

* Dr Hamilton maintains that cavities which cannot be dilated bronchial tubes often have an epithelium "most typically columnar and ciliated;" and he explains the occurrence of bronchiectasis, and the sinuous and irregular outlines of the cavities which he believes to be of such a nature, by referring it to the traction of bands of fibrous tissue radiating away from the sides of the cavity at different points. What seems to prove that the cavities in question are really vomicæ is that the earlier stage of the process of dilatation is never seen. If the view adopted by Dr Hamilton were correct, one ought, towards the margin of the affected part of the lung, to see tubes which could still be traced on to their extremities, but the sides of which were beginning to bulge out here and there. On the other hand, what one does commonly find are all possible transitional varieties between smooth-walled cavities and unmistakable vomicæ. The former are seen towards the apex, where the mischief is of oldest date; the latter lower down, where it is of more recent origin. Moreover, smooth-walled cavities often riddle the substance of a diseased lung in all directions, communicating freely with one another on every side, so that an ulcerative process must clearly have been concerned in their formation.—C. H. F.

an unequal extent; in sixty-eight the right lung was alone affected, in sixty-one the left.*

That such relics of long-past pulmonary mischief belong to the same affection which, when it goes on and destroys life, is called phthisis, cannot be doubted. For their seat is in or near the apex of the lung, the affected part is more or less indurated, and it is often puckered on the surface or adherent to the chest wall. On section it presents fibrous bands, or tough masses of fibrous tissue, often deeply pigmented, and sometimes containing calcareous nodules.

Kurlow has found by experiment that such obsolete tubercle is sometimes still infectious when injected, and reproduces tubercle in animals; and Dr Vincent Harris has found tubercle bacilli in old preparations from St Bartholomew's Museum.

The caseous nodules are not infrequently gritty from deposition of lime salts, or converted into hard smooth calculi.

The puckering of the pulmonary tissue often looks like a cicatrix, and it probably represents former vomicæ which have undergone obliteration. Dr Theodore Williams has shown that this process of contraction of a vomica is often attended by shifting of its position. Unless it is closely in contact with a firmly adherent pleura, the most fixed part of its wall is that which contains the openings of bronchial tubes; consequently it often shrinks away from the front of the lung towards the root. Dr Ewart, in his 'Gulstonian Lectures' for 1882, gives diagrams showing that the pulmonary pleura, if not too extensively fixed by adhesions, may be drawn inwards over such a receding cavity until it forms a deep chink or fissure.

The space made by the shrinking of a vomica is often partly filled up by the adjacent tissue becoming emphysematous, the bullæ having been probably formed during inspiration, as explained by Gairdner (vol. i, p. 1062). More frequently, however, the lower part of the upper lobe of the lung, or (in the case of the right lung) the fore-part of the middle lobe, is uniformly enlarged; if there is great contraction of one lung, the upper lobe of the opposite one may increase in size until it passes across the median line. Other organs at the same time undergo displacement. The liver or the stomach is dragged upwards, according as the right or the left lung is the one which is diseased; and the heart may be pulled over either to the right, or beyond its natural position to the left. Lastly, the upper ribs are drawn inwards, so that the chest wall, especially below the clavicle, appears flattened or even hollowed.

The conversion of a great part of the upper lobes of the lungs into cicatricial tissue, with thickened and adherent pleura, has led to the term "fibroid" (more correctly fibrous) phthisis, and there is no objection to the term as a descriptive one, like hæmorrhagic or pneumonic phthisis. But the attempt to separate these chronic cases with fibrous cicatrization from the tuberculous disease was a step backwards in pathology.

Summary.—We may conclude our account of the anatomy of phthisis

* A point of interest is that the proportion of cases at different ages went on regularly increasing for each decennial period up to sixty years of age. Among persons aged from ten to twenty there were 12 cases of phthisis; from twenty to thirty, 105; from thirty to forty, 131; from forty to fifty, 156; from fifty to sixty, 157; from sixty to seventy, 36; from seventy to eighty, 153. It is true that no positive conclusion can be drawn from this fact, in the absence of information as to the proportion of persons at different ages in the total number of autopsies, but it is difficult to escape the inference that the time at which the pulmonary lesions were originally developed must, in a considerable number of instances, have been during adult life.

by saying that one of its characteristic marks is its multiformity. In every case there is more or less evidence of bronchitis, in every case the effects of pleurisy, in every case discrete tubercles, grey or yellow, and infiltrating caseous tubercle. In every case there is catarrhal broncho-pneumonia, with caseous degeneration, softening, and destructive ulceration, causing more or less developed vomicae; and in almost every case there is some attempt at repair, shown by fibrous induration, contraction, and cicatrization.

On the other hand, pleuritic effusion is rare, except in the earliest stage, and empyema is still rarer. True lobar fibrinous pneumonia is an infrequent and probably accidental complication; and neither abscess nor gangrene is met with. Pneumothorax sometimes occurs, and must always be remembered as a possible event. Emphysema, chiefly of the anterior edge of the lungs, is present in almost all chronic cases.

The anatomical lesions which most frequently accompany pulmonary phthisis are ulceration of the *ileum* and of the *larynx*. The latter will be described in the next chapter; the former will be noticed among the diseases of the intestines. Next in frequency comes tuberculous pleurisy, then peritonitis, tuberculous meningitis and tuberculosis of the spleen, liver, and kidneys, of the testes in men, and the Fallopian tubes in women.

In children the most frequent lesions are found in the spleen, liver, peritoneum, and pia mater.

Symptoms.—The clinical recognition of phthisis, as of pulmonary diseases in general, is based partly upon symptoms, partly upon physical signs; and there is no disease in which diagnosis depends more on the concurrence of the two kinds of evidence. Symptoms alone, when no signs can be detected, may justify a strong suspicion that phthisis is present; but until this is confirmed by signs this suspicion never reaches certainty. On the other hand, when one discovers physical signs of the disease in a person whose health appears perfect, and weight normal—as, for example, in a candidate for life insurance,—the inference is that they depend upon a lesion, which although it was phthisical, yet is now obsolete, at least for the time. Probably physical signs never develop themselves in phthisis without symptoms having preceded them, although the patient may fail to notice or may wilfully conceal them. A description of the symptoms of the disease will therefore best precede that of the signs.

The symptoms of phthisis fall into two groups: those which point directly to the lungs; and those which concern other organs, or the whole body. We will take the latter first.

Wasting.—Of the general symptoms, the most important is progressive loss of flesh. This often occurs with extreme rapidity. Rühle mentions the case of a very bulky woman, who had weighed 240 lbs., and who lost 40 lbs. in the four weeks before she came under his care for hæmoptysis, although no physical signs could be detected. Ultimately phthisical patients often lose a quarter or even a third of their weight. The explanation of the wasting is not always obvious. There is often great loss of appetite, and especially distaste for fat in every form, while in some cases vomiting is added to anorexia; but many patients who eat well, and appear to digest what they eat, still lose flesh steadily; nor is the wasting always accounted for by the degree of pyrexia or the amount of sweating. There is no question that diminished appetite and imperfect assimilation of food,

hectic fever and continued colliquative diarrhœa, profuse purulent expectoration and excessive sweating, each tend to produce loss of flesh; but it almost seems as if the growth of tubercle, like that of cancer, has a certain wasting effect, independently of diminution of the income or increase of the expenditure of the organism.

Atrophy probably affects all the tissues more or less; but the heart becomes much less reduced in size in phthisis than in other wasting diseases, as, for example, cancer, probably because the right ventricle has increased work thrown upon it by the obstruction in the lungs. On the other hand, in very chronic fibrous cases the heart may be found over weight, from hypertrophy of the right side.

With the emaciation there is often failure of strength. The patient is no longer able to walk without fatigue; the duties of the day tire him, so that he is glad to get home and lie on the sofa until he goes to bed; and in the morning he feels unfit for the day's work. Nevertheless in many cases the mental activity and the muscular strength persist to a surprising degree, even in an advanced stage of the disease.

Anæmia is often an early symptom of phthisis. The face becomes pale, the hands are white and transparent. In women scantiness or suppression of the catamenia may be one of the first indications that the health is failing. Œdema of the ankles often occurs as the disease advances, but it is seldom considerable unless due to venous thrombosis. It depends rather on weakness of the left ventricle from anæmia than upon dilatation of the right ventricle from pulmonary obstruction; and there is never general dropsy or albuminuria, as in cases of chronic bronchitis.

The *skin* is usually moist, but occasionally harsh and dry, with diminished sebaceous secretion—the state known as *xerodermia* or *pityriasis tabescentium*. The growth of the *hair* is also changed. The straight lanky whiskers and beard of consumptive patients, and the long thin hair upon the chest, often suggest the nature of their disease.

Pyrexia.—When consulted by a person who has lost colour and weight, we first ascertain whether the pulse is quickened and whether the temperature is raised in the evening. One should notice if the palms of the hands are hot, if the cheeks are flushed, and if there is much perspiration at night. The pyrexia of phthisis in different cases varies widely in character and range, but is very seldom absent.*

In the most acute cases, which in Germany are called “phthisis florida,” the pyrexia may be continuous throughout the twenty-four hours; the temperature may reach 104° Fahr., and keep above 102°, unless profuse sweating occurs, when it usually falls one or two degrees. It is remarkable that with this high fever delirium is rare; and it is quite an exception for the patient to pass into a “typhoid” condition, with stupor, sordes on the lips, and a dry, brown tongue. Moreover, phthisical patients often retain a better appetite than is usual with a like degree of pyrexia, and suffer less from thirst.

Scarcely less acute is the course of other cases in which the daily range of temperature is very wide, the maximum reaching 103° or 104°, while the minimum may be 98·4°, or even lower. Rühle justly remarks that a subnormal temperature, alternating with a high fever, is more unfavourable

* Dr Theodore Williams, however, in vol. lviii of the ‘Med.-Chir. Transactions,’ says that in several of his cases, though active disease was going on in one or both lungs, no rise of temperature took place. In one case five observations were made every day for a week, and the thermometer was never found above 99°.

than when the fall is not below normal. Sometimes the patient experiences a slight rigor or a sensation of chilliness, and then passes through hot and sweating stages, like those of a paroxysm of ague.

In less severe cases the range of the temperature is smaller; the thermometer may indicate 100° or 101° towards evening, but during the rest of the day it is scarcely above the normal point. Even when pyrexia is generally present, none may be detected during intervals of days or weeks. Of the causes of the differences in degree of pyrexia in different cases of phthisis no satisfactory account has yet been given. The late Dr Wilson Fox ('Med.-Chir. Transactions,' vol. lvi) thought that it is generally proportioned to the extent of the intercurrent inflammation; but probably it depends chiefly upon rapidity of production of fresh tuberculous areas, with increase of the toxins of the specific bacilli.

It must also be remembered that in many cases of phthisis, as of Enteric fever (vol. i, p. 139), there are, beside the pathogenic bacillus, streptococci and other septic organisms present, which undoubtedly help in producing pyrexia, with pulmonary suppuration and intestinal ulceration.

Sweating comes on most during sleep, and some patients cannot doze for half an hour during the day without their clothes becoming saturated. A paroxysm of cough sometimes starts a fit of perspiration.

Pulse.—The heart's action is nearly always accelerated in phthisis, and its rate indicates the activity of the disease almost as well as the temperature itself. Like the pyrexia, it is highest in the evening. It is much affected by slight exertion, and even by a change of position from sitting to standing. The frequent radial pulse is generally soft and feeble, and sometimes its rapidity is out of proportion to the degree of fever.

Vomiting is occasionally the most marked of the early symptoms of phthisis, and grave errors have been committed in supposing that the patient's complaints were all due to disorder of the stomach. This symptom may also be the direct result of a fit of coughing.

Diarrhœa is a frequent symptom in the later stages of phthisis. It is often due to the presence of tuberculous ulcers of the small intestine, or rather, perhaps, to a catarrhal state of the mucous membrane which precedes and accompanies such ulcers. Occasionally diarrhœa from this cause persists for many weeks before any physical signs of pulmonary disease can be detected, so that the case has been regarded as one of Enteric fever; but more often the phthisical patient complains of constipation. In advanced cases another cause of diarrhœa is the development of lardaceous disease in the intestinal mucous membrane.

A minor point, on which French writers have insisted, is the presence of a red line on the gums close to the teeth. Whether it is seen more often in persons who are consumptive than in others is doubtful.

Dyspnœa is a much less marked symptom in phthisis than might have been expected. The gradual onset of the disease, and the loss of muscular power and of body-weight keeping pace with the destruction of the lungs, probably account for the fact that a patient, even with advanced phthisis, is often able to breathe quietly, and to carry on conversation with comfort, so long as he is sitting still. As Watson remarked, persons who fear, but will not believe, that they are consumptive, will fetch a deep breath, and bid us remark how thoroughly they can distend their lungs. But any exertion causes obvious hurry of breathing; and towards the last, orthopnœa is sometimes present in an extreme degree, so that the

patient gasps for breath, while his face and hands are livid and bathed in sweat.

It is no doubt as a consequence of obstruction to the pulmonary circulation that in the more chronic cases of phthisis, and in these only, we see clubbed finger-ends with curved nails (*ungues adunci*). The same hypertrophy affects the toes also, as in cases of empyema, of chronic bronchitis, and of heart disease.

Pain is not often distressing in cases of phthisis. There may be pain in the shoulder, or beneath the collar-bone, or lower down; but in many cases even this seems to be muscular. The pleurisy which invariably fixes the lung to the surrounding structures seems to be painless; but pleurisy lower down, where there is more movement of the parietal upon the pulmonary layer, is less frequent, and may be attended with sharp pleurodynia.

Aspect and temper.—A phthisical patient often betrays the nature of his disease to the experienced physician at the first glance. Apart from the question (to be considered presently) of there being a special configuration indicative of a phthisical tendency, a bright eye and a flushed cheek, associated with a pale face, wasted frame, slender fingers, and lank hair, at once suggest consumption.

The consumptive patient is generally hopeful (*spes phthisica*), and, in spite of much suffering, is as a rule cheerful and uncomplaining. In this respect phthisis contrasts with disease of the heart, but still more with affections of the abdomen, particularly of the rectum.

Cough and sputum.—Passing from the general to the pulmonary symptoms of the disease, we may first remark that *cough* is often the earliest indication of phthisis. At first it may be very slight, hardly more than a clearing of the throat; or it may occur only in the morning, or after exertion during the day. It sometimes disappears for a time, to return later on. But ultimately it becomes more and more frequent, until it often causes great distress. It is when cough has been the first symptom noticed, that the disease is said to have arisen out of a “neglected cold.”

A dry cough (*tussis sicca*, βήξ κενή) is characteristic of early phthisis, hence the phrase “a dry phthisicky cough.” After a time, however, there is more or less mucous expectoration. As the disease advances, the expectoration becomes muco-purulent and streaked with blood; and it may ultimately be almost pure pus, or pus so mixed with blood that it has a uniform brick-dust colour.

In other cases the sputum consists of round or disc-like pellets that remain distinct from one another in the mucus; they are called “nummular sputa,” from their resemblance in size and shape to coins. If received into water they are seen to have a loose flocculent surface, as if they were made of wool, or as if they had been “nibbled at,” to use an expression of German writers. They consist of inspissated mucus or muco-pus, and contain no air, so that they fall to the bottom of the spittoon. They are probably formed in a vomica, not in a narrow tube through which air was constantly passing backwards and forwards; and have become inspissated by absorption of their liquid constituents, until dislodged by a more than usually violent cough. Accordingly, the general opinion that nummular sputa are distinctive of phthisis is not without reason. But it must be remembered that the necessary conditions for their production are afforded by saccular bronchiectasis, as well as by pulmonary vomicae.

Under the microscope, beside pus-corpuscles and epithelial cells, red blood-discs, elastic fibres, and bacilli are the characteristic elements of the sputa in phthisis.

It has long been known that fragments of pulmonary tissue sometimes occur, in which the shape of the alveoli is still plainly visible. Dr Fenwick, in the 'Med.-Chir. Trans.' for 1866, showed that their detection is much facilitated by boiling the sputum with an equal part of solution of caustic soda (gr. xv to ʒj). This dissolves the mucus in three or four minutes. The resulting liquid is then poured into a conical glass, which is filled up with water, and the deposit which forms is carefully examined in a shallow cell. Dr Fenwick in one case found 800 fragments in the expectoration of twelve hours. We do not expect to find them at so early a stage that there are no physical signs, but the method is of great value when phthisis supervenes upon chronic bronchitis, and the physical signs are apt to be ambiguous. At an advanced stage, when cavities are present, elastic fibres may always be found in the expectoration even though the disease appears to be quiescent.

The detection of the *bacillus* of tubercle in the sputum has now become an important means of diagnosing phthisis. There are many methods of staining these organisms.*

The following procedure, devised by Neelsen, has been found the most convenient in our wards. A minute quantity of the most purulent portion of the sputum is spread in a very thin layer upon the centre of a glass slide and thoroughly dried over a spirit lamp without charring. The slide is then immersed for five minutes in a beaker containing some carbolic fuchsin solution † which has been previously warmed to about 100° F., or till it begins to steam. The slide is next washed alternately in two beakers, one containing dilute sulphuric acid (20 p. c.) and the other tap water, until the colour, which speedily disappears from the preparation when dipped in the acid, does not return when it is placed in the water. By this means the fuchsin is washed from all parts of the sputum except from the bacilli of tubercle, which hold the stain with great tenacity. To produce a contrast-stain the slide is now immersed for two or three minutes in a concentrated alcoholic solution of methylene blue, washed rapidly in methylated spirit, and thoroughly dried over a lamp. The process is completed by placing a drop of Canada balsam on the sputum and covering with a thin cover-slip. The tubercle bacilli, stained red, are now easily seen in the blue field under a sixth objective, with a bright light concentrated by means of a sub-stage condenser.

Hæmoptysis.—This is the most important symptom of phthisis. Mere streaks of blood in mucous or purulent sputa usually come from the gums or fauces or larynx, and not from the lungs at all. Uniformly rusty sputum is the indication of pneumonia, not of phthisis. The characteristic hæmoptysis of pulmonary tubercle is pure blood, liquid or frothy with air.

In many cases this is the first symptom of the disease. The patient perhaps feels a little tickling in the throat, and finds that his mouth contains something with a saltish taste; he looks at his handkerchief, and is horrified to see that it is stained with blood. In some cases he brings up a large quantity at once on the first occasion, but this is not the rule. Hæmorrhage continues more or less for some hours, often recurring with each bout of coughing, and if unchecked by treatment will go on more or less for several days.

* See Dr. Hencage Gibbes's paper in the 'Lancet' of August 5th, 1883; and Dr. Klein's account of Koch's original method, with those of Ehrlich and Weigert, in his 'Micro-organisms and Diseases'; also a full account by Dr. Crookshank in his 'Bacteriology,' and by Dr Woodhead in his 'Practical Pathology.' Like all aniline dyes, the colour is apt to fade, but if the slides are thoroughly washed and treated with nitric acid this may be overcome. Specimens still show perfectly well which were made seven or eight years ago.

† Fuchsin 1 gramme, absolute alcohol 10 c.c., carbolic acid 5 grammes, water 100 c.c.

From the days of Hippocrates it has been thought that the hæmoptysis is in such cases the *cause* of the consumption which ultimately develops itself; and two centuries ago Dr Richard Morton included a *phthisis ab hæmoptoë* among his species of that disease.

“Hoc tamen perpetuo fere observare licet: quoties scilicet hæmoptoë præcedit, phthisin pulmonarem subsequi solere” (‘Phthisiologia,’ lib. iii, cap. v). He gives three cases illustrative of the fact.

Herodotus relates a still earlier history of *phthisis ab hæmoptoë*, probably the earliest on record. One of the generals of cavalry in the great host with which Xerxes invaded Europe in B. C. 480, was Pharnuches, but he never crossed the Hellespont. For as the army was defiling out of Sardis a dog chanced to run under his horse’s feet, and the horse, being frightened, reared and threw Pharnuches. After his fall he brought up blood, and the sickness ended in consumption (πρωὶν δὲ αἷμα ἤμυε καὶ ἐς φθίσιν περιῆλθε ἡ νοῦσος), lib. vii, cap. 88.

The doctrine that blood-spitting is the cause of pulmonary phthisis was revived by Niemeyer. But there is no sufficient evidence that the extravasation of blood into a healthy lung is ever the starting-point of disease. Under various other conditions—as, for example, after injuries to the chest, in purpura, and in chronic heart disease—we have frequent opportunities of observing the effects of pulmonary hæmorrhage and hæmoptysis; and the result is not the development of phthisis.

Niemeyer stated that in one case, four weeks after an attack of hæmoptysis, he found a bronchial tube filled with adherent softening clot; and a similar case was recorded by Dr Weber in vol. ii of the Clinical Society’s ‘Transactions.’ But as a rule, unless a patient has actually been suffocated by hæmoptysis, one does not find any clots in the bronchial tubes after death.

No doubt, hæmoptysis often takes place before signs of mischief in the lungs can be detected; but auscultation and percussion frequently fail to reveal lesions which are really present in the lungs, if they are deeply seated or widely scattered.

Hæmoptysis, again, is often traceable to some violent effort or strain, such as rowing, or running a race, or lifting a heavy cask; but there is little doubt that in such cases the lung was already diseased. We must admit hæmoptysis as a first symptom in the course of phthisis, but not as a first pathological event.

That hæmoptysis is often followed within two or three days by an increase in the temperature, and by signs of inflammation of the lung and pleura is true. A chart given by Bäumlér in vol. ii of the Clinical Society’s ‘Transactions’ shows a rapid rise of temperature from the second morning after the commencement of the bleeding until the sixth day, when it reached 103·8°, and then a gradual fall until the eleventh day, when it became normal. It must be in the experience of every physician that such a febrile attack is frequent after hæmoptysis, and that before it subsides one can often make out distinct signs of consolidation of one apex, which were absent before; but there is every reason to believe that the hæmoptysis is the direct effect of the development of tubercles in the lung.*

* Hæmorrhage may be immediately fatal at a time when there is neither ulceration nor obvious consolidation of the lung substance, and when the only lesions found after death are recent miliary tubercles. It seems very likely that the growth of tubercles in the walls of the alveoli may be attended with an invasion and softening of the coats of many of their capillaries, while at the same time the blood-pressure in them is augmented in consequence of compression of other capillaries.—C. H. F.

When hæmoptysis is considerable, it is almost always due to rupture of a branch of the pulmonary artery crossing the side of a vomica or enclosed in a trabecula; and Rasmussen, of Copenhagen, first made known the fact that, as a rule, the hæmorrhage is preceded by an aneurysmal bulging of the coats of the vessel. A translation of his paper appeared in the 'Edinburgh Medical Journal' for November and December, 1868, and for August and September, 1869. See Douglas Powell's table of cases in the 'Pathological Transactions' for 1871.

Dr Fagge recorded an example in a child under three years of age: which shows that the aneurysm is not the result of atheroma, like an ordinary aortic or popliteal aneurysm. Rasmussen traced the pulmonary aneurysm to the unsupported state of the arterial wall when one side of it is exposed in a vomica. The size of an aneurysm in a vomica varies from that of a pea to that of a nut; but Powell observed one which was much larger. The vomica in which it is found is usually an old one with fibrous walls; and the point of rupture is a little hole or fissure just large enough to admit a probe. Hæmorrhage may have recurred on several different occasions, at intervals of days or weeks, before the fatal issue. Indeed, death is not often the direct result of an attack of bleeding, and the patient may sink exhausted after having ceased to spit any blood for several days; but in other cases he dies almost instantaneously, with a rush of blood from the mouth and nose; or he may be choked by the blood before it appears externally, so that the hæmorrhage is not suspected until an autopsy is made. On the whole, it is remarkable how rarely even profuse hæmoptysis is the immediate cause of death in phthisis.

Instances are not uncommon in which, without having formed an aneurysm, the branch of the pulmonary artery from which fatal hæmorrhage had occurred is found to be perforated by a process of ulceration.

Lastly, in some cases, even of advanced phthisis, in which the lungs contain many vomicæ, it is not possible, after the most careful research, to discover the source of the hæmoptysis.

In cases of ruptured aneurysm of a branch of the pulmonary artery, the blood which is expectorated is usually of a bright red colour. Some writers have thought that this appearance proves it to have been derived either from a bronchial artery or from a pulmonary vein; but it is extremely rare for blood from the lungs to be dark coloured.* In all probability the bright red, arterial appearance which is usually seen, depends not on the blood being derived from a vessel containing arterial blood, but on its having become aerated while it is in the bronchial tubes, where it is freely exposed to the air, as is shown by the frothy state in which it reaches the mouth.

In almost all cases of hæmoptysis, after the bleeding has ceased, there is for some little time afterwards expectoration of clotted blood, or mucus intimately mixed with blood. This gradually alters in appearance, and becomes reddish brown in colour; and this change shows that the hæmorrhage has ceased.

Concretions.—When the tuberculous process in a part of the lung has

* One such case was related by Niemeyer, in his 'Clinical Lectures.' The patient had brought up enough blood to fill three basins within a few minutes; it was found to have a thin frothy layer on the surface, but below this it was coagulated into a dark, almost black cake, like the blood of venesection. Probably the appearance depended on the known physiological fact that effused blood gradually becomes reduced below the surface, the oxy-hæmoglobin yielding its oxygen to the white corpuscles.

become quiescent, and calcification of some of the cheesy material has occurred, the patient may spit up the concretions thus formed, sometimes with a little hæmorrhage. Rühle seems to think that there must be at the time fresh softening, so that a further advance of the disease may be anticipated; indeed, Morgagni long ago stated that the expectoration of pulmonary concretions is an unfavourable symptom. But this belief does not appear to be borne out by present experience, and the expectoration of pulmonary concretions proves at least that a reparative process has been going on. In one case of the writer's it took place at a considerable interval of time after the subsidence of all active symptoms; and the patient in question is living at the present time. It must be remembered, too, that exactly similar concretions may come from a caseous mediastinal gland having opened into the trachea, or one of the bronchi by ulceration. A case in point occurred at Guy's Hospital in 1874; the man, who had been spitting up pieces of calcareous matter every two or three weeks, was admitted into the hospital and died there; and at the autopsy it was found that round the affected gland there was an abscess which had opened into the œsophagus as well as into the right bronchus.

Physical signs—(1) *Formation of tubercles*.—There are no trustworthy physical signs of the first stage of phthisis, which follows the deposition of the bacilli, and the formation of tubercles in the air-vesicles and bronchioles. Even when catarrh of several lobules has begun, the signs may be very slight and doubtful, and repeated examinations at intervals of some days or even two or three weeks, may be required, before one ventures to express a positive opinion.

Amongst the earliest changes to be detected is diminished mobility of the upper part of the chest on one side. Standing behind the patient, with one hand placed lightly below each of his clavicles, we can feel that the expansion of the two sides is unequal; one lags slightly behind the other, or one stops in its movement while the other continues to rise.

On percussing with great care, and closely comparing corresponding regions of the chest, we may make out that there is more or less deficiency of resonance, either in front or behind. It is important to examine the spaces above the clavicles as well as those below them; and by employing different degrees of force in succession one may find that a particular stroke brings out an impairment of resonance better than others. The supra-scapular regions must also be carefully examined; deep percussion is required to bring out differences here.

On auscultation it may be found that the respiratory murmur is not alike on the two sides. If over one apex it is permanently deficient, this points to the lung which is affected. In other cases the presence of early phthisis causes the respiratory murmur to be "harsher" or at least louder than natural. But all loud or puerile breathing is apt to lose its soft quality, and to become more or less harsh or coarse in character; and a loud and "harsh" respiratory murmur, instead of indicating a lesion where it is heard, may be compensatory of one on the opposite side.

Another and more important early sign of phthisis is that expiration is too distinct and prolonged, or there may be a slight pause between it and inspiration.

Again, the inspiratory sound may be interrupted from irregularity in the play of the chest walls. The interruptions may be so frequent that

the inspiratory murmur has been compared with the sound produced by a revolving cog-wheel (*respiration saccadée*); but to this sign, as to harsh breathing, too great importance has been assigned. Walshe observed it at one or both apices when free from consolidation of any kind.*

One of the signs of consolidation of the apex of the lung is increased loudness of the cardiac sounds in the corresponding subclavian region. In some cases a pulmonary systolic murmur (dextro-sigmoid) is heard; but this may be due to traction by adhesions, or to anæmia.

A somewhat later sign of phthisis is the presence of non-consonating moist sounds at the affected apex, often audible only after the patient has coughed. Consonating râles belong to a later stage of the disease.

(2) *Consolidation*.—Much consolidation may take place in the apex of a lung without any physical signs beyond those which are mentioned in the last paragraph, the reason being that while certain lobules become blocked, others still contain air, and it is very long before a uniform infiltration makes the pulmonary tissue as solid as hepatised lung in pneumonia. Still, while the process of solidification slowly advances, dulness becomes more obvious, and the respiratory murmur acquires the bronchial character. At the same time the voice is transmitted by the stethoscope with increased loudness, and the tactile fremitus is increased. These signs are most important; but we must bear in mind what was stated above (vol. i, pp. 1031, 1036) of the normal presence in many persons of bronchial breathing and bronchophony in the first right intercostal space.

Two other conditions may modify the effect of the tuberculous consolidation of the apices. One is the presence of adhesions with thickening of the visceral pleura, to which much of the dulness in phthisis is due; and the other is complementary emphysema of the anterior edge of the affected lung, which keeps the percussion note resonant.

Moist sounds may or may not accompany the tubular breathing of phthisical consolidation. When present they have a consonating character. A common combination is for the inspiration to be attended with râles, which obscure the bronchial breathing, and immediately afterwards a blowing expiratory sound is heard, but no râles.

When the disease has existed for several months, the regions above and below the clavicle on one or both sides are usually found to be flattened or slightly hollowed. This is probably at first due to wasting of the pectoral muscles, and want of full inspiratory expansion; but afterwards to shrinking of the apices in addition.

(3) *Excavation*.—The bronchial breathing in a case of phthisis may become more pure, more blowing, more tracheal in character, without there being any further change in the lung than increasing consolidation. When we hear well-marked amphoric breathing, we are seldom wrong in diagnosing the presence of a large vomica. One might have anticipated that the percussion-sound over a large empty cavity would be resonant, or even tympanic. This, however, is very seldom the case; the thick adherent pleura and the condensed lung-tissue round the wall of a vomica serve effectually to

* In one of his cases the cog-wheel rhythm was probably due to the action upon a healthy lung of an irritable heart; for the separate sounds which make up cog-wheel breathing may be synchronous with the cardiac pulsations ('Revue mensuelle,' 1877). It is, however, quite probable that when a portion of the lung is solidified, the shock given by the beating of the heart, directly or through the blood-vessels, may produce a greater effect than on normal lung-tissue.

check the vibrations of the thoracic parietes, so that a toneless flat sound is heard on percussion.

A frequent peculiarity of the percussion-sound over a large vomica is that it resembles the noise produced by jingling coins, or by striking the loosely clasped hands over one's knee — Laennec's *bruit de pot fêlé* (vol. i, p. 1026). For its production in a perfect form the walls of the cavity and the thoracic parietes must be elastic and yielding, the percussion-stroke must be somewhat heavy, and the cavity must communicate freely with the bronchial tubes, and these again with the external air through the open mouth.* There are, however, other conditions in which this percussion-sound may be heard. It is more or less present if the finger which is percussed does not lie flat on the surface of the chest; according to Dr Gee, it is sometimes obtained over the upper part of the front of the chest in cases of pleuritic effusion, sometimes over islets of unsolidified lung embedded in tissue hepatised in acute pneumonia, sometimes in cases of malignant tumour. In healthy children also it is often heard when the child's mouth is open and a sudden percussion-stroke drives in the yielding parietes of the chest. But, when combined with other signs of phthisis, the cracked-jar sound is an almost certain proof of a large empty cavity in the lung.

A sign which occasionally attracts the notice of the patient himself as well as of the physician, is that the sounds of the heart can be heard like the ticking of a watch, sometimes at a distance of several feet.† This singular sign would doubtless be less rare than it is, were it not that a cavity of sufficient size for a quantity of air to be driven out of it by each beat of the heart very seldom exists, except in the upper lobe.

On auscultation over a cavity the ordinary inspiratory murmur is never heard; occasionally there is silence; most often loud tubular or amphoric breath-sounds, unless they are rendered inaudible by consonating rattles.

What is called after Laennec, *le souffle voilé*, is a high-pitched blowing sound heard at the end of inspiration. It was regarded by Skoda as a sign of a large vomica; but Dr Fowler considers it rather indicative of sacculated dilatation of the bronchi.

After a severe fit of coughing, when inspiration occurs again, there may sometimes be heard a continuous, rather sharp, *suction sound*, which follows the ordinary inspiratory part of the breath-sounds, as the air enters again the half-emptied vomica.

Another sign heard over a cavity was described by Seitz as the *metamorphosing murmur*.‡ It is marked at the commencement of inspiration by an unusually harsh sound, which lasts only during one third of the inspiratory period, and gives place during the remaining two thirds to soft bronchial breathing, accompanied by an echo.

The moist sounds are often "large" enough to be called gurgling, and various *metallic phenomena* may present themselves in a large empty vomica, like those of pneumothorax (cf. vol. i, p. 1112).

Vocal resonance often amounts to pectoriloquy; but Walshe rightly in-

* The cracked-pot sound was exquisitely marked in a patient of mine who had, outside the thorax, beneath the pectoral muscles, an abscess-cavity which contained air and communicated with a pneumothorax by a hole through the intercostal muscles.—C. H. F.

† Many years ago my father showed me a case of this kind which had come under his observation. The sounds were sometimes audible across a good-sized room, but I found that when the patient, a young woman, was made to close her mouth, I could instantly stop them by pressing together her nostrils. Just such a case was brought under the notice of the Clinical Society in 1880, by Dr Frederick Taylor.—C. H. F.

‡ See v. Niemeyer's lectures on 'Phthisis' (New Syd. Soc. tr.), p. 54, Bäumler's note.

sists on the fact that over a large cavity there may be dead silence, not only respiratory, but also vocal.

One very rare effect of excavation of the lung is the production of subcutaneous emphysema from perforation of a vomica. A case in point occurred at Guy's Hospital in 1882. The patient had been slowly sinking for weeks, and shortly before his death there was a slight crackling below the clavicle and at the root of the neck. Several similar instances are on record. As pneumothorax is not present, it must be assumed that ulceration extends through both layers of the pleura, the space between having been previously closed by adhesions.

(4) *Involution*.—The physical signs of retrogressive or obsolete phthisis (fibrous or cirrhotic tuberculosis of the lung) vary widely. Shrinking of the upper part of the chest may go on until the clavicle and shoulder are obviously drawn down. The cyrtometer will show the diseased side to be much smaller than the sound. The percussion-sound is usually very dull; indeed, as Rühle remarks, extreme dulness in phthisis is usually a favourable sign, for it means that dense fibrous cicatricial tissue has taken the place of patches of tuberculous inflammation. The breathing is tubular, and the expiration prolonged; but râles are most often absent.

The heart becomes uncovered by retraction of the lung, especially if the left is the one more affected. Its impulse may be seen and felt over a much more extensive area than usual, even as high as the third intercostal space. The stomach may be drawn upwards to the level of the sixth or the fifth rib. Or, if the right lung is diseased, the heart's apex may be displaced to the right side of the sternum, and the liver may be dragged up as high as the fourth rib.

(5) *Extension*.—While watching the changes in the part first affected, we must also look out for signs of invasion of other parts, or of the opposite lung. The frequency of excavation in the apex of the lower lobe makes it advisable to listen carefully on a level with the shoulder-blade, after it has been drawn outwards by the patient crossing his arms.

In advanced cases the question is often not what parts of the lungs are diseased, but what parts remain capable of carrying on respiration. It is surprising how small an area, at the extreme base of one lung, may give a healthy pulmonary murmur. Here it will probably be loud, compensatory or "puerile" breathing.

One must not over-estimate the significance of rhonchi and râles when heard over the whole of the back of a lung, for they do not prove that the corresponding lung-substance contains more than scattered or clustered tubercles, or even more than temporary concomitant catarrh. In cases of acute and general miliary tuberculosis it has often appeared during life that large tracts of the pulmonary tissue were breaking up, and yet after death the pulmonary tissue between the tubercles has been found still crepitant. It is remarkable how, on first being called to a case of phthisis, we may find such extensive signs as well as active symptoms that we regard the case as being in the last stage, and yet after a few days the acute catarrh passes off, the fever clears up, and the patient only shows permanent disease of one apex.

But in the majority of cases of phthisis the discrepancy between physical signs and *post-mortem* appearances is in the opposite direction. Clinically disease is perhaps discovered in the upper lobe of one lung; the autopsy shows that nearly the whole of that lung is affected, and also the upper lobe

of the other. We must admit the fact that the presence of well-marked disease in one lung adds greatly to the difficulty of detecting early mischief in the other apex; no doubt because one has lost the standard of comparison on which one is accustomed to rely. On the whole, where there are well-marked signs of advanced phthisis in one lung we may be pretty sure that there is early phthisis in the other. It is extremely rare to find vomicae on one side and no tubercles on the other. This was, however, the case in a young mulatto, who died under the writer's care after an unusually rapid course of pneumonic phthisis; one lung was full of tubercles, the other was only œdematous.

Diagnosis.—The recognition of phthisis, if based, as it should be, upon symptoms as well as signs, is often easy. But there may be the greatest difficulty in arriving at a right conclusion, and sometimes the only prudent course is to reserve one's opinion for a time. In practice the doubtful cases are generally those in which physical signs are either wanting or at least slight and obscure, so that one hesitates as to whether the disease is pulmonary at all, or whether there is not some deeply seated new growth, or disease of the internal lymph-glands, or of the thoracic duct, or of the adrenals, to account for the patient's pallor, weakness, and wasting.

Again, we may sometimes be in doubt whether the patient, if a man, is suffering from syphilitic cachexia, or is the victim of hypochondriasis and aggravated dyspepsia; if a woman, whether she is only hysterical. In all cases of this kind the thermometer is of the greatest value. One hysterical affection, which has often been mistaken for phthisis—the “anorexia nervosa” of Sir William Gull—has been already described (vol. i, p. 971).

When the suspicion of phthisis is based on the fact that the patient “spits blood,” a glance at the sputum is sometimes sufficient to decide the question. What is expectorated may be a rather slimy liquid, uniformly tinged of a pink or purple colour, so that it looks like plum juice. It is, in fact, saliva and mucus from the mouth, and the blood has come from the gums or cheeks. Rühle remarks that this sort of hæmorrhage often occurs in the night, from the patient making sucking movements of the lips and cheeks during sleep; and thus the pillow may show stains of blood, the origin of which seems at first inexplicable. The writer was once sent for early, to see a patient formerly visited for symptoms of phthisis, who had after many years spat blood again. There was no other sign of fresh mischief, and the blood was the result of slight and forgotten epistaxis during the night.

Again, sanguineous expectoration may be due to the rupture of small vessels at the back of the fauces during violent coughing, or “hawking up” of phlegm. Varicose veins in the pharynx sometimes reveal the source of hæmorrhage.

Hæmoptysis sometimes occurs in elderly persons, not from phthisis or aneurysm, but from degenerated vessels—whether bronchial or pulmonary is not ascertained. This occurred in the writer's knowledge a few years ago, and almost at the same time in two eminent physicians; and it was the subject of a paper read before the Medical Society by the late Sir Andrew Clark. The condition is clinically allied to that which leads to cerebral hæmorrhage, epistaxis, and hæmaturia; but it is not certain whether the vessels are atheromatous or the seat of arterio-capillary degeneration.

Hysterical girls and more responsible persons will sometimes delibe-

rately simulate hæmoptysis, and as their own blood is most convenient for the purpose, the microscope will often fail to detect the fraud.

True hæmoptysis may occur as a symptom of cardiac disease, of purpura, or any of the severe forms of anæmia; and as the result of fractured ribs and penetrating wounds of the chest; also from aneurysm, embolism, or cancer of the lung.

Lastly, hæmoptysis, like other hæmorrhages, has been supposed to be often vicarious of the catamenia. Watson cites a case which was observed by Pinel at the Salpêtrière, that home of all that is marvellous in disease, in which a woman was said to have menstruated through her lungs from the age of sixteen to that of fifty-eight, often to the extent of two quarts of blood during a period of two days, while she nevertheless remained plump and healthy. Rühle will only admit that in patients who are already phthisical suppression of the catamenia (or of a hæmorrhoidal flux) may be followed by vicarious hæmoptysis. He mentions cases in which this recurred at intervals of from four to six weeks, until a few leeches were applied to the anus with a corresponding regularity. Such determination of hæmoptysis in a phthisical woman to the menstrual period is possible enough, though it is certainly far from common. But that a woman with healthy lungs, suffering from amenorrhœa, should discharge blood by the trachea instead of the vagina is a statement which has not yet been shown to be true even as an exception.

In children, where we scarcely ever have the advantage of searching the sputum for bacilli, the difficulty of diagnosis is often great. One sign, seen in the most chronic cases of phthisis, is clubbing of the fingers; but this may be due to bronchitis or bronchiectasis, to cirrhosis of the lung, to congenital or acquired valvular or pericardial disease, or to empyema.

Cases in which in the writer's experience phthisis has been wrongly diagnosed have been apical pneumonia (lobar or catarrhal), pulmonary syphilis, and empyema in children. Those in which it has been overlooked have been mistaken for enteric fever of unusually protracted course, for primary bronchitis in elderly people, for tuberculous meningitis, or for idiopathic anæmia. Phthisis is one of the three maladies which Sir William Gull used to say every one sometimes failed to discover—the other two being syphilis and scabies.

Cardiac disease in children, gastric ulceration, and chlorosis often simulate phthisis in aspect, but can always be distinguished by careful and repeated physical examination.

At present the application of the Röntgen rays to the diagnosis of tuberculous consolidation of the lungs has not been of more than scientific interest, but "stereoscopic skiagraphy" has, in Dr Walsham's hands (1899), given promise of future usefulness.

Course and events.—Phthisis varies greatly in the rapidity of its progress, but its duration is almost always for many months, and sometimes for many years. Trousseau used to say that the only *phthisie galopante* is military tuberculosis of the lungs.

The most rapid case of true phthisis on record is probably one related by Traube in the 'Berliner klin. Wochenschrift' for 1867.

A man, aged twenty-eight, died, after only thirteen days' illness, of "acute tuberculous (caseous) pneumonia." The attack began with rigors and pyrexia; a few days later hæmoptysis set in and continued. At the autopsy all parts of the left lung presented

patches of lobular hepatisation, the centres of which were caseating, especially in the upper lobe; a similar affection in an earlier stage existed in the right lung. But even in this case both apices showed traces of old lesions.

Several cases have occurred in Guy's Hospital, in each of which there was a definite history that the duration of the patient's illness, from its commencement to its fatal termination, was only from five to twelve weeks. In two instances the attack was attributed definitely to a chill: one man said that he got wet through while working in a potato-field, after which he shivered and became hot, and was never well again; the other, that on a particular occasion he slept with his window open. In almost every one of these cases vomicae had formed before death, especially in the upper lobes.

The sudden commencement of some of the rapidly fatal cases of phthisis is of great importance in regard to their diagnosis from cases of acute pneumonia of the apex; for the most serious errors of diagnosis have been made between the two diseases. The mode of onset usually affords a means of arriving at a right judgment, but this is not always sufficient. Until the case develops itself, the best distinction is perhaps that of Traube, that even in the most acute phthisis bronchial breathing is not heard until much later than in pneumonia of the apex—not until the end of the second week, or later. If, however, one finds extensive consolidation in a case of phthisis when it first comes under one's observation, there is a possibility that it may, in part at least, be the result of intercurrent pneumonia. For when fibrinous pneumonia occurs in a person who already has phthisis, it seems to run as favourable a course as in a healthy subject.

Even acute phthisis ("phthisis florida") may, instead of going straight on to a fatal termination, become arrested, and afterwards run a chronic course.

Rühle relates a case in a girl, who seemed to have but a short time to live when she was transferred to his charge from that of Niemeyer, his predecessor at Greifswald. Yet her symptoms subsided, she was discharged from the hospital with signs of a cavity in the left upper lobe, and did not die until the following year, having in the meantime given birth to a child. Pregnancy possibly checked the progress of the disease.

The course of ordinary chronic cases is, almost without exception, interrupted by intervals during which the patient improves. Cough may almost disappear; even the evening temperature may become normal; the appetite returns, the face is no longer pale, and the ordinary weight is regained. It is true that this favourable change commonly takes place under medical treatment, and is a proof of its value; but often instead of inspiring reasonable hope, and more strict observance of the physician's advice, the improvement leads the patient to suppose himself cured, and to throw away all rules and precautions.*

Duration.—Widely different estimates as to the duration of phthisis have been formed by eminent physicians. Laennec, Andral, Bayle, and

* In January, 1874, a hatter, aged thirty-seven, who said that he had been ailing for six months, came to me with signs of phthisis at both apices. His morning temperature was 101.4°. His father had died of consumption. Notwithstanding my urgent advice to give up work, he did not rest for a single day. The only difference he made was that instead of living away from his workshop in the Borough, so that he was exposed to changes of temperature in going backwards and forwards, he now slept in the same building. For a week or two the physical signs increased, moist sounds becoming audible all over the left lung. But his symptoms quickly improved, and by the end of May he was as stout as ever, and said that he felt nearly well. The signs at the apices, however, still remained. In the following year I heard that he was in good health, with only a little occasional cough. But in 1879 his symptoms returned, and he died in September, 1881.—C. H. F.

Louis each put the "mean duration" at about two years. Austin Flint, in Philadelphia, found that, excluding acute tuberculosis, the average duration of phthisis was thirty-three months. These estimates apply to the first forty years of the century. At present the duration of phthisis is much longer. Dr Pollock, analysing 3566 cases observed by him at the Brompton Hospital, found that the "average duration" of these cases while under observation was more than two years and a half, and in the course of that time only 127 ended fatally. What was the real average length of the disease among the whole number of cases he could not tell, but it must clearly have been much longer. It is, however, difficult to believe that these cases represent the ordinary course of the disease. There must surely have been a large proportion of exceedingly chronic cases, and cases running a rapid course must for some reason have been unusually rare. Still more remarkable are the statements made in vol. liv of the 'Med.-Chir. Transactions' as to the duration of life among 1000 cases of phthisis seen by the late Dr C. J. B. Williams in private practice between 1842 and 1864. Of the patients in question 198 were known to have died; in them the average duration of the disease was nearly seven years and three quarters. In the remaining 802 patients, who were alive when last heard of, its average duration had already been more than eight years. Among these cases, however, none were included which had not been at least one year under observation; and this restriction, besides keeping out of the list all rapidly fatal cases, doubtless weeded it of most who failed to improve under treatment, and therefore ceased to attend.

That life is sometimes maintained for a great length of time after phthisis has developed itself has long been well known. Sir Thomas Watson alludes to a patient of Dr Gregory's who was at least seventy-two years old when he died, and who from the age of eighteen had never been free from symptoms, "being often hectic, and frequently spitting blood."

The writer has a patient who furnishes almost a parallel history. He comes of a very phthisical family, and developed signs of the disease when under twenty years of age. He then went a voyage to Australia for his health. After repeated hæmoptysis, he recovered enough to marry at the age of twenty-five, and in 1875, when about forty years old, came under the writer's care. There were the signs of consolidation of one apex, and of less advanced disease of the other lung. He has again and again spent the winters at Bournemouth, at Algiers, and in Egypt; again and again has had attacks of pyrexia, bronchitis or hæmoptysis, but he has survived them all, has become stout, and now at sixty-five is a grandfather, and considers himself out of the physician's hands.

Immediate causes of death.—The fatal termination of phthisis is sometimes sudden and unexpected. In 1866 a gentleman aged twenty-six, who had long been ill, went up to London from Brighton one day to transact some business. At the London Bridge station he was seized with alarming symptoms, and was taken down to Guy's Hospital, where he died within a quarter of an hour from the beginning of the attack. In 1868 a labouring man aged twenty-three, who had been indisposed for some time, was at his usual work in the Borough, when about 2 p.m. he began to suffer from dyspnœa; this rapidly got worse, and he was carried to the hospital and died in two hours. In neither case did the autopsy show why death should have occurred at that particular time.

Pneumothorax often brings more or less immediate danger to life. Nevertheless, when the immediate effects are got over, the consequent collapse of the lung appears to be unfavourable to rapid progress of the disease; so that if the other lung is but slightly affected the patient's con-

dition may, at least for a time, improve. Some practitioners, acting on this hint, have ventured to puncture the pleura and thus produce pneumothorax, with the hope of checking severe hæmorrhage.

An accident, happily rare, that may bring phthisis to a sudden close is pulmonary *embolism*, resulting from thrombosis of the femoral vein.

Syncope is sometimes the cause of death, or sudden exhaustion of the respiratory centre. From one cause or the other, it is not very uncommon for consumptive patients to be found dead in bed.

In other cases phthisis ends fatally by tubercles invading other organs, from tuberculous meningitis or peritonitis, from tuberculous pyelitis, or from solitary tubercle of the brain.

It is a remarkable fact that *hæmoptysis* is seldom the immediate cause of death in phthisis. Considering that it is present in the great majority of consumptive cases, it is exceptional to find a patient "choked in his own blood," although it occasionally happens.

The most serious symptom towards the last is often *diarrhœa* from tuberculous ulceration of the intestine, or, less commonly, the *dysphagia* and other distressing symptoms produced by a like affection of the larynx.

Of non-tuberculous affections, the following are not infrequently fatal complications of phthisis: parenchymatous nephritis, typhlitis with peritonitis, and abscess of the brain.

The coincidence of *fistula in ano* with phthisis is one which requires brief mention. Dr Pollock found it occur far more often in males than in females, and most frequently in persons from thirty-five to forty-five. The disease of the lungs has generally advanced to excavation before the fistula appears. He believed with other physicians of experience that in such cases no operation should be attempted; for, even when it is successful, the phthisis is apt to become more active two or three months later. On this point, however, there is difference of opinion, and there is no evidence that the cure of a fistula in a person not already consumptive renders him liable to phthisis.

Finally, *lardaceous degeneration* has a share in bringing many cases of phthisis to a close. If the intestines be involved, an intractable diarrhœa may result, which cannot be distinguished during life from that of tuberculous ulceration. If it affects the kidneys dropsy sets in, and the patient acquires the aspect of Bright's disease. Indeed, tubal nephritis sometimes comes on in phthisis without there being any lardaceous change discoverable in the renal glomeruli or vessels. According to observations made by Dr Theodore Williams ('Med.-Chir. Trans.,' 1882), the occurrence of albuminuria in phthisis has the effect of making the range of temperature lower.

Cases of recovery.—Phthisis was once regarded as almost certainly fatal; we now know that this is not the case, partly through recognising its existence in early and curable stages, partly through the knowledge derived from morbid anatomy.

Again and again we find in the deadhouse traces of obsolete phthisis in adhesions, cicatrices, and calcareous deposits which occupy the apices of one or both lungs in the bodies of those who have died of some acute and independent injury or disease (cf. p. 42). Such persons must have suffered from phthisical symptoms some time in their lives, and now and then we have direct evidence that in early life they spat blood and were treated as cases of undoubted phthisis.

. Hospital practice gives too unfavourable an estimate of the prospects of consumptive patients; for the poor seldom apply for either indoor or outdoor relief until the early stages are past and the disease has gained a firm hold on one or both lungs. In private practice we see cases earlier, and the desirable treatment can be better followed out. But here many of the cases which recover come to us in what used to be called the "pretubercular" stage, when we rather forebode than diagnose the fatal disease. They improve under treatment, and we often do not see them again.

Notwithstanding these drawbacks few physicians have not been fortunate enough to see unmistakably phthisical patients improve, recover health, and continue many years without return of their symptoms.

The following are some of the cases of "cured" or arrested phthisis which have come under the writer's notice.

A lad of seventeen, tall, pale, and delicate in appearance, with cough and physical signs of catarrh at one apex. Was sent to Algiers, and returned two years later stout and well, with no symptoms and no physical signs of the disease. He remained well when seen three or four years later.

The patient above mentioned (p. 59), in good health after forty years of phthisical symptoms and signs.

A young man about nineteen, whose mother died of phthisis, who was of typically phthisical aspect, and who developed cough with anæmia and loss of flesh. He was sent into the country to live in the open air for several months, lost his cough, gained weight, and recovered. He had a "break-down" with suspicious symptoms when about thirty-five, but quickly recovered, and remained free from all phthisical symptoms. At the present time he is above sixty years old and in good health.

A young theological student was attacked by hæmoptysis and other signs of consumption. He was sent to Montpellier, where he spent more than a year, and finally recovered his health. He lived a most useful and laborious life, and died from disease of the kidneys and of the cerebral vessels at eighty-one.

A young physician of great distinction and greater promise was attacked with pleurisy and other symptoms of unmistakable tubercular character: there had also been several cases of phthisis in the family. He went out to an elevated region in South America, and has continued well and able to practise for more than twenty years.

A medical student with decided symptoms of hereditary phthisis, after careful treatment for seven years in this country, settled at a watering-place in the south of Europe, and has pursued his profession there for the last eighteen years.

Prognosis.—To give a correct prognosis in phthisis is no light matter; and those physicians who have the largest experience are those who most strictly abstain from prediction.

We have followed usage in dividing the *anatomical* course of phthisis into three stages: the *first* stage including the origin, formation, and growth of tubercles, with the lobular catarrh they set up; the *second* their "softening," caseation; the *third* consolidation, or the process of excavation, with formation of vomicæ. But the moist sounds which are supposed to indicate "softening" are apt to be fallacious. These pathological stages have reference only to the local process in certain parts of the lungs, and not to the disease as a whole; for while vomicæ exist in one or both apices, fresh tubercles are being formed lower down or in the other lung. To speak of these as *clinical stages* of phthisis leads to a misconception of their significance in prognosis. To a patient, and to his friends, it must seem obvious that the third stage of phthisis must be the worst. And yet it is no exaggeration to say that the contrary would often be nearer the truth.

The first condition which we must try to estimate in forming a prognosis in a case of phthisis is its greater or less tendency to advance rapidly in that particular patient. In different persons the differences in this respect

are enormous, and can seldom be predicted. As a rule, however, the progress is quickest in those who have a strong inherited tendency to consumption, and the progress is most retarded in those who keep up their appetite and weight.

The death of both parents from consumption is the most serious and disheartening fact of all; next, perhaps, that of the mother; then that of the father, and much less so that of one brother or sister, particularly in a large and otherwise healthy family. In the writer's experience it is rare to find more than one case of phthisis among collateral members of the household unless one or both of the parents has been also diseased. One case among several children may be accidental; more than one may be the result of infection. Now and then we find one member of a phthisical family free from the disease to which all the rest have fallen victims.

Next to hereditary tendency may be put the age of the patient. In childhood phthisis runs, as a rule, a rapid and fatal course; but after the earlier period of adult life has been passed (15—25), it is certainly, as a rule, less acute, and often more amenable to treatment.

In women we more often meet with acute and, so to speak, "malignant" cases from the first; at least the writer's best cases of recovery have been in men.

Physical signs of extensive disease of the lungs are so far unfavourable, but one not unfrequently meets with what seems to be extensive phthisis on both sides of the chest, which afterwards subsides; a temporary catarrh disappears and leaves only a comparatively small district of solidified lung. Moreover, some of our worst cases are those of loss of flesh, pyrexia, and night sweats in young adults, where the physical signs are for a long time extremely slight. On the other hand, some of the most chronic cases are marked by the characteristic signs of a large cavity coinciding with good general health. The mere fact of a vomica large enough to give typical signs of its presence in a patient who is going about, eating, and sleeping, and keeping up his weight, shows that his tissues have great power of healing, and that he has had tuberculous disease for a long time without being killed by it. Hence the truth of what seems the paradox above stated, that the condition known as the third stage may justify a better prognosis than that known as the first.

To put the matter briefly, one may say that, apart from family history, the less important facts for prognosis in phthisis are those which concern the lungs, and the more important those which concern the whole body. While a patient puts on flesh and is free from fever, he is doing well, whatever takes place in his chest; while he is losing weight, and suffers from anorexia, sweating, diarrhoea, and hectic, he is going down hill, however little sign of disease auscultation of his chest may show.

The most important object in the prognosis, as in the treatment of phthisis, is not the respiratory but the nutritive organs; we must cure it, if we cure it at all, by the stomach.

The formation of a large cavity takes a long time, especially if its walls are to acquire a smooth lining. Hence, whenever the morbid process spreads with much rapidity through one or both of the lungs, the opportunity for such cavities to develop is wanting. In other words, the fact that a case presents the physical signs of a single large cavity is proof that its course has been such as warrants a comparatively favourable prognosis. As a matter of experience, patients with single large vomicae often go on

year after year with but little change in their condition, and even with enjoyment of life.

Again, one is compelled to speak very guardedly of the probable duration of phthisis, if physical signs indicate that the morbid process is still actively going on in any part of the lungs, whatever may be the stage to which it has reached in the apices. One must not forget that, although in the lung first affected its progress may have been slow, it may hurry on to a fatal termination when it passes to the other side of the chest.

The degree of severity of the general symptoms is very important in regard to prognosis, especially the rate of the pulse and the height of the temperature. But the existence of pyrexia, as has been shown by Dr Theodore Williams, is not incompatible with gain of weight—nor even, we may add, with the subsidence of many of the other symptoms of the disease—provided that the patient eats and digests well.

The majority of cases of acute or “pneumonic” phthisis occur in young subjects, whereas “fibrous” phthisis, the most chronic form of the disease, is more frequent in those who are advanced in years. This seems to have led to the idea that the prognosis is more favourable in proportion as the patient is older, but this does not quite follow.

With respect to hæmoptysis it is seldom directly and immediately fatal. It often occurs in some of the most chronic and favourable cases; and even when it appears early, or as the first recognised symptom, it does not seem to denote a peculiarly rapid or intractable course of the disease.

Cough is not itself a symptom of bad omen, but profuse expectoration is; and still more, chronic diarrhœa.

Night sweats with hectic fever indicate a rapid and fatal course. Laryngeal ulceration is another grave complication, and scarcely less so, tuberculous pyelitis or orchitis.

Phthisis in children.—Consumption is comparatively rare under puberty; more frequently children are the subjects of general tuberculosis, particularly of the peritoneum and the pia mater. In such cases the lungs are often found full of tubercles; but these are disseminated, and at the same time they are found almost constantly in the spleen, very frequently in the liver and kidneys, and always in some of the lymph-glands, mediastinal, cervical, mesenteric, or lumbar. Cases of more ordinary tuberculous catarrh of the apex, with caseous softening, run usually a rapid course with much pyrexia. This unfavourable prognosis of phthisis under puberty is in curious contrast with the far from hopeless forecast one may give in cases of tuberculous enteritis and peritonitis, and the good prognosis, under modern treatment of caries, arthritis, and tuberculous disease of the lymph-glands or the skin in children.

Senile phthisis.—Chronic tuberculous disease of the lungs is far from infrequent after fifty or even after sixty, although it seems so in comparison with its far greater frequency at earlier ages of adult life.

In the writer's experience, however, it is absolutely as well as comparatively rare for phthisis to be acquired late in life. Almost all the cases he has seen above middle age have proved to be the reawakening of long-past phthisis in youth.

The following is a case in point. A gentleman of sixty-nine, who had led a long and very active life in the country, retired to London, and soon began to be troubled by violent

fits of coughing, ending in vomiting. He consulted several physicians, and was supposed by some to have whooping-cough, by others asthma, or aneurysm, or malignant disease. There was some emphysema, but no physical signs of phthisis; he gradually, however, lost flesh. The pulse was frequent, and there was occasionally slight pyrexia in the evening. Still there was no hæmoptysis, and no bacilli could be found in the scanty mucous expectoration. The absence of pain and other symptoms of thoracic aneurysm seemed to exclude that belief, and the absence of pleural effusion and lymphatic enlargement were against the presence of cancer. The larynx was unaffected. At last, after several visits, the fact was elicited that between eighteen and twenty he had been in "delicate health," had a slight cough, and once (or perhaps twice) brought up a very little trace of blood in the effort of coughing. This patient became gradually emaciated, and the diagnosis of phthisis was confirmed by an examination of the body after death.

Phthisis in old people is usually chronic, with much bronchitis and little fever or hæmoptysis. The symptoms are chiefly pulmonary, and, although the larynx or intestines may be ulcerated, it is very rare for the lymph-glands or serous membranes to be tuberculous. The right side of the heart is sometimes dilated, and some degree of anasarca may follow—a condition, which is extremely rare in cases of phthisis affecting children.

Ætiology.—We have already stated the evidence that tuberculosis is a specific disease, with the constant presence of a definite bacillus, and found that it fulfils the criteria laid down (vol. i, p. 368). We have also in the present chapter endeavoured to show that pulmonary phthisis is pathologically one and the same—tuberculous disease of the lungs. It follows that phthisis depends on the presence of Koch's bacillus, and the conclusion is corroborated by its presence in the sputa being the most trustworthy means of checking our diagnosis in doubtful cases.

The discovery of the bacillus in all tuberculous lesions has completely altered our views as to the origin of phthisis. Yet the constant presence of a microbe is far from deciding the whole ætiology of a disease. In the most exclusively and directly contagious maladies, syphilis, typhus, smallpox, none has as yet been certainly discovered; while erysipelas, enteric fever, and cholera, in each of which one is present, are not infectious in the popular sense of the word.

Phthisis requires, beside the bacilli, a reacting living organism; and in tuberculosis we have, as in other specific diseases, to determine the power of resistance of the tissues to its invasion. This varies with different contagia. That of smallpox and of syphilis is so efficient, that once taken into the lymph-channels or the blood it probably produces the disease in every human being. But even here what is so certain a poison in man fails with the lower animals; and the most efficient contagia may fail in protected organisms. Probably most persons are more or less immune to enteric fever and to erysipelas, and many are so to scarlatina.

It seems likely that the microbes of tubercle are so widely diffused that most dwellers in cities, at least, must again and again be exposed to their action; yet comparatively few become tuberculous. Hence we must, in addition to the necessary "exciting" cause, the bacillus, recognise "pre-disposing" causes in whatever weakens the resistance of the organism generally or of the lungs in particular, and so allows of the "efficient" cause of all the symptoms of phthisis—*i. e.* the lesions which follow the permanent lodgment and multiplication of the bacillus.

That consumption is not, as a rule, directly communicated from one person to another, seems clear from general observation. A statement was published in 1867 by Mr Vertue Edwards, who had then for seventeen years

been resident medical officer at the Brompton Hospital. In that period he remembered personally fifty-nine resident medical assistants, whose duration of office averaged quite six months. Of these he believed all but two to be alive; one had died of aneurysm, one of some cause unknown; three, still living, were said to be consumptive. Very many nurses had been in residence from periods varying up to eight, twelve, or even twenty-four years. Of the head nurses, who slept each in a ward of fifty patients, only two were known to have died—one of apoplexy; the other of phthisis. No under nurse, so far as he was aware, had died of phthisis. The matron and her two predecessors, as well as the chaplain and his two predecessors, were all alive. Of the physicians, whether for in-patients or out-patients, all were living except two; one had died of causes unconnected with disease of the lungs, the other of unknown cause, after twelve years' absence from the hospital. Mr Edwards himself was lately living at a good old age.

In Dr Weber's cases, recorded in the Clinical Society's 'Transactions' for 1874, the disease seemed to pass immediately from husbands to their wives. The husbands, all of whom were phthisical before marriage, were nine in number; but the deaths from phthisis among their wives were as many as eighteen: one lost four wives in succession, one lost three, four lost two each, three lost one each. In seven out of the nine husbands there was a decided family taint; the wives were with one exception free from any such taint, and they were all healthy at the time of marriage. The disease ran a very rapid course in all the wives, terminating in several instances within twelve months, and being never prolonged beyond eighteen months. It was not caused by anxiety or fatigue in nursing the husbands, for the husbands were all apparently well, and none of them succumbed to phthisis until long after their wives. Almost all the wives bore children to their husbands, so that it is perhaps possible that infection took place through the foetus; but about the health of the children nothing is said.

Infection from husband to wife might take place through the ovum or directly from a tuberculous epididymis lesion, but there was no evidence of either; and it seems more probable that the infection was transmitted by the breath.

On this subject see the facts recorded in the 'Collective Investigation Record,' "Report on the Communicability of Phthisis;" and also a paper by Dr Burney Yeo, in which he skilfully states the case for regarding phthisis as a contagious disease ('Brit. Med. Journ.,' April 18th, 1885). But Dr Longstaff's statistical correction of the figures there adduced shows, as Dr Ransome remarks, that the proportion is not greater than would result from merely accidental coincidence.

The fact that phthisis does not commonly spread from a patient who remains in his home to brothers, or sisters, or other relatives, is the more striking because they must be supposed to have inherited more or less predisposition to the disease.

The possible contagion of phthisis is no new doctrine. Morton wrote in 1697, "I have often found by experience that an infected person may poison a bedfellow by a kind of miasm like that of a malignant fever." In Italy consumption has always been regarded as a contagious disease; and now the presence of bacillus of tubercle in cases of phthisis affords a ready explanation of its being communicable. That it is not readily communicated seems probable, but it certainly is so under favourable conditions, as in the case of husband and wife, and other persons living in close and habitual contact.

In discussing the subject of tubercle in general (vol. i, p. 369), we saw that direct infection by contact with another patient is infrequent in the ætiology of tuberculous diseases. Probably the most frequent mode of infection is the air; for the dried sputum of phthisical persons preserves the bacillus for a long time, and in crowded towns there must be abundant opportunities of infection from so common a disease.

Conveyance by air.—Breathing air rendered impure by overcrowding or by defective ventilation may perhaps act indirectly by weakening the resistance of the tissues; but it certainly acts directly, by increasing the chance of infection, since few work-rooms or dwelling-houses would continue many years without a phthisical inmate. Organic particles differ from gases like carbonic acid in being far less readily diffused through the air; they cling to textile fabrics. Every hospital physician is familiar with the peculiar odour belonging to the clothes of the women of the poorer classes; one perceives it as soon as they enter the out-patient room. The organic matters cannot be removed by merely causing a current of air to blow through a room for a few minutes in the day; still less will they escape through a door towards which there is no active draught. Probably the only way of getting rid of them is by free oxidation; and most rapidly in full sunlight.

Strictly speaking, overcrowding and defective ventilation are not convertible terms; in practice we scarcely meet with one apart from the other. It is possible, no doubt, for one person occupying a large room so to close up all the openings as to render the air impure. But there is never overcrowding without bad ventilation, because when many persons are huddled together in small rooms, the admission of fresh air exposes some of them to draughts, and is sure to lead to one aperture after another being shut up.

Many years ago the late Dr MacCormac, of Belfast, drew attention to the prevalence of phthisis in the close and crowded houses of the poor, and ascribed it to breathing air already contaminated, *i. e.* as we should now say, contaminated by the bacilli of tuberculous sputa.

The proof that confined air is a cause of phthisis rests mainly upon the evidence of statistics as to the frequency of the disease among soldiers, artisans, and inmates of prisons. As regards soldiers, a Royal Commission upon the Sanitary Condition of the Army, which reported in 1858, brought to light the fact that the death-rate from consumption in all branches of the service was in excess of that of the civil population of large towns, and that among the Foot Guards it was more than twice as great. Since neither the clothing of the soldier, nor his food, nor the nature of his occupation could be supposed to be the cause of this excess, the cause appeared to be his lodging. In barrack dormitories the cubic space actually given to each man was often only half or two thirds of 450 feet, the minimum allowed by regulation; and the air in these rooms became offensive before morning. The conclusion at which the Commission arrived has since been confirmed by the great fall in the consumptive death-rate, especially among the Foot Guards, which has followed the introduction of sanitary improvements.

In Dr Ransome's Milroy Lectures it is stated that in Canada, a country comparatively free from phthisis, the death-rate among English soldiers was 23 per 1000, compared with an average throughout England of less than 10 per 1000, and a death-rate in Manchester of only 12·4. After the barracks had been properly drained and ventilated, the mortality had sunk from 23 per 1000 in 1865 to 9·5 in 1872, and 6 in 1874.

Dr Farr long ago stated his belief that the prevalence of phthisis in the

armies of Europe is due in part to the inhalation of expectorated tubercular matter dried, broken up into dust, and floating in the air of close barracks.

As to workmen, we have evidence given by Dr Guy before the Buccleuch Commission of Inquiry into the State of Large Towns. He had elaborately investigated the relative liability to phthisis of different classes of the population of London. He found that the disease was more fatal to artisans than to tradesmen, and more fatal to tradesmen than to professional men and the upper classes. Even hawkers, standing about in the streets and exposed to all inclemencies of weather, had the advantage over men employed in workshops. Among printers he instituted a very close comparison as to the frequency of symptoms of lung disease, arranging the men in classes according to the amount of air-space in the rooms in which they worked. Of 104 men having less than 500 cubic feet of air to breathe, 13 had suffered from blood-spitting and 13 others from catarrh; of 115 men having from 500 to 600 cubic feet of air, 5 had suffered from blood-spitting, 4 from catarrh; of 101 men having more than 600 cubic feet of air, 4 had suffered from blood-spitting and 2 from catarrh.

With regard to prisoners, there is the contrast between two prisons in Vienna, cited by Parkes. In the Leopoldstadt prison, which was very badly ventilated, there died in the years 1834—1847, 378 prisoners out of 4280, or 86 per 1000; of whom no fewer than 220, or 51·4 per 1000, died from phthisis. In the well-ventilated House of Correction in the same city there were from 1850 to 1854, 3037 prisoners, of whom 43 died, or 14 per 1000; and of these 24, or 7·9 per 1000, died of phthisis. Diet and mode of life were, it is believed, the same in both prisons. The great prevalence of phthisis among prisoners was long ago pointed out by Dr Baly, as the result of an examination of the records of the Millbank Penitentiary; but he was unable to decide how far it was due to defective ventilation.

Conveyance by food.—The tuberculous contagion may be conveyed by means of milk from diseased cows, and may thus gain entrance directly to the intestine and indirectly to the lungs. The same property has been ascribed to imperfectly cooked meat from tuberculous cattle. A Royal Commission on this important subject began its inquiries in 1890, and published its report in 1895. The evidence was that the chief danger of tuberculosis in cattle is the affection of the udder in cows, which may contaminate the milk with tubercle bacilli. Raw meat may be contaminated with a knife which has cut through a tuberculous lymph-gland or udder; but well-cooked meat, like boiled milk, is safe. Beside the chance of the internal parts of a joint not being sufficiently heated, there is the possibility of butchers and cooks infecting their fingers by handling raw meat from tuberculous animals. This, however, appears to be very unlikely, for even when the lungs and serous membranes are tuberculous, the muscle and fat seem seldom, if ever, to be affected. (See Dr Sidney Martin's excellent article on Tuberculosis in Allbutt's 'System of Medicine,' vol. ii, pp. 29, 30.)

Hereditary predisposition.—Of indirect causes of phthisis, inheritance is the most important. It is a matter of universal experience that in some families deaths from phthisis occur, generation after generation, with terrible frequency. When a candidate for life insurance has lost a parent or more than one brother or sister from consumption, it is held that an addition to the premium is necessary to cover the increased risk; and if both parents have died of phthisis, or more than two other near relatives,

the "life" is generally regarded as almost uninsurable on reasonable terms.*

Among persons affected with phthisis, the proportion of cases in which the same disease can be traced in their relatives appears from certain inquiries by Dr Theodore Williams, recorded in the 'Med.-Chir. Trans.' for 1871, to be 48·4 per cent. The patients were seen in private practice, so that the results are probably as little inaccurate in the way of omission as can be expected in such inquiries, although it would be an advantage, for the purpose of comparison, if we knew to what extent a similar family history exists in the population generally. Probably the proportion is larger than 50 per cent., for cases of consumption are often concealed under such phrases as pneumonia, bronchitis, pleurisy, effects of childbirth, or result of change of life. Out of 484 cases in which phthisis was traced among the relatives of patients themselves phthisical, there were 120 in which the disease had existed in one or both of the parents, but 224 in which it affected only brothers or sisters.

Family predisposition to consumption probably means not direct inheritance, as of syphilis, or gout, or epilepsy, or hæmophilia, but inheritance of a tendency to bronchial catarrh, or to dyspepsia and malnutrition, which causes a "vulnerability" in certain stocks, an inability to resist the invasion of the tubercle-bacillus. There is no evidence of a specially strong tendency to phthisis in the children of parents actually consumptive, one or both of them, at the time of procreation. Moreover congenital tuberculosis is so rare that the authentic cases recorded in the human race are limited to about a dozen cases: so that we may say with Bollinger that "die congenitale Tuberculosis so gut wie auszuschliessen ist die." Phthisis is certainly not directly transmitted by sperm or germ, like syphilis; and the liability imparted does not seem to be one to disease or early death in general, but to tuberculosis in particular: possibly some defect in the power of the leucocytes to deal with the invading microbes, or in the chemical properties of the liquor sanguinis.

The experience of insurance offices as well as of private practice is that a phthisical tendency is more frequently transmitted by the mother than by the father.

Personal predisposition.—It is an old belief that persons of a particular bodily frame and physiognomy are particularly prone to tuberculous diseases; and this has been called a *diathesis*. Little value, however, can be attached to the statements of early writers on the subject, because "scrofula," as it was called, was often confounded with rickets or with congenital syphilis; and, according to Sir Thomas Watson, the numerous signs of the "scrofulous diathesis" varied widely with the "temperament" of the individual, whether "nervous," "sanguine," or "bilious."

It seemed a step in advance when Sir William Jenner, in 1860, proposed to distinguish two diathetic states—tuberculosis and scrofula. Now, so-called scrofula or struma, originally meaning any swelling of the neck (pig's neck), had in Jenner's day come to mean caseous lymph-glands. But these are themselves tuberculous, and so are other forms of struma, as caries of bones and joints, and "pulpy disease" or "white" swelling of the latter.

Jenner's description of tuberculosis seems to refer to a habit of body

* In Prof. Erb's words, "For the production of phthisis—let strict bacteriologists and contagionists say what they will—the most important factor is undoubtedly predisposition." —'Volkmann's klin. Vorträge,' No. 271 (1900).

which, while not incompatible with symmetrical growth and physical beauty, shows a want of power of resistance to disease in general, rather than to the invasion of tubercle in particular. Certainly multitudes die of phthisis who never exhibit the features of the tuberculous diathesis.*

The fact is that the words *scrofula* and *struma* have been applied in so loose and arbitrary a way, quite independently of chronic swelling of the cervical lymph-glands, or even of evidence of caseous disease in any part of the body, that they should be entirely discarded. Moreover, "tendencies" to tuberculosis which are never carried out can scarcely be the subject of useful discussion, nor can the exploded doctrine of temperaments be revived until we again accept the four Galenical humours and their eucrasia or dyscrasia.

Mr Francis Galton and the late Dr Mahomed recorded in the 'Guy's Hospital Reports' for 1881 the results of "An Inquiry into the Physiognomy of Phthisis by the Method of Composite Portraiture." Although they obtained from the photographs of 442 phthisical patients two types of face—the one of narrow ovoid shape, the other a broad face with coarse features—yet this was only by the careful selection of a few out of the whole number; and they found a larger proportion of narrow ovoid faces among patients who were not phthisical than among those who were.

The phthinoid chest.—There still remains the question whether a tendency to phthisis is indicated by any particular form of chest. Dr Gee recognises two shapes of chest in persons predisposed to consumption.

One of them is the "alar," or "pterygoid" chest of Galen and Aretæus. This is narrow and shallow, with drooping or increased obliquity of the ribs, sloping shoulders, and a long thorax; the "winged" appearance is caused by the projection of the scapulæ. The throat is often prominent, the neck long, and the head carried forwards.

The other is the "flat" chest; this is flattened in front, the costal cartilages losing their curve and becoming straight; or the sternum may be depressed below the level of the costal ends of the cartilages. Persons who are extremely flat-chested often have broad shoulders, so that, as one stands facing them, one might fancy them to be well developed. Traube laid stress on the significance of a flat chest as indicating a liability to consumption, and Wilks used strongly to insist on it.

But probably the "flat chest of phthisis," like the "rounded chest of bronchitis," is an acquired condition, the result of disease, not the indication of its future advent. Neither the alar nor the flat chest is seen in early phthisis with more frequency than other varieties of ill-shapen chest, which often result from neglect during childhood, and are consequently very common among the poorer classes.

* As leading features of *tuberculosis* Jenner gave the following:—"Nervous system highly developed; mind and body active; figure slim; adipose tissue small in quantity; organisation generally delicate; skin thin; complexion clear; superficial veins distinct; blush readily; eyes bright, pupils large; eyelashes long; hair silken; face oval, good-looking; ends of long bones small, shafts thin and rigid; limbs straight; also tendency to "inflammation of the mucous membranes of a peculiar kind; so-called strumous ophthalmia; inflammation of the tarsi; catarrhal inflammation of the nose, pharynx, bronchi, stomach, and intestines; inflammation and suppuration of the bronchial glands on trifling irritation; obstinate diseases of the skin; caries of bone."

He described *scrofulosis* as follows:—"Temperament plegmatic, mind and body lethargic; figure heavy; skin thick and opaque; complexion dull, pasty-looking; upper lip and alæ of nose thick; nostrils expanded; face plain; lymphatic glands perceptible to touch; abdomen full; ends of the long bones rather large; shafts thick."

Again, one cannot dissociate congenital from acquired deformities of the thorax in regard to their possible influence on the subsequent occurrence of phthisis. Freund, in 1859, maintained that what caused a small and contracted chest was often a premature ossification of the cartilage of the first rib; and Dr Hutchinson (vol. i, p. 1040) did not suppose that a defective vital capacity of the lungs indicated a tendency to phthisis, but rather that it was a sign of the actual presence of the disease at an early stage. Again, deformity of the chest from lateral curvature seems not to carry with it increased liability to consumption. It is very doubtful whether the habit of stooping at a desk, or the work of a tailor or shoemaker or weaver, specially favours the development of phthisis; nor is there evidence that the foolish practice of compressing the base of the chest by stays can produce such an effect.

As to the predisposing effect of other diseases of the chest in leading to tuberculosis, we may say that true pneumonia (acute fibrinous or lobar) is seldom or never followed by phthisis, that acute or chronic bronchitis, bronchiectasis, and emphysema do not predispose, and perhaps may, to some extent, protect from it; but that pleurisy and broncho-pneumonia, especially that of measles, are frequently followed by consumption.

Predisposition from general causes.—Insufficient food is often assumed to predispose to phthisis, but it would be difficult to adduce the proof; and the same doubt applies to the presence of anæmia. Certainly both emaciation and anæmia may go on to an extreme degree in general hospitals where cases of phthisis are always present, and yet no tubercle develops. If such deteriorating influences lead to phthisis at all, it is by weakening the power of resistance of the tissues.

Child-bearing has a remarkable relation to phthisis. It is a well-known fact that during gestation consumption is scarcely ever fatal; the patient survives until labour is over, when the symptoms soon become more urgent than ever, and death may follow rapidly. Cases associated with child-bearing generally run an acute course.

These considerations are so important in regard to life insurance, that it is only prudent to count all deaths of mothers "in childbed" or "after delivery" as due to phthisis, unless there is explicit evidence of death from flooding or from puerperal fever.

Alcoholic intemperance has a doubtful position in the ætiology of phthisis. Clinical experience shows that drunkards die in large numbers of this disease; but it is often difficult to exclude the operation of other causes. Some writers have even held that the use of alcohol is rather prophylactic against than productive of phthisis, and observations in the United States have been cited in favour of this view. Walshe and Wilks did not accept intemperance as a cause of consumption, while Williams and Fagge had little doubt of it. Lately Dr Isambard Owen, of St George's Hospital, found that, among the phthisical patients attending there, 50 per cent. were, on their own showing, excessive drinkers, while only 33·5 per cent. of the other patients confessed to habits of intemperance; or, put in another way, of 100 non-consumptive patients, 41·5 professed to be temperate; of 100 consumptives, only 23.

It must be remembered that drunkenness goes with poverty, exposure, and unhealthy living, which are acknowledged to be predisposing causes of bronchial and alveolar catarrh, and so of phthisis. Again, most of the publicans, barmen, and other intemperate classes who consult a "chest

doctor" will be subject to disease of the chest. Is phthisis less common in Italy than in Great Britain, in Constantinople than in Glasgow? Are total abstainers more exempt from consumption than temperate persons? And do intemperate persons recover from phthisis? These are questions not yet answered.

It has often been supposed that phthisis in drunkards is usually chronic and fibrous rather than caseous and acute. Huss of Stockholm put forth this opinion about 1850, and it has derived support from the fact that alcohol causes a growth of interstitial fibrous tissue in the liver, and probably in the kidneys. But it is certain that acute forms of phthisis, with tuberculous peritonitis, are often seen associated with hepatic cirrhosis; and "fibroid phthisis" is only the extreme result of what almost every case of phthisis shows in some degree, a power of repair by cicatrization, adhesions, and fibrous thickening.

Diabetes is a frequent cause of phthisis, which is usually of an acute type (vol. i, p. 446).

Syphilis is sometimes followed by the development of a destructive disease of the lung, which on *post-mortem* examination is found to be typically tuberculous. Dr Fagge recorded thirteen cases of this kind at Guy's Hospital between the years 1863 and 1873. But this is not beyond the chance coincidence of two diseases so common as lues and phthisis. Moreover, among syphilitic patients, especially in hospital practice, many are intemperate, destitute, and every way careless of their health. The striking action of antisiphilitic remedies in relieving certain cases of supposed phthisis is explained by recognising a syphilitic disease of the lungs capable of simulating phthisis, but pathologically distinct. This disease, as distinct from true or tuberculous phthisis occurring in a syphilitic subject, has been described in the chapter on chronic inflammations of the lung (*supra*, p. 31).

The following conditions affect the lungs directly, and act as predisposing causes of phthisis by producing precedent catarrh.

1. *Inhalation of dust.*—Ramazzini, in his work, 'De Morbis Artificum,' published in 1703, seems to have been the first to point out that certain classes of workmen are liable to have their lungs injuriously affected by dust given off by the materials in which they work. Of late years the question has been thoroughly studied, both in England and in Germany, by Peacock, Greenhow, and Arlidge, by Hirt, Albrecht, Bäumlér, and Zenker, who named the chronic disease due to this cause *Pneumoconiosis* (*κόκκισ*, dust). The nature of the mischief set up by breathing dust may be a mere catarrh of the trachea and bronchial tubes, leading to chronic cough and emphysema, and perhaps proving fatal by dilatation of the right side of the heart and dropsy. But in many instances there is consolidation of the lung, which may lead to the formation of cavities, and spread from apex to base, as in other cases of phthisis; so that it has been called "miner's," "weaver's," and "knifegrinder's consumption."

As a rule cases of pneumoconiosis advance slowly to a fatal termination in the fibrous form of phthisis. But cases of caseous softening are far from rare, and characteristic tubercles the size of a millet-seed or a pea, opaque and yellow, occur in the lungs of miners, potters, or stone-masons. Anatomically the chronic broncho-pneumonia of grinders and potters cannot be separated from chronic phthisis; and the presence of Koch's bacilli has conclusively confirmed the true nature of pneumoconiosis.

The materials which give off dust that may lead to phthisis are of various kinds. They may be classified as follows.

Particles of carbon.—As far back as 1813 Pearson, in the 'Philosophical Transactions,' threw out the suggestion that the black discoloration of the lungs and of the bronchial glands, which is found in most adults, but not in children, consists of particles of carbon "introduced with the air in breathing," and originally "derived from the combustion of coal, wood, and other inflammable materials." A similar opinion was expressed by Laennec. In 1831 Dr Gregory recorded the case of a labourer in the coal mines of Dalkeith; both lungs were throughout of a uniform coal-black colour, and yielded, when washed, a dark matter, which was found by Dr Christison to resist the action of concentrated nitric acid and of chlorine, and to yield by distillation products just like those which result from the distillation of coal. The conclusion seemed irresistible that the organs had been discoloured by the penetration of coal-dust from without. "Spurious melanosis," therefore, and *Anthraxis*, proposed by Stratton in 1837, were the terms applied to this condition of the lungs. Hasse in 1841, and Virchow in 1847, gave reasons for doubting this explanation, and ascribed the pigmentation to a pathological deposit of granules derived from the blood.* In 1860, however, the following case was recorded by Traube.

The patient was a man who for about twelve years had been engaged in loading and unloading wood-charcoal. He had long been accustomed to expectorate a black substance, and when he died his lungs were found to be almost everywhere of a black colour, yielding to pressure a black frothy fluid which stained the fingers like thin Indian ink. Both in the sputa, and in the pulmonary tissue, there were found minute foreign bodies of irregular form, with pointed processes. That these were fragments of wood-charcoal was evident, not only from a comparison with particles of the charcoal brought from the place where the man had worked, but also from the fact that some of them showed the circular discs characteristic of the woody fibres of Coniferae.

A second similar case came before Traube a few years later, and in 1866 Virchow recanted his former views, and Rindfleisch admitted that "anthracosis" denotes the true nature of the disease.

In describing a specimen of "miner's lung" which had been sent to him from Scotland ('Edinburgh Medical Journal' for 1858), Virchow stated that scarcely any of the black matter was found in the interior of the alveoli; and in their walls it lay not beneath the endothelium, but between the elastic fibres and the connective tissue. It was still more abundant in the interlobular and peribronchial fibrous tracts, and beneath the pulmonary pleura. This account has been often confirmed since. The particles of carbon may be present in the costal and diaphragmatic pleura, as well as in the bronchial glands; and at Guy's Hospital we once found some of it free in the upper and back part of the pericardial space, close to an intensely black gland that lay just outside.*

* Virchow's difficulty in believing that these granules were brought in from the air is explained by our present knowledge that leucocytes are capable of taking particles of foreign materials into their substance, and that animal membranes are permeable. The distribution of inhaled particles of dust was studied experimentally by von Jus, in the 'Arch. f. exp. Pathologie' for 1876. He injected cinnabar into the air-passages of dogs, and found that the particles were rapidly taken up by altered leucocytes (phagocytes), so that five days later scarcely any pigment was left in the pulmonary alveoli; within six hours some of it reached the bronchial glands, being first deposited in their cortical layer, but ultimately reaching their medulla: much, however, remained in the lungs, being accumulated in the connective tissue, between the lobules, round the vessels and the tubes, and beneath the pleura. In other words, von Jus found that its distribution corresponded precisely with what had been described by Virchow in the case of the miner's lung.

The phthisis of anthracosis is marked by a special symptom, the "black spit," which often continues a long time after a miner has ceased to follow his trade. Dr Greenhow showed to the Pathological Society in 1869 the lungs of a collier who, about ten days before his death, suddenly spat up matter closely resembling black paint, and continued to expectorate four or five ounces daily until he died; in the right lung there was a large irregular cavity, containing a quantity of black pulpy residue.*

Sputa, however, may be black without there being any lesion of the pulmonary tissue but anthracosis; hæmoptysis or the detection of elastic tissue or of bacilli in the sputum is needful to prove that phthisis is present.

Oxide of iron.—In 1864 Friedreich asked why, if the black lungs and bronchial glands of coal-miners are due to inhaled carbon, the workers in red sandstone quarries should not have their lungs reddened by the dust. Now, Zenker had at that time in his possession the lungs of a woman who for seven years before her death, was making the little paper books in which gold-leaf is laid. The paper has to be coloured red with peroxide of iron, rubbed in with a piece of felt. The occupation is a very dusty one; and the woman's lungs were found after death to be throughout of a bright brick-red colour, so that their cut surface looked just as if it had been daubed over with red paint. The microscope showed that granules of oxide of iron were present, beneath the pleura, in the interlobular fibroid septa, in the peribronchial sheaths, in the walls of the alveoli, and even in leucocytes occupying their interior. In the twentieth volume of the 'Pathological Transactions' there is a coloured drawing, taken from a specimen in the possession of Dr Wilson Fox; the tint is much browner than in Zenker's drawings published in the 'Deutsches Archiv' two years before. Zenker proposed to name the affection Siderosis (*σίδηρος* = iron). In 1874, Merkel, in 'Ziemssen's Handbuch,' was able to refer to seven other cases, one of which followed the use of red oxide of iron for polishing glass. He had also met with two instances in which the lungs were blackened by the black oxide of iron, and one in which ferric phosphate was present; there was no difficulty in detecting the iron in the sputa by hydrochloric acid and ferrocyanide of potassium.

Silica and alumina.—It has long been known that workmen whose occupations expose them to siliceous or argillaceous dust are prone to die of phthisis. Much information with regard to the excessive mortality from this cause in certain districts of England is contained in a paper by Greenhow in Sir John Simon's third 'Report to the Privy Council,' published in 1861. Merkel proposed to term the resulting disease of the lungs Chalicosis (*χάλιξ* = gravel). The presence of silica in the pulmonary tissue seems to have been first detected by Peacock and by Greenhow. Kussmaul showed (in vol. ii of the 'Deutsches Archiv') that this substance is present in greater or less quantity in the lungs of all persons (though not in those of a foetus), having doubtless been derived from the dust of the streets and roads blown up by the wind. In a railway signalman, stationed in a very sandy district, Meinel found that silica actually formed as much as 18·2 per cent. of the ash of the lungs after incineration; even in a stonecutter who died of phthisis, and whose lungs were analysed by Kussmaul, the amount was not greater than 24·7 per cent. of the ash. Under the microscope the particles of silica may be seen as bright bodies of round or angular shape.

* This specimen forms one of the excellent series illustrating pneumoconiosis in the Museum of the Middlesex Hospital.

Stonemasons, lithographers, and millstone makers suffer from this cause. Still more fatal is the grinding and polishing of steel instruments—from scythes to needles—such as is carried on in Sheffield and Birmingham. Whether the exact nature of the work be needle-pointing or fork-grinding, or the sharpening of fish-hooks, the result differs but little; a large number of the men die prematurely, some between twenty and thirty, and more between thirty and forty: very few survive the age of forty without suffering more or less from pulmonary symptoms. This has been long known at Sheffield as “grinder’s rot.” What is termed “dry-grinding” is much more injurious than “wet-grinding;” for in wet-grinding the wheel, as it revolves, dips into water and deposits a large part of the dust. But even wet-grinders are exposed to dust in “hacking” their grindstones, which generally has to be done every day.

Potters, again, are exceedingly apt to be attacked with phthisis; “flat-pressers” suffer more than “hollow-pressers;” but the worst off of all are “china-scourers,” whose business is to rub off the loose flint powder from the china with sand-paper, after it has been baked. Another dangerous occupation is pearl-shell cutting.

By the use of respirators, and by other preventive means, the mortality in these trades has of late years been greatly reduced.

Similar injury may result from the inhalation of vegetable matters in the carding of cotton, and the hackling of flax. In two men who had worked in a tobacco factory, Zenker found “brown spots in the lungs and in the bronchial glands, evidently due to the deposition of powdered tobacco.”

According to Hirt’s tables of mortality from phthisis in various trades in Germany (quoted by Dr Ransome) the highest death-rate is among flint-workers and filecutters, then come grinders and stonecutters, next brush-makers, and then cigar-makers and glasscutters.

Pathology of pneumoconiosis.—It is clear that foreign particles of various kinds may find their way into the lungs, may be deposited in the pulmonary tissue, and may either remain there or be ultimately transported to the bronchial glands. In many cases they remain innocuous, but when insoluble and gritty, they cause a bronchial catarrh which invites infection by the tubercle bacilli.

Whatever may be the nature of the irritant, the resulting affection of the lungs shows the same characters. Greenhow in 1865-70 exhibited to the Pathological Society the lungs of a collier, a copper-miner, a razor-grinder, a stone-worker, a potter, a flax-dresser, and a pearl-shell cutter; and the identity of the anatomical affection pointed to a common pathological process. That this process is tuberculous is shown by its beginning in the upper lobe, and by the constant presence of the bacillus of phthisis.

2. *Cold and wet.*—A firmly rooted belief is that consumption is often the result of accidental causes, such as getting chilled by remaining in wet clothes, exposure to a draught when heated in dancing, or sleeping in damp sheets, and generally—a neglected cold.

That catarrh is apt to lead to phthisis is shown by the frequency with which, in children, pulmonary tuberculosis follows whooping-cough or measles. But the same liability is not shown by persons subject to chronic bronchitis. The specific seed of phthisis finds its entrance in the soil of the bronchioles and alveoli, and hence is apt to follow the broncho-pneumonia or

pulmonary catarrh set up by the irritation of dust and other foreign bodies, or the lobular changes which follow measles.

In more than one instance Dr Fagge found consumption distinctly traceable to moving into a damp house, the patient having been quite well before.

It is probable that some of the cases of this kind, however, as of those to be next mentioned, are due not to damp and cold houses or soil, but to direct contagion from tubercle bacilli persisting as saprophytes in the soil, or in the corners of ill-kept houses.

3. *Damp soil*.—Increased liability to attacks of pulmonary catarrh seems to account for the influence of damp and ill-drained localities on the frequency of phthisis. In 1862 Dr H. I. Bowditch, of Boston, in addressing the Massachusetts Medical Society, brought forward evidence which led him to believe that, in that State, consumption prevails most on a damp soil, and seldom occurs where the soil is dry. This evidence consisted chiefly of the replies of medical men living in 183 townships to inquiries as to the frequency of phthisis, and as to the moisture or dryness of the localities.

Still more important is a body of facts collected in England by Sir George Buchanan during the years 1865 and 1867, and published in Mr Simon's ninth and tenth reports to the Privy Council. The inquiry began in a tour of inspection made for the purpose of ascertaining the results of sanitary works that had been carried out in twenty-five towns, containing an aggregate population of 606,186. It was found that there had been a great diminution in the total death-rate, and that the prevalence of enteric fever had become much less, especially where a good water-supply had taken the place of a bad one, and where efficient drainage had displaced cesspools or middens. The number of cases of phthisis had also markedly diminished; and this coincided with drying the ground by drainage of the subsoil.

The following table shows the amount of change in the death-rate from phthisis in twenty-four of the towns visited. Two examples are given in detail as follows.

In 1851 Mr Rammell had reported of Salisbury as follows:—"Numerous streams of water, supplied by the Avon, run through most of the streets. . . . The soil is of a porous gravel, containing everywhere a great deal of water, which rises to within a short distance of the surface. There have been several instances of the cathedral being flooded by the water of the subsoil. The foundations of the houses are almost without exception damp." The water supply is from wells "dug about eight or ten feet deep, the water rising to within three or four feet of the surface." Mr Middleton had drawn public attention to the same point in his address to the British Association in 1864. Apart from the bad system of drainage which the canals of Salisbury were made to serve, he clearly showed that they were also injurious by keeping the subsoil constantly damp.† In 1853 efficient drainage works were begun in Salisbury, and they were completed in 1855. In 1865 Dr Buchanan writes as follows:—"The subsoil is now dry, and cellars of considerable depth can now be made in different parts of the town, which do not become flooded at any time. On an average the subsoil water has been lowered four or five feet all over the city. The cathedral has never been flooded since the drainage works. As is shown in the table, the annual death-rate from phthisis fell in Salisbury from 44½ per 10,000 in 1844-52, to 22½ per 10,000 in 1857-64." ("Benefits of Sanitary Reform at Salisbury," 1865.)

Of another town, Banbury, Mr Rammell had reported in 1850 in the following terms:—"The drains are not at all at a sufficient depth to drain the cellars of the houses. In the principal streets of the town water is raised from the cellars into the drain by buckets, and creates a nuisance." Sanitary operations were begun there in 1854. "At present," says Dr Buchanan, writing in 1865, "the sewers and drains all act efficiently. . . . Many of the wells of the town have been dried by the sewers." As appears from the table, the phthisis death-rate for 10,000 has declined from 26½ in 1845-53 to 15½ in 1857-64.

Town.	Previous death-rate per 10,000 from phthisis.	Degree of change in death-rate from phthisis.		Influence of sewage works on subsoil.
		In total population	In females between 15 and 55.	
Salisbury . . .	44 $\frac{1}{8}$	-49 p. c.	?	Much drying.
Ely	32	-47 p. c.	?	Much drying.
Rugby	28 $\frac{3}{4}$	-43 p. c.	-48 p. c.	Some drying.
Banbury	26 $\frac{2}{3}$	-41 p. c.	-36 p. c.	Much drying.
Worthing	30 $\frac{1}{2}$	-36 p. c.	-41 p. c.	Some drying.
Macclesfield . .	51 $\frac{1}{2}$	-31 p. c.	-22 p. c.	Much drying.
Leicester	43 $\frac{1}{3}$	-32 p. c.	-16 p. c.	Drying.
Newport	37	-32 p. c.	-13 p. c.	Local drying.
Cheltenham . . .	28 $\frac{3}{4}$	-26 p. c.	-25 p. c.	Some drying.
Bristol	33 $\frac{1}{2}$	-22 p. c.	-18 p. c.	Some drying.
Dover	26 $\frac{1}{3}$	-20 p. c.	-18 p. c.	Local drying.
Warwick	40	-19 p. c.	-10 p. c.	Some drying.
Cardiff	34 $\frac{3}{4}$	-17 p. c.	?	Much drying.
Merthyr	38 $\frac{1}{2}$	-11 p. c.	-12 p. c.	Some recent drying.
Stratford	26 $\frac{2}{3}$	-1 p. c.	-4 p. c.	Some local drying.
Penzance	30 $\frac{1}{4}$	-5 p. c.	0	No change.
Brynmaur	28 $\frac{1}{3}$	+6 p. c.	-8 p. c.	No notable change.
Morpeth	30 $\frac{1}{2}$	-8 p. c.	+12 p. c.	No change.
Chelmsford . . .	32 $\frac{1}{4}$	0	+11 p. c.	Slight drying.
Penrith	39 $\frac{1}{3}$	-5 p. c.	+27 p. c.	No change.
Ashby	25 $\frac{1}{2}$	+19 p. c.	-10 p. c.	Some drying.
Carlisle	32	+10 p. c.	+11 p. c.	Drying (with local defects).
Alnwick	28 $\frac{1}{2}$	+20 p. c.	+36 p. c.	No drying.

In 1867 Buchanan was directed by the Privy Council to make a special investigation in Surrey, Kent, and Sussex, to see whether any relation could be traced between the prevalence of consumption in these counties and the moisture of the soil.

He pointed out that where the soil is pervious, its being moist or dry must depend entirely upon whether the water which reaches and sinks into it can escape from beneath. It is no advantage for a place to be situated on gravel if the subsoil water cannot get away.

As a rule, one may say that a permeable district will be dry if it lies high in relation to the places round it, damp if it lies low. On the other hand, among impervious soils, the question of dryness or moisture is almost entirely one of the inclination of the surface. Even among clays there is a great difference in dampness, according to the flat or sloping character of the ground.

Buchanan further made a comparison between retentive and pervious soils with regard to the prevalence of phthisis in a limited area, which in part is formed by the Weald clay, in part by the Hastings beds of mixed sands and clays.

In the following table, the districts are arranged in order of the death-rate from phthisis, those being placed highest in which it is least. Where there are gravels over the Weald clay the figure is divided between the last two columns.

District (in order of phthisis death-rate).	Percentage of population resident on						Total on	
	Higher beds, mostly Lower Greensand.		Weald clays.		Hastings beds.		Sands + half gravel over Weald clay.	Clays + half gravel over Weald clay.
	Sands.	Clays.	With gravel.	Without gravel.	Sands.	Clays.		
Hastings	95	5	95	5
Cranbrook	1	6	84	9	84	16
{ East Grinstead	12	82	6	82	18
{ Tunbridge	1	24	7	64	4	76	24
{ Hambleton	49	...	20	31	59	41
{ Battle	80	20	80	20
{ Rye	4	79	17	79	21
{ Maidstone	43	1	45	11	66	24
{ Cuckfield	21	1	...	25	48	5	69	31
{ Uckfield	1	82	17	82	18
{ Hailsham	34	61	4	61	38
{ Ticehurst	67	33	67	33
Tenterden	29	42	29	42	58
Horsham	56	44	...	44	56
Petworth	30	70	30	70

Still more striking, perhaps, are certain comparisons between particular sets of districts which differ—if pervious, in being *high-lying* or *low-lying* respectively; if impervious, in being *sloping* or *flat*.

(1) As between high-lying and low-lying *pervious* soils, a contrast is afforded by the chalk districts. No soil is drier than chalk at a good elevation; there are no streams, for water cannot be reached by ordinary wells. In many districts, however, the bulk of the population who live on chalk occupy valleys with the water-line in the chalk not far below their houses; and in the south of Sussex a large part of the area reckoned as chalk is a damp plain on the sea level. The death-rate for phthisis at North Aylesford and Dover (both of which lie high) was 289 and 296 respectively; while those for Worthing, Lewes, and Westbourne (all of which lie low) were 419, 426, and 498.

(2) As between sloping and flat *impervious* soils, a striking contrast is presented by the London clay and the Weald clay. The former is disposed in long slopes or hills; the latter undulating, or level ground. The former is covered over large areas by gravel to many feet in thickness; the latter has only very level gravels, and these are seldom thick. The former generally has the direction of drainage away from it; the latter is bounded to the north and south by higher grounds, so that other beds drain into it. Accordingly the London clay is commonly much less wet than the Weald clay. The difference between the two formations in respect of their phthisis death-rate, Buchanan found unmistakable. All districts with even a third of their population on Weald clay had a high mortality from phthisis, whereas a district might include much London clay with no such death-rate.

It is right to mention that Dr Kelly, the Medical Officer of Health for East Sussex, has since expressed doubts of there being any intimate relation between dampness of the soil and phthisis. He finds that in the years 1861–70 the order in which the several districts have to be placed in regard to their death-rates from phthisis is different from that given by Buchanan; he remarks that consumption seems most common in places which are bleak and exposed as well as damp; and he insists on the fact that in West Sussex (as indeed throughout England and Wales) there has of late years been a great decrease in the mortality from consumption, although there has been no change in the drainage. Dr Kelly

is inclined to attribute the decreased death-rate of phthisis mainly to the progress which has taken place in the condition of the rural population.

Hirsch also doubts the direct effect of good draining, and quotes the experience of Berlin, Dantzic, and Brunswick, of improved drainage being followed by increased death-rate from phthisis.*

Apart from dampness and cold as causes of pulmonary catarrh, and so indirectly contributing to the prevalence of phthisis, it has been supposed that certain *winds* favour it. Dr William Gordon (in a paper read before the Royal Med. and Chir. Society, Oct., 1900) brought forward statistics on this point from Devonshire; but the explanation, if the facts are confirmed, would probably lie in Dr Cayley's suggestion, that in strong south-west winds people shut their houses, and that close dwellings favour infection.

Experience is almost unanimous as to the importance of placing consumptive patients in places protected from winds blowing from any quarter; but windy districts are of changeable temperature.

The analogy of tuberculous affections of the kidney and the joints, would lead one to admit that direct injury to the chest might set up phthisis. But analogy is an uncertain guide, and considering how common are injuries to the chest, and how common a disease is phthisis, instances of a connection between them ought to be more frequent if it existed.

It may seem unnecessary to discuss predisposing causes at such length when we have the efficient cause of phthisis in the tubercle bacillus. But though enterica and cholera are also proved to be contagious, their prevalence depends as much on favouring conditions as on the specific microbe. In the case of tuberculosis the soil is so important, and the seed, though essential, so all-pervading, that the conditions we have been discussing are scarcely less important now than before Koch made his great discovery.

Opposing pathological conditions.—Among the ætiological relations of phthisis, we must not forget certain conditions which have been, or still are, supposed to be antagonistic to its development.

That one of these is *malaria* is not generally accepted, although Walshe brought forward some evidence in point, and no doubt phthisis is rare in malarial regions (p. 83).

We seldom discover together, either during life or in the deadhouse, *carcinoma* and tuberculosis. This is probably due to the facts that each of these diseases is usually fatal, and that one of them chiefly attacks older, the other younger persons. Nine or ten cases occurred at Guy's Hospital during twenty years, in which the lesions of phthisis were found after death in patients who died of cancer of the stomach, uterus, œsophagus, or some other organ. One was a woman aged twenty-two, another a man aged twenty-four, a third a man aged thirty; the rest were older, and one had reached sixty-seven.

Antagonism between phthisis and *gout* has also been generally accepted, and probably not without reason. Both diseases are common in men between thirty-five and forty-five, yet cases of their concurrence in the same patient are rare. That gout and active phthisis may coexist is proved by three cases observed during life and recorded in the 'Guy's Hospital Reports' for 1873 (vol. xix, p. 338), while in a fourth patient with gouty

* See on this subject, and the ætiology of phthisis generally, the Milroy Lectures before the College of Physicians by Dr Ransome, in March, 1890, and his 'Essay on Tuberculosis,' 1898.

deposits in the joints, signs of old phthisis and some recent clusters of tubercles were discovered in both lungs.

It appears that at least one kind of valvular *disease of the heart* is a hindrance to the development of phthisis. Mitral stenosis is exceedingly common in young persons, and it often does not affect the general health for several years. That this lesion should be very rarely found in those who die of consumption is therefore remarkable. Traube could not remember to have met with an instance, although he had seen several cases in which those with regurgitant disease of the aortic valves developed phthisis. In our records of autopsies at Guy's Hospital, Dr Fagge could only find four examples in twenty years.*

Contrary to Rokitansky's opinion, it is now well known that those who have congenital narrowing of the right sigmoid orifice are very apt to die of tuberculous disease of the lungs. Traube saw two examples of this; and two occurred in Dr Fagge's practice at Guy's Hospital.

Rokitansky also believed that pulmonary emphysema and dilatation of the bronchial tubes, if carried far enough to cause venosity of the blood and cyanosis, afford protection from pulmonary tuberculosis.

Apart, however, from the crucial instance of congenital disease of the heart, there are striking exceptions to Rokitansky's rule. In 1864, for example, a girl of seventeen was admitted into Guy's Hospital with extreme dyspnœa and dropsy, and with clubbing of the fingers and toes. The bronchial tubes were found widened out into great sinuous passages, so that the cut surfaces of the lungs showed hollow spaces as extensive as the remains of the pulmonary tissue. Yet there were scattered yellow tubercles, especially in the left lung, spreading from the apex downwards. In 1874 a woman, aged thirty, died, who had long been more or less subject to cough, which for nine months before had become continuous. There was extreme emphysema of the bases and anterior parts of the lungs, and the tubes contained a large quantity of pus; but both lungs also showed scattered grey tubercles and patches of translucent grey consolidation, with points of caseation breaking down here and there into cavities.

It is, however, generally believed that pulmonary *emphysema* is unfavourable to the development of tubercle, and there is reason to believe that this is the fact. Even when emphysema supervenes upon primary phthisis the latter disease seems to be checked in the part so affected.

The supposed immunity of humpbacks from phthisis is not borne out by observation. Angular curvature usually depends on tuberculous caries of the vertebræ, and is frequently followed by tuberculous disease of the lungs (see a paper by Dr Fagge in the 'Guy's Reports' for 1874).

Age and sex.—One of the aphorisms of Hippocrates is that phthisis is most frequent between the ages of eighteen and thirty-five (v. 9 and viii, 7) but that it is a disease confined to young adults is not stated by the father of medicine, and is far from true. Phthisis may occur at all periods of life. At Guy's Hospital Dr Fagge found little if any diminution in the number of fatal cases for each quinquennial period up to the age of forty-five; several occurred in persons between sixty and seventy, and two at the age of seventy-two. At Copenhagen the proportion of deaths from phthisis to those from other causes increases with advancing age, and the

* One of Dr Moxon's beautiful drawings in our pathological theatre at Guy's Hospital shows how ill tuberculous inflammation of the lungs thrives in cases of mitral disease.

same statistical result is reported from Würzburg and other parts of Germany. Statistics of Dr C. J. B. Williams's patients appear to show that, other things being equal, the duration of phthisis increases with age, and it is a matter of general observation that the disease in children runs an acute course, while senile phthisis is usually chronic. This would bring the average age at which it begins earlier than the statement of ages at death indicates. Moreover it is probable that some of the aged patients succumbed to a recrudescence of a disease they had suffered from between eighteen and thirty-five, as in the case quoted above (p. 62).

The period at which appeared the first decided symptoms of what afterwards developed into phthisis was found by Dr Williams to be distributed as follows among 1000 private patients:—195 cases at an age below twenty (of these only 13 under ten), 667 between twenty and forty (and of these 418 below thirty), 94 between forty and fifty, 30 between fifty and sixty, and in 14 cases symptoms only began at an age above sixty.

More men than women die of consumption in the hospitals of London, although the Registrar-General puts the rates of males to females in the population generally as 3·77 to 4·13. Of Dr Williams's 1000 cases 625 were males and 375 females. Among the out-patients at the Brompton Hospital Dr Pollock found 60·7 per cent. male, and 39·3 per cent. female.

Sir Hugh Beevor has drawn attention to the fact that the mortality from phthisis in England and Wales is equal for the two sexes for the first five years of life; and then becomes greater in boys. After twenty the death-rate is higher in women, but from the climacteric period onwards fewer women than men die of phthisis ('Medical Magazine,' 1900, p. 333).

Incidence on sex or age probably depends on the more frequent operation, at different periods of life and in one sex rather than the other, of various predisposing causes, particularly of exposure and pulmonary catarrh.

In adult women consumption shows itself, as a rule, at an earlier age than in men, and more frequently runs a rapid course.

Dr Ransome has shown that in large towns many more men than women die from consumption, while in country places more women die than men ('Parkes Weber Essay,' Table iii, p. 9), and he believes that this is due to men working in crowded factories, shops, and offices, while women living in the country stay indoors much more than the men.

Distribution.—Phthisis is pandemic, but on the whole, more prevalent in temperate regions than in those which are very hot or very cold. It is endemic throughout Europe, and is most abundant in large cities, particularly in Paris and Vienna; but also in New York and Philadelphia, in Alexandria, and in Melbourne. It is probably as common in the United States, in China, and in Australia as in England; but is less so in the East Indies and within the tropics generally, although imported cases from Europe are unfavourably influenced by a hot climate. It is said to be very common at Lima, only 12° south of the line. It is extremely rare in Iceland, in the highlands of the Andes, in New Zealand, and in some oceanic islands, as well as in Syria, Arabia, and Persia, and in Upper Egypt. It is rare in swampy and malarious regions, as was first pointed out in 1812 by Dr Wells (the author of the essay on 'Dew'), and since confirmed by Boudin and many other observers. No quarter of the globe, and no race of men is known to be completely exempt from consumption.*

* See Dr Ransome's second lecture ('Brit. Med. Journ.,' March 8th, 1890).

Phthisis is well known among the lower animals, particularly the Quadrumania and the Ruminants. In monkeys the frequency of tubercle in the spleen, liver, and lymph-glands, and the acute course in the lungs, resemble phthisis in children; but tuberculous meningitis is rare. Of all mammals rabbits and guinea-pigs are most susceptible. Pulmonary tuberculosis is common among birds, chiefly in the caseous form, with abundance of Koch's bacillus. It is very rare in goats, also in dogs, cats, and other carnivora, and is not nearly so common in horses as in cattle (see Mr J. B. Sutton's remarks, 'Path. Trans.,' vol. xxxvi, p. 546, and Dr Creighton's monograph on 'Bovine Tuberculosis in Man;' also Dr Campbell's paper in the 'Guy's Reports,' xlviii, pp. 20—26).

Prophylaxis.—It is doubtful how far it is possible to guard directly against the invasion of bacilli. Suspected milk may be boiled or Pasteurised, tuberculous cows slaughtered, crowded rooms may be ventilated, and the sputa of phthisical patients may be disinfected, but at present there is want of proof that "consumption hospitals" are dangerous, or that the health resorts for *poitrinaires*, like San Remo and Davos, are more unhealthy than other places. It seems probable that, though infective, phthisis is not easily caught, except under special circumstances—as is the case with two other infectious diseases, syphilis and enteric fever.

Since it is now established that phthisis is always tuberculous, *i. e.* is due to infection by the specific bacillus (vol. i, p. 368), and since the usual channel of infection to the lungs is by the inspired air, we ask how the infection may be prevented. The access of the bacilli to the skin, the tonsils, the intestinal and genito-urinary mucous membranes may be gained by food, by milk, by direct contact, or in other ways (cf. *supra*, pp. 65, 66); but the lungs are invaded—occasionally, perhaps by mucus ejected from a phthisical patient in coughing, but as a rule—by dried-up sputa which are carried about in the dust of chambers.

The first object, therefore, is to keep phthisical sputa moist, the second to render them innocuous by carbolic acid (phenol) or by heat,* and the third to persuade patients not to expectorate except in a spittoon containing an antiseptic, or into paper or rags which can be burnt. In a free country it is impossible to prevent the breach of rule, except by the pressure of public opinion, and we must hope that not only consumptives but others will gradually learn to give up the practice of spitting in the streets.

On the other hand, there is no reason to believe that the tuberculous bacilli are conveyed by the breath, or that phthisis is directly contracted in this way, except in the most exceptional conditions.

The possibility of dried sputa being conveyed by body-linen or bed-clothes, renders it desirable to disinfect them by heat after removal. It does not seem established that the contagion of phthisis clings to houses like that of erysipelas or plague, though some cases bear out the possibility; but it is most desirable that dust should not be allowed to accumulate, and that it should never be disturbed by sweeping, but removed by wet cloths or by washing down.

Indirect but most important prophylaxis against phthisis is afforded by

* The writer many years ago used solution of corrosive sublimate to receive the sputa of phthisical patients in his wards; but it coagulates the surface of a nummular mass, and leaves the interior unaffected. A 5 per cent. solution of phenol is much better; but when no antiseptic is at hand if the spit cup always contains water and is always emptied down a drain the danger is very small.

early surgical treatment of tuberculous disease of joints, bones, and lymph-glands.

The hereditary transmission of consumption is not altogether beyond the scope of prophylaxis. The physician's advice, and the enlightened general opinion which he in time can produce, may do much to prevent the inter-marriage of cousins who both belong to a phthisical family, and to dissuade from marriage those in whom the disease has already appeared. If a mother is known to be phthisical, it is undesirable for her to suckle her children.

Those who come of a consumptive stock should live on a dry and elevated soil. The rooms in which they live or sleep or work should be airy and well exposed to sunlight. Their food should be nutritious and fattening. They should spend most of their time in the open air, and avoid sedentary occupations; and they should be accustomed to exposure to the weather. Cold bathing is advisable, provided that there is always a good reaction after a bath.

Special care should be taken during convalescence from whooping-cough and measles; and the recurrence of attacks of bronchial catarrh should be sedulously guarded against. The habit of breathing with the mouth shut is an effectual preservative from most of the chances of catarrh, and possibly may make bacillary infection more difficult. As the adult stage of life begins, moderation in all things should be impressed on every one who would avoid the risk of phthisis. Temptations to intemperance and to dissipation must be resisted; but often the tendency of those who have a phthisical predisposition is rather to asceticism than self-indulgence, and physically this may be scarcely less harmful.

The son of a phthisical miner, or potter, or weaver should avoid such kinds of work, and prefer country life to that of towns.

The steady diminution of the mortality from phthisis which has been observed in England (from 2567 per million in 1858, to 1541 per million in 1888, and 1307 in 1899), and also in New England (from nearly 4 pro mille in 1857 to about 3 pro mille in 1883), depends probably upon better drainage and better lodging; and, if so, is a great encouragement to prophylaxis in these directions. In an interesting series of maps presented to the International Congress at Berlin in 1899, the mortality in the British Isles takes, with Italy and Norway, the lowest rank—Ireland having the largest, Scotland the next, and England the smallest figure; while in the comparison between the great cities of Europe the highest death-rate from phthisis is in Moscow, St Petersburg, Vienna, and Paris; then in Berlin, and last in London.

The same favourable result is more fully set out in the returns of the Registrar-General for England and Wales between 1838 (when the statistics are first available) and 1894, compiled by Dr Ransome, and verified by Dr Tatham. The total death-rate from phthisis in each myriad (10,000) of the population has fallen in these fifty-seven years from 37 or 38 to 14 or 15, and the diminution of deaths from this cause has been steady and almost uniform, the only important check being from 1850, when it had fallen from 38 to 26, to 1853, when it had risen again to 29. It is remarkable that the returns of death from other tuberculous diseases have not appreciably diminished—from caries and chronic synovitis, lymphadenitis, tabes mesenterica, and meningitis, which used to be returned under the head of scrofula. The mortality from these forms of tuberculosis is chiefly con-

fined to children. Drs Ransome and Tatham give the death-rate from phthisis in London in eleven years (from 1886, when the population was a little more than four millions, to 1896, when it reached nearly four and a half millions) as oscillating between 2 and 1·8 or 1·9 for each myriad between 1886 and 1893, and falling below 1·8 to 1·7 between 1893 and 1896.*

Treatment.—We have at present no means of directly attacking the bacilli when they have gained lodgment in the human body, nor of specifically strengthening the organism—*i. e.* its phagocytes—against them. It is not absurd to hope that some day a chemical remedy may be discovered, like those for syphilis or for ague; but more probably we may look for the discovery of some antitoxin like that which is useful against diphtheria. The Carnivora and horses are almost immune against tuberculous infection, and their serum has been tried as a prophylactic, but without success. Koch's supposed cure for tubercle, the so-called Tuberculin, was of uncertain composition, and after being fully tried was found to be useless, injurious, or dangerous. A new edition of the remedy has since been put forth ("Tuberculin R." 1897), but the results so far have not been encouraging, and those who remember the lamentable events which followed the International Congress of Medicine in 1890 may be forgiven if they look with distrust upon specific remedies from the same source.†

At present the aim of rational treatment is to bring the phthisical patient into a physiologically sound and vigorous state, and to guard against relapses. When treatment is begun early, begun when phthisis is rather a suspicion or a fear than a diagnosis, and even in some more advanced cases, it is wonderful how frequently partial success at least is attained; and in not a few cases we are privileged to see our treatment result in as complete and permanent a cure as can be desired.

The most important step is, if possible, to remove patients from confined rooms and crowded streets to well-ventilated dwellings, dry soil, and pure air; to insist upon their being out of doors all day, indoors by sunset, and in bed all night. At the same time they should be fed with nutritious but unstimulating food, particularly with milk, bread and butter, bacon, eggs (and cod-liver oil), with as much meat as they can digest, and with malt liquor only to help appetite and digestion. When a youth belonging to a phthisical family shows signs of wasting, pallor, weakness, and want of healthy appetite, he may often be saved from the consumption which threatens him by being sent to live in the country for several months, with strict attention to early hours, nourishing diet, and sedulous avoidance of all that is exciting, enervating, and exhausting.

It is in many, though not in all, cases desirable for a patient with *very early symptoms* of the disease to leave England for a time, and take a long sea voyage—to the Cape or Australia or Buenos Ayres or Japan. Sailing

* See an interesting paper by Sir Hugh Beevor on "The Declension of Phthisis" ('Lancet,' April 15th, 1899).

† Among specific systems of treatment which like "tuberculin" have been introduced with high praise and found useless in practice, may be mentioned the serum treatment of Prof. Maragliano of Genoa (1895), the subcutaneous or rectal injection of "antiphthisin" by Dr Denison, of Denver, Colorado, the hypodermic use of nuclein by Dr Wilcox, of New York, the inhalation of *igazolo* (formic aldehyde) by Cervello, of Palermo (1899), and hydro-therapeutic treatment in general. See Dr C. T. Williams's paper in the 82nd volume of the 'Med. Chir. Trans.' (1899).

ships are preferable to steamers, because of the greater length of the voyage, which renders the changes of climate less sudden and trying; while the greater cleanliness and quiet, together with the easier motion, are strong recommendations. But, unfortunately, sailing vessels suitable and well equipped for passengers are now seldom to be found. The heat in crossing the tropics is injurious, while during the latter half of the journey to Australia the weather is often cold and stormy. As large a part of the day as possible should be passed in the open air, and exercise on deck should be taken whenever possible. The stay in Australia or in Cape Town should be made as short as possible before the return voyage. No patient should be sent to the antipodes who has pyrexia in the evening; a hot climate is very apt to bring on hæmorrhage, and even slight hæmoptysis is a strong objection to a long sea voyage. Lastly, how much a patient is likely to suffer from sea-sickness cannot be foretold unless it has been proved by former experience.

The climates formerly recommended for consumptive patients were, in the 18th century, Lisbon, where Doddridge died, Pisa, and Leghorn, where Smollett ended his life. Afterwards Montpellier became a favoured resort, then Rome, where Keats died (perhaps the worst place of all), then Madeira and Malaga. These warm and "soft" climates fell out of favour after the middle of the 19th century, and the writer remembers the late Dr Owen Rees, about 1860, teaching that the best place for a patient with phthisis to go to was Canada. But Cannes, Nice, Mentone, San Remo, and the other towns along the French and Italian Riviera are still thronged by consumptive patients; and on other shores of the Mediterranean, Algiers, Ajaccio, Palermo, Corfu, and Egypt still have many visitors, who at least enjoy sunshine and freedom from smoke.

For the last thirty years, however, the strong tendency has been to advise residence in a high mountain region, or at least in a dry, bracing climate much above the sea level.*

Among mountain resorts during the winter are Davos, in the Grisons, about 5200 feet above the sea, the adjacent village of Arosa, St Moritz, Samaden, or Pontresina in the Engadine, or the summit of the Maloja Pass. The peculiarity of the weather in these elevated stations is the stillness and the dryness of the air. In the shade and at night the cold is extreme, but as the sun is powerful, and as the sky is generally clear, patients are able to take exercise nearly every day—walking, skating, skeying, or "tobogganing." At night the double windows in the bedrooms are left slightly open; yet so motionless is the air that the temperature within scarcely falls below 50° Fahr., even when it is from 2° to 16° Fahr. outside. Many who are very liable to "take cold" elsewhere are free from the tendency at Davos. The proper time for a patient to arrive there is about the first or second week in October, or even earlier. It is generally supposed to be undesirable for him to remain after the beginning of April, when the snow melts. He should on no account return to England before the first week in June; and during the interval the choice seems to lie between Beatenberg

* On the climatic treatment of phthisis see "The Treatment of Phthisis by Residence in High Altitudes," by Dr Theodore Williams ('Med.-Chir. Trans.,' 1888); and Dr Weber's Croonian Lectures on "The Hygienic and Climatic Treatment of Consumption" ('Lancet,' March, 1885); the "Climatic Treatment of Phthisis in Colorado," by Dr Charteris, of Glasgow ('Lancet,' November 26th, 1887); an interesting personal narrative of a patient at Nordrach, in the Schwarzwald, given by Dr Fowler ('Diseases of Lungs,' pp. 393-6, 1898); and Erb's lecture on the subject in Volkmann's Clinical Lectures, No. 271 (1900).

above the lake of Thun, Montreux on the Lake of Geneva, with the various hotels above it, and Monte Generoso above Lago Lugano. The fact that hæmoptysis has occurred does not appear always to forbid sending a consumptive patient to Davos; but the actual presence of pyrexia is an objection, and still more so irritability of the larynx or trachea.

There are other mountain climates from which phthysical patients derive great benefit without being exposed to great cold. This fact was remarked long ago by Dr Archibald Smith, who practised for many years in Peru; indeed it seems to have been familiar to the Peruvians themselves, who regard the valleys of the Andes, from 8000 to 10,000 feet above the sea level, as almost unfailing in the prevention and cure of consumption. As a general rule, it may be said that the nearer the equator the greater the elevation which is necessary to render a mountain region salutary for phthisis. The chief resorts in the Cordilleras appear to be Huanuco and Jauja; but Walshe used to recommend, as more accessible, the plateau of Santa Fé de Bogotà in New Granada, where the mean temperature of each quarter of the year is within a degree or two of 86° Fahr.

Other mountain regions to which phthysical patients may be sent are the table-lands of California and Mexico, Colorado, the neighbourhood of Denver or Kansas City in the States, or Manitoba in Canada. One can hardly doubt that in the Himalayas also there must be valuable resorts; but perhaps the present military sanatoria there may not be at a sufficient elevation for the climate. The writer has seen excellent results from a whole phthysical family going to settle in Colorado.

A prolonged stay in the Southern Hemisphere during what would be the winter of Europe, but is of course summer there, is often very serviceable to phthysical patients. But in Australia a residence in the large towns is at least as bad as in England. Melbourne, in particular, is dusty, with rapid changes of temperature and cold winds, and Sydney and Brisbane are very hot. The best health resorts appear to be certain places in the interior of New South Wales, especially Bathurst, Goulburn, Boural (3000 feet above the sea), and Currajong; but, above all, the Darling Downs, in the south of Queensland, where the weather is cool, dry, and bracing. Tasmania and certain sheltered parts of New Zealand appear to be also suitable.

The South African climate is favourable to phthysical patients, but no stay should be made at the sea-coast; Mr Otter says not within 100 miles of it, nor at a less elevation than 1500 feet. The high lands of the Orange River Colony are dry, equable, and bracing. The drawbacks (before the war) were the dust-storms, the badness of the roads, and the roughness of the accommodation. Bloemfontein has an exceedingly dry climate, but the daily range of temperature is great.

The chief object, however, is to avoid towns, and to choose an equable, dry climate, with good nourishment and comfort. The value of this or that "health resort" has probably been overrated; for it seems likely that the good effects of change of climate depend partly upon its improving the general health and increasing the resistance of the organism to the further progress of the disease, partly upon its protecting the patient from fresh attacks of bronchial catarrh.

It is also important to avoid fresh infection, because tubercle has no restricted period, and does not confer immunity against a future attack. In favourable conditions the bacilli die, the caseous matter dries up, and even

vomicæ may cicatrise. Then, if no fresh invasion take place, the patient may be said to be cured.

When the complete arrest of phthisis is no longer probable, much may still be done to prolong the patient's life and to give him relief from suffering. The climates called sedative are thus very useful; for example, the Channel Isles, Madeira, and the Grand Canary, Samoa or Hawaii in the Pacific, San Remo and some other places on the Riviera, Torquay, Falmouth, Dawlish, Fowey, Penzance, and the Scilly Isles. The desert behind Algiers, and that of Egypt suit some patients very well, although the latter climate is said to have deteriorated since it passed under English protection. Probably a voyage up the Nile is the best thing for patients who dislike cold, and who habitually feel better the hotter they are. That special benefit follows from the air having resinous smells from pine forests—as at Arcachon and Bournemouth—is easier to assume than to explain or to prove.

It must not be forgotten that phthisis sometimes becomes quiescent without any change of climate whatever. On the other hand, although it is a heavy responsibility to advise or allow a patient with phthisis to leave home and its comforts and spend a winter in South Africa, among the Andes, or even at Davos, there is no question that, if he chooses to run the inseparable risks, he has at least a chance of unexpected benefit. A sea voyage is to be deprecated when the consumptive patient is really ill; if the weather is bad it is as injurious as can be, and even in fine weather he often suffers from heat or cold or monotony, and wishes he had stayed at home.

In the last stages of phthisis, when palliation of symptoms is all that can be hoped for, when we have to deal with cough, hectic fever, diarrhœa, and with the other less constant complications, it is most undesirable to send a patient away from friends and home.

One great difficulty we meet with in carrying out climatic treatment in patients liable to, or suspected of phthisis, or suffering from undoubted but still early and favourable disease, is to induce them to continue the treatment long enough. They come back from a voyage, or from a winter and spring spent abroad, stout, strong, and cheerful, with improvement or even disappearance of the physical signs, as well as of the symptoms of phthisis; and they naturally wish to regard themselves as permanently cured and fit to engage again in the pleasures and business of life like other people. This is what too often undoes the benefit gained. When Colorado or South Africa or Egypt has proved beneficial, the patient should make his home there not for months but for years, if he is to be permanently cured.

Of late years disappointment in the extravagant hopes raised by those who recommended one or other of the regions just briefly described, has led to an attempt to secure the same climatic benefits in Europe without the expense and the risks of long voyages and travels. This has been done in Germany by founding Sanatoria for consumptive patients in the hill country—first by Dr Brehmer (1859) at Görbersdorf, in Silesia, afterwards in the Thüringer Wald, the Taunus, and the Black Forest. There have long been similar sanatoria at Les Avants and at Davos in Switzerland, and in the Adirondack Hills in the State of New York. More recently the like provision has been made at Bournemouth, Farnham, and other favourable places in the south of England, and even in the colder and moister Scotch climate. The treatment consists in the combination of pure air, dry soil,

sunny aspect, and various arrangements to ensure regulated exercise and sheltered walks, abundant food, and experienced advice as to diet, clothing, baths, and drugs; and it is probable that we may find the results of such treatment in England, France, and Germany, not inferior to those obtained by travelling a thousand miles in search of what does not exist on earth—a perfect climate. One important advantage in these British sanatoria is that the patients have not to return to a totally different climate or to face a long and tedious journey when returning home. (See an interesting, but too dogmatic, pamphlet by Jaruntowsky, translated by Dr Clifford Beale, and reports of British sanatoria in the ‘Practitioner’ for July, 1900.)

Physic and diet.—There remains to speak of the treatment of phthisis by drugs, a very subordinate, but still necessary part of a rational method. The following is a list of drugs, regarded by Louis as important fifty years ago:—Steel, digitalis, chloride of sodium, carbonate of potash, chloride of ammonium, chloride of lime, hydrocyanic acid, creasote, and iodine. The inhalation of diluted chlorine gas, also, was fully considered and tried in fifty cases of phthisis in the Paris hospitals, and in no instance was there a successful result. Most of the other “remedies” are deservedly forgotten.

Quinine to give appetite or to moderate fever, preparations of iron for anæmia, digitalis to improve the pulse, sedatives to relieve cough or vomiting, and laxatives and stomachics to help the appetite and digestion—these are rational and useful means of treatment; but chlorine has been followed into obscurity by the hypophosphites, by inhalation of carbonic acid gas,* and by inflating the patient’s bowels with sulphuretted hydrogen.

Creasote has lately revived in reputation, and when taken in capsules its unpleasant taste is obviated. It often checks expectoration, relieves gastric catarrh, and thus improves appetite: the dose should begin with half a drop and be gradually increased up to half a drachm. Dr Ransome finds that 5—10 drops of creasote in an emulsion are generally tolerated, and believes it to be of decided benefit. Some physicians, including Dr Poore, have found their patients derive benefit from eating garlic. A recently popular treatment of phthisis is by the allied guaiacol, either as such, or as the carbonate. It is said to relieve cough, check expectoration, and diminish pyrexia, and probably it may have these effects in suitable cases; but the present writer, after giving it a trial, has not seen any striking or frequent benefit apparent from its use. Iodoform in doses of 1—2 grains in pills has often been tried, and sometimes with apparent advantage. Cinamic acid (Zimmtsäure), introduced by Landerer in the form of a sodium salt (so-called “Hetol”) in 1898, is still recommended.

It is well, perhaps, to mention some drugs which have been tried and found useless or directly injurious (in addition to “tuberculin”),—iodine and iodides, mineral acids, hypophosphites, ammonia, chlorides, preparations of gold or of silver, and common salt.

Treatment by inhalation of compressed air,† of oxygen,‡ or of other gases, is of very limited utility. A mode of treatment, introduced by Dr Bergeon, of Lyons (1887), gaseous enemata of sulphuretted hydrogen, has no theoretical probability to recommend it; it is unpleasant in its action,

* This remedy, recommended in 1795 by Dr Beddoes and James Watt, the engineer, was once much employed, and has lately been revived.

† See some recent lectures by Dr T. Williams (‘Brit. Med. Journ.’ 1885, vol. i, p. 769).

‡ See Dr Ransome’s cases (‘Parkes Weber Essay,’ pp. 73—77), which seem to the writer almost conclusive against it.

and after being tried (perhaps with more patience than was due to it) has been shown to be useless.

Since the bacilli of tubercle have been recognised as the cause of phthisis, physicians have naturally attempted to destroy them by antiseptic methods. Iodoform and thymol have been given by the mouth with this object, and more recently sodium sulphocarbolate and phenyl-propionic acid. Creasote, thymol, and eucalyptol have been inhaled by means of orin-nasal respirators (of which the late Sir William Roberts, Dr Burney Yeo, and others have devised ingenious forms); steam inhalers have been supplied with such antiseptic agents as creasote or phenol ("carbolic acid"), and similar solutions have been administered by Siegel's spray apparatus. Sometimes apparent benefit to the local and general symptoms has resulted, and the number of bacilli in the sputum has diminished. But experiments in the laboratory show that Koch's bacilli are extremely difficult to kill, and survive prolonged immersion in germicide solutions. Iodine, corrosive sublimate, and phenol seem to have the most power in destroying them. Perhaps the most ingenious method of "bacillicide" treatment is that invented by the late Dr Cantani, of Naples, who introduced the common *Bacterium termo* of putrefaction into the lungs in a spray, with the hope of its destroying the specific bacillus. Unfortunately the latter is the more powerful of the two; and Dr Theodore Acland reported, after a visit to Naples, that the results of this treatment are negative. 47

Only one medicine has borne the test of long and wide experience, and that is not a specific remedy, but a kind of food which has remarkable power of fattening the patient. Cod-liver oil was introduced for cases of chronic "rheumatism" (vol. i, p. 528), but was first advocated as peculiarly useful in consumption by the late Dr Hughes Bennett. It is best given after food, in doses of from a drachm to half an ounce two or sometimes three times a day. If it causes nausea or vomiting, cream may be substituted for it, or glycerine; and children sometimes improve on extract of malt, with or without cod-liver oil. Nothing, however, is so good as the oil, and with perseverance small doses can almost always be taken, at least during cold weather. It may be taken alone, or in orange or ginger wine, or as an emulsion. Chewing a bit of orange-peel is the best way to prevent the after-taste. In children cod-liver oil is often rubbed into the skin, but the smell is extremely unpleasant, and probably olive oil is just as good. 30

The first aim in the early stages of phthisis, or of threatened phthisis which has not yet begun, is to improve the patient's appetite and digestion. This is one object of change of climate. If he gains weight he generally is doing well. Usually small doses of alkalies, with the milder and less astringent bitters and gentle laxatives, act best in this direction. The combination of carbonate of soda with rhubarb and calumba, long famous at Victoria Park and other hospitals, is excellent for the purpose, with those who prefer disagreeable medicine. In the later stages laxatives must be administered with great caution, for nothing is then more injurious than diarrhoea. Sometimes strychnia and gentian or quassia are borne well and help the appetite. 54

Fatty food, such as bacon, milk, butter, cream, dripping, is indicated. Wine with meals is as a rule desirable; but in early cases, before there is much cough, malt liquors, and particularly porter, are more useful. Brandy is, as a rule, best adapted for advanced cases, and even then should be given in moderate doses; but rum and milk, either before breakfast or

between breakfast and lunch, is a well-known and useful way of giving a nourishing stimulant.

Treatment of special symptoms.—When phthisis sets in acutely, the patient should be limited for a time to a light diet, consisting mainly of milk, without wine or other stimulant. Whether hæmorrhage has occurred or not, a good prescription, if there is much pyrexia, is Niemeyer's pill of quinine (gr. j), digitalis (gr. $\frac{1}{2}$), and opium (gr. $\frac{1}{4}$), to be taken every four or six hours. When the acute symptoms have passed off, the patient may go to the seaside for a few weeks, or to some dry and healthy place inland, such as Tunbridge Wells or Malvern, or Ben Rhydding if the season be suitable. On the Continent he is often sent to Lippspringe, near Paderborn, or to Soden and other health resorts in the Taunus. The seaside seems in such cases to be undesirable.

When severe hæmoptysis appears, the patient must be kept strictly in the recumbent position for two or three weeks. He should not be allowed to talk, and his diet should be limited almost entirely to milk. Everything that is given to him should be cold. Of styptics it is difficult to say which is the best; ergot, gallic acid, acetate of lead, turpentine, hamamelis, have each their advocates, and it sometimes seems necessary to try one after another. A large ice-bag may also be placed over the chest, but it is doubtful what effect, if any, this has on the pulmonary circulation. Probably its chief value is that the patient is prevented from sitting up. The most valuable hæmostatic is immobility in the recumbent position, and the use of enough laudanum, morphia, or paregoric to prevent coughing. After the hæmorrhage has ceased the patient should cautiously be allowed to sit up, and the amount of food is gradually increased, while the pulse and temperature are carefully watched from day to day.*

The *cough* of phthisis must be combated by sedatives. Most prescriptions contain a small dose of opium or morphia, together with tolu, aniseed, benzoic acid, or some other of the so-called expectorants. Paregoric, the pill of ipecacuanha and squill, and the opiate electuary, are well-tried and usually effectual remedies; but as soon as we can we should leave them off, lest they should impair the patient's appetite. When cough is constant and rest at night impossible, no drug is so deservedly trusted as opium.

In most cases of phthisis there is early and marked *anæmia*, indicating the use of steel. This may be given as the sulphate, with small doses of Epsom salts, or in one of the milder preparations, or (if borne well) in the most efficient form, the muriated tincture, with glycerine and quassia or chloroform water. Arsenic may succeed when preparations of iron fail, and now and then has a remarkable effect in restoring appetite, and adding fulness as well as colour to the cheeks. Lately it has been recommended in febrile phthisis, combined with salicylates.

Diarrhœa is often a most distressing complication, and does much to hasten the fatal termination. It is best treated by bismuth and soda, or by the aromatic chalk powder. Dover's powder is often needful in addition, and occasionally a starch and opium enema or a compound lead suppository will give more relief than anything given by the mouth.

The only other symptom that needs special mention is the *night-sweating*. This may sometimes be checked by sponging the chest and the arms at bedtime with vinegar and water. Sometimes it ceases if a sub-

* Calcium chloride in ten-grain doses has been lately used on the basis of the careful observations on coagulation of the blood made by Dr Wright, of Netley.

cutaneous injection of atropine ($\frac{1}{200}$ to $\frac{1}{100}$ of a grain) is given at bedtime, or a dose of belladonna, oxide or sulphate of zinc, gallic acid, or strychnia. But in too many cases it persists in spite of all treatment.

Surgical treatment has been repeatedly applied in the case of large chronic vomicae with thick adhesions, which are, in all but origin, cases of circumscribed empyema (see the judicious remarks in Fowler and Godlee's work, p. 424).

MILIARY TUBERCULOSIS OF THE LUNGS.—It was an advance in pathology when it was discovered that the lungs may be the seat of extensive tuberculosis without the anatomical changes of phthisis. And it is clinically important to remember that pulmonary tuberculosis in this form is not a chronic wasting disease with hæmoptysis and the familiar symptoms and signs of phthisis, but an acute fever with cyanosis and other symptoms of capillary bronchitis, ending after a short course in death by coma.

Nevertheless it was a mistake to separate the two diseases altogether, as was done by many pathologists, when the unity and tuberculous nature of all cases of phthisis were doubted or denied. For both are identical in cause—the invasion of the same microbe; and in adults at least we usually find that the outbreak of acute miliary tuberculosis throughout the lungs is only a sequel of old and quiescent consolidation of the apex; so that, in fact, acute and universal pulmonary tuberculosis is but an extreme case of the gradual downward spread of miliary tubercles throughout the lungs, which is part of the constant process of phthisis.

In children, acute miliary tuberculosis of the lungs is frequently, as it is exceptionally in adults, secondary not to pulmonary phthisis, but to caseous tuberculosis of the lymph-glands or other parts. At any age it is never a direct and primary invasion of the lungs by the bacillus, and at all ages it is associated with acute miliary tuberculosis of the serous membranes, the pia mater, and other organs.

If the bacilli also invade the membranes of the brain, they usually, though not invariably, give to the disease the clinical features of meningitis. If the peritoneum is much involved, the abdomen alone may appear to suffer. So that it is often almost an accident whether a case is regarded during life as one of tuberculous meningitis, tuberculous peritonitis, or miliary tuberculosis of the lungs; while there are other cases which run their entire course without definite localisation. Nor does the severity of the pulmonary symptoms always answer to the number of miliary tubercles in the lungs; in cases clinically regarded as tuberculous meningitis the lungs are often found as full of tubercles as the pia mater.

Anatomy.—Many cases occur in which the greater abundance and more advanced state of the tubercles in the upper lobes compared with those towards the bases, prove that the proclivities of the pulmonary tissue in different regions produce their effect on this form of tubercle as well as on phthisis. As a rule, if a general outbreak of miliary tubercles occurs when the apex of only one lung is affected with phthisis, the tubercles in that lung are more numerous than on the opposite one. Sometimes they are grey, and do not undergo caseation, but fibrous induration (vol. i, pp. 363, 366); sometimes they become caseous almost as soon as they are formed. In some exceptional instances the tubercles near the apices soften in the centre, and form minute vomicae.

Usually the lungs are oedematous, sometimes congested, and, as a rule,

two additional lesions may be found if looked for—collapse of certain areas of the pulmonary tissue, and lobular catarrh.

Physical signs.—These are in most cases vague and doubtful. Probably miliary tubercles are never set sufficiently close together, even in the apex of a lung, to impair the percussion resonance of the corresponding part of the chest. Sometimes the sound is slightly dull beneath one or both of the clavicles; but this may be due, not to the tubercles, but to collapse of the surrounding tissue. Dr Eustace Smith remarks that in children the latter interpretation is borne out by the fact that variations may be observed from day to day, the resonance returning where it had been deficient; and the case of an adult patient will be presently mentioned in which the same was observed. Again, it is not uncommon for the presence of pulmonary emphysema to render the percussion-sound over-resonant; and the progressive emaciation of the patient makes the still spongy parts of the lung give a more resonant note. In most cases any dulness under the clavicle is due not to the acute tuberculosis, but to the preceding chronic phthisis.

With the stethoscope one may be able to detect nothing abnormal, even when tubercles exist in great numbers. Now and then, especially towards one apex, the vesicular murmur may have a rough, harsh quality compared with the other side, or the rhonchi of bronchitis may be present, or mucous rattles. The expiration, too, may be prolonged. In such cases the smaller tubes are found after death to be filled with muco-pus—clear proof of bronchitis. Sometimes the moist sounds are so consonating in quality over the upper lobes as to denote diffuse infiltration of the pulmonary tissue. In one such case we found many small cavities, especially in the left apex, evidently of older date than the miliary grey tubercles, which filled every organ in the body; and the patient had been troubled with a cough for three months, although his acute illness began only ten days before death. In many instances the autopsy has shown that the lesions were much less advanced than had been thought during life.

In the case of a woman aged twenty-five, admitted into Guy's Hospital on July 19th, 1882, the only physical sign was a slight crackling sound heard at the right apex after she coughed. On the 28th there was a marked crepitant râle in both upper lobes, especially along the anterior edges of the lungs; and during the next three or four days its character became so consonating that we were almost disposed to look upon the disease as acute pneumonic phthisis rather than as miliary tuberculosis. But at the autopsy, made on August 4th, the lungs, though bulky and œdematous, everywhere contained air; the tubercles were discrete and caseous only in the upper lobes.—C. H. F.

Occasionally a sound like a pleuritic rub has been heard. In one case, recorded by Jürgensen ('Berl. klin. Wochensch.,' 1872), a soft rub was felt as well as heard for five days, and after death no pleurisy was present, but a multitude of miliary tubercles on the surface of the lung. Burkart has since stated (in vol. xii of the 'Deutsches Archiv') that he has twice detected a rough friction-sound, due to the presence of obsolete tubercles.

Clinical symptoms.—There is always more or less *cough*, generally short and hacking, and often dry. The *sputum*, when present, may be clear mucus; occasionally it is muco-purulent, or contains streaks of blood. Pure blood is seldom spat in any considerable quantity, but the following case shows that this may occur.

In 1869 there was brought into Guy's Hospital the dead body of a child aged five, who was said to have been well on the previous evening, and to have eaten some herring for supper. In the course of the night it was found to have brought up blood, and to be very

ill, and it died on its way to the hospital. An autopsy showed that there was acute general tuberculosis; and some of the tubercles in the lungs were already caseating, especially in the upper lobes; but no definite source for the bleeding could be discovered; the pulmonary tissue was mottled with blood drawn into it by inhalation.—C. H. F.

In all probability the cause of hæmoptysis in such cases is the extremely congested state of the vessels immediately around the tubercles, which often gives them the appearance of being surrounded by a reddish-brown border after death; in fact, obvious points of capillary hæmorrhage may sometimes be seen, not only in the lungs, but in other organs.

Far more significant than cough or sputum is *dyspnœa*. The respirations gradually increase up to fifty or sixty in the minute, and in children to eighty or ninety. In the woman aged twenty-five, whose case was mentioned above, it was counted at fifty-six on the day of her admission. After a time the patient becomes conscious of shortness of breath; there is orthopnœa, the movements of the thoracic muscles are forced, and the nostrils work, while the cheeks, the lips, and the nails become purple. This cyanosis, more than any other symptom, suggests the idea of pulmonary tuberculosis, when there is no long-standing emphysema or heart disease to account for it.

Sometimes albumen appears in the urine, probably from venous congestion, and there is not infrequently slight œdema of the legs.

Pyrexia is invariably present, but varies greatly in its degree and course. Sometimes the temperature ranges up to 104° or 105° , but more often it remains at a lower level, perhaps not exceeding 102° . Its progress is irregular. For two or three days there may be scarcely any differences in the thermometric readings in the twenty-four hours; and then the usual diurnal variations may appear in an exaggerated form, or what is termed the *typus inversus* may show itself, *i. e.* the morning temperature is higher than that of the evening.

The onset of the pyrexia is usually gradual, and the patient does not take to his bed until he has been ailing for some days. But Rühle speaks of an initial rigor as not infrequent. There are febrile symptoms, as headache, depression, thirst, and loss of appetite. The skin is often wet with perspiration. Epistaxis occurs in some cases, and herpes may appear about the mouth.

The *pulse* is generally very rapid—often out of proportion to the height of the temperature. There may be a flush on the cheeks, but the face is more usually pale, and at last becomes livid.

In most cases of miliary tuberculosis, enlargement of the spleen may be detected by careful percussion, if not by palpation. Rühle says that if tubercles are developed in the spleen it may be tender on pressure, and as large as in enteric fever; and this was the case in a patient under the writer's care in December, 1888.

Towards the end a typhoid state may supervene, with sordes, a dry brown tongue, subsultus, delirium, and coma. Death is sometimes preceded by a rise of temperature, sometimes by a fall and by collapse.

Diagnosis.—One is often helped in the recognition of miliary tuberculosis of the lungs by indications of a like affection of some other organ. Thus the case may at any period of its course become complicated with symptoms of tuberculous meningitis or peritonitis. Occasionally tuberculous synovitis or caries may help in clearing up the diagnosis.

A striking example of this was recorded by Laveran in the 'Progrès Médical' for 1877. A man, aged twenty-two, was attacked with articular pains, especially in the knees.

Effusion occurred into the right knee-joint, and when admitted into hospital he was supposed to be suffering from subacute rheumatism. However, at the end of a week great dyspnoea set in and high fever, the temperature ranging from 102° to 104° . A fortnight later he died from acute tuberculosis. The synovial membrane of the right knee was found to be injected and covered with greyish granulations the size of pin's heads, which could be felt. A few were also present in the left knee.

In 1867 a woman died in Guy's Hospital of tuberculous meningitis, whose right knee had become swollen and painful in the course of her illness. At the autopsy all that was noted was that the synovial membrane was very vascular and œdematous, and that the cartilage over the external condyle of the femur was slightly eroded. But it is not at all unlikely that tubercles were present.—C. H. F.

Cornil and Ranvier have shown that in cases of acute tuberculosis miliary granulations may often be found in the cancellous tissue of the bones, especially in the vertebræ, the sternum, and the ribs.

From a clinical point of view the most important disease to distinguish from acute tuberculosis is enteric fever. Both diseases occur in patients of the same age, both are attended with pyrexia, both run an acute course, and in both the lungs, as well as the abdomen, are the seat of disturbance. The temperature in tuberculosis is less regular, and is interrupted by sub-normal readings; the ratio of respiration and pulse to temperature is greater, and cyanosis comes on earlier. Moreover some chronic disease of the pulmonary apices is often to be discovered. The rash and the characteristic stools are absent, but both may be so in exceptional cases of enteric fever. Ehrlich's diazo-reaction of the urine in Enterica may also occur in acute tuberculosis; but, so far as the writer knows, the serum-test of typhoid fever does not.

Next to enterica we must remember the possibility of pyæmia, and particularly of internal pyæmia, such as may follow endocarditis or caries of the internal ear. In these cases the temperature is usually more irregular, and both higher and lower at intervals, than in acute tuberculosis.

Except in children, acute miliary tuberculosis of the lungs is accompanied by physical signs of previous phthisis, often of old standing or apparently obsolete; or if these are masked by the more recent malady, we have a history of cough, wasting, or hæmoptysis; or the clubbing of the fingers may point to long-standing cyanosis. The prevalence of phthisis or other tubercular disease in the family is another point which often helps in deciding the diagnosis of an acute febrile attack.

Acute capillary bronchitis, when it occurs in a young adult, is sometimes most difficult to distinguish from miliary tuberculosis of the lungs; or, rather, it is difficult to decide whether capillary bronchitis is due to the presence of tubercles or is the only disease. In young adults the latter is improbable, but the writer had two fatal cases during the winter of 1889-90, in a previously healthy young man and a young woman, in both of whom, after death, no tubercles or other complication were discovered.

As a help to diagnosis, the most remarkable seat of miliary tubercles is one where they can actually be seen during life. In 1857 Manz discovered tubercles in the *choroid* of each eyeball in the body of a girl who had died of acute tuberculosis. In 1867-8 Cohnheim, investigating this point carefully in all the cases of miliary tuberculosis—eighteen in number—that came under his notice in the Pathological Institute at Berlin during a period of fourteen months, found that in every instance one or both of the eyes showed choroidal tubercles. In April, 1867, the ophthalmoscope was for the first time used, apparently by v. Graefe himself, for the discovery of these tubercles during life in a patient of Griesinger's; and in November of

the same year Mr Soelberg Wells showed the Pathological Society a specimen of choroidal tuberculosis, which he had detected in a child five days before her death. Although choroidal tubercles are frequently met with, they are not so often present in miliary tuberculosis as Cohnheim supposed.

One of the most remarkable cases was recorded by Fränkel in 1872 in the 'Berliner klin. Wochenschrift.' A delicate girl of six was attacked in May of 1871 with slight shivering, and her temperature rose occasionally to $100\cdot4^{\circ}$. Then partial ptosis appeared, and afterwards paralysis of some of the ocular muscles. On May 22nd the ophthalmoscope showed a white patch to the upper and inner side of the disc in the left eye; it was as large as the disc itself, and had a rounded form, except that in one direction it was drawn out into a point. By the 1st of June it had increased in size by one half. On account of its characters being so different from those generally described as belonging to tubercles in the choroid, Fränkel hesitated to diagnose it as tubercular. The child now went into the country with her parents and remained there until August, when she came back apparently in perfect health. The patch in the fundus of the eyeball, however, was more prominent, though not larger than before. On August 21st she became ill with gastric symptoms and pyrexia, and died on October 1st. On September 10th five fresh miliary tubercles had been detected in the choroid. Vision remained unimpaired until death.

In acute cases the number and the size of the tubercles may increase from day to day; and if nothing is at first detected by the ophthalmoscope, the instrument should be used again and again. The tubercles vary in size up to 2·5 mm. in diameter, and Ponfick met with one that measured 5 mm.; but Cohnheim seems often to have detected them (in the dead body) when they could only be seen after carefully removing the choroidal pigment, and even then they were too small to be visible by the naked eye. They occur in the neighbourhood of the disc or of the yellow spot more often than towards the equator of the eyeball.

Ætiology.—Pulmonary tuberculosis is seldom the result of caseating tuberculous glands which had been recognisable during life. Many of those who are attacked, whether children or adults, are robust and healthy-looking. On the other hand, one must always be prepared for the supervision of miliary tuberculosis in cases of phthisis; for acute miliary tuberculosis is probably, without exception, secondary to chronic tuberculous caseous inflammation of the lungs or some other organ.

Burkart records the case of a woman, aged twenty-eight, who died after a fortnight's illness, and in whom the lungs, the peritoneum, and the kidneys showed recent miliary tubercles, while in the intestine there were the typical lesions of enteric fever. He also cites eight cases recorded by Birch-Hirschfeld, in which acute tuberculosis developed itself after enteric fever. In children the disease often follows measles, scarlet fever, and smallpox.

Acute tuberculosis occurs at all ages, but is most frequent in young adults. Among forty cases observed at Guy's Hospital by Dr Fagge during five years, one patient was an infant aged nine months, three were patients between one and ten years, seven between eleven and twenty, thirteen between twenty-one and thirty, six between thirty-one and forty, four between forty-one and fifty, six between fifty-one and sixty. This list does not include the cases in which the clinical symptoms were those of tuberculous meningitis; if they were taken in, the proportion of children would be far higher. Burkart gives very similar figures. Among the above cases there were almost exactly twice as many males as females; among Burkart's the proportion was as sixteen to two.

Prognosis.—The duration of the disease is commonly three to four weeks, reckoning from the first marked symptoms up to the time of the

patient's death ; but occasionally it may be protracted for three or four, or even for eight months without going on into ordinary phthisis. Clinically, acute pulmonary tuberculosis resembles severe primary bronchitis, and is not seldom mistaken for it until an autopsy reveals the real nature of the case. Indeed, as Burkart has remarked, it is common for miliary tuberculosis to develop itself in lungs which are already emphysematous from old bronchitis ; twelve of his eighteen cases were examples of this, and their course was often very prolonged. In no fewer than six of these, in fact, the tubercles were already calcified or fibrous, with a lustre like that of mother-of-pearl, so that they might fairly be considered obsolete. The cause of death was sometimes pleural effusion, sometimes dilatation of the right side of the heart, sometimes Bright's disease ; and the presence of the tubercles seemed to be little more than an accident. A similar obsolescence of pulmonary tubercles was recorded by Dr Fagge.

This question of the possible involution of miliary tubercles in the lungs is of great interest. We usually regard the fact that a patient recovers as proof that he was not suffering from miliary tuberculosis ; although Dr Bristowe believed that the disease may be occasionally arrested, with more or less permanent damage to the lung. He speaks of the lung, after the arrest of the discrete tubercles, as becoming "seamed throughout with minute patches of cicatrical tissue, the fibres of which have something of a stellular arrangement, and within the limits of which the lung-tissue presents, from the presence of concurrent emphysema, a coarsely spongy character ; occasionally, in the centres of the scars, minute fibroid knots or concretions may be recognised."

Perhaps the only way in which recovery from miliary tuberculosis could be proved would be by the discovery of tubercles in the choroid of the eye ; or by the patient, after recovery, dying from some other cause, and the tubercles being found in a state of obsolescence.

With regard to the *treatment* of miliary tuberculosis of the lungs, we can only seek to obviate the tendency to death from dyspnœa. Cupping between the shoulders, or bleeding from the arm, inhalation of oxygen gas, subcutaneous injection of strychnia, and similar measures may, it is conceivable, keep the patient alive until the brunt of the attack is over, and allow of the tubercles undergoing involution if they can.

From what has been said of the difficulty of diagnosis, we may often derive hope from the possibility that our diagnosis may be wrong.

ASTHMA

“Anthony Henley’s father, dying of an Asthma, said, ‘Well, if I can get this breath once out, I’ll take care it shall never get in again.’”—SWIFT.

ASTHMA—*History—Description of an attack—Diagnosis and relation to structural lesions of the lung—Ætiology—Nature and physiology of the disease—Prognosis and treatment.*

HAY-FEVER—*Onset and course—Ætiology—distribution among persons and places—treatment.*

FROM ancient times in Greece until the present century the word asthma* was used to mean what we now call dyspnoea. Like Pleurisy and Colic, it was a symptom, not a disease in the modern sense of the term. Even at the present day we hear persons spoken of as “asthmatic” who are suffering from bronchitis, phthisis, emphysema, Bright’s disease, or affections of the heart. Soon after the discovery of auscultation Rostan and some other French physicians scarcely recognised any primary disease which required the name of asthma. But of its existence there can be no doubt; and now that true or spasmodic asthma has been separated from other disorders with which it used to be confounded, its characters are found to be definite and well marked. The phrase “cardiac asthma” is no longer admissible, and “asthmatic” should be reserved to apply to asthma in its restricted modern sense.

The true pathology of asthma is unknown, and this chapter might have been placed in the vicinity of Spasmodic Affections of the Nervous System instead of here; but at present it is most conveniently retained among disorders of respiration.

Symptoms of an attack.—Asthma is a paroxysmal disease. It sets in generally with remarkable suddenness; most frequently in the middle of the night, between 2 and 4 a.m., but in some cases between 6 and 8 a.m., or in the afternoon; while the forenoon is almost always the period in the day when the patient is freest from it. In the same case it usually begins at about the same hour.

The patient, who has gone to sleep in perfect health, wakes up in the morning with a sense of oppression of the chest; and this soon passes on to the most extreme distress of breathing. Sometimes, however, the seizure is preceded by symptoms which previous experience enables him to recognise as premonitory—a peculiar drowsiness, flatulence, sneezing, a troublesome itching under the chin, or the passing of copious pale limpid urine like that secreted in hysteria.

* Ἀσθμα (ἀσθμαίνω), *panting*, is a Homeric as well as a Hippocratic word.

There is sometimes a constant preliminary sensation, paræsthesia of various kinds, which Romberg happily called an asthmatic aura.

The urgent dyspnœa of the developed attack compels him to sit up, and perhaps drives him to throw open the window, in the hope of getting air more freely. Or he may be obliged to sit with his elbows planted upon a table, or to stand with his hands grasping some article of furniture above his head, in order to fix the shoulders and so assist the muscles of forced respiration. His face, at first pallid, becomes livid or purple, his eyeballs stare, his hands and feet are cold, his skin is covered with a profuse sweat, and his expression indicates extreme anxiety. In fact, he may appear to be at the point of death.

Examination of the chest shows the following conditions. The breathing is not accelerated, but of normal frequency, or even slower than natural. Its rhythm is reversed, the inspiration being short, while the expiration is greatly prolonged. With the former there may be slight wheezing, but with expiration it is audible all over the room. The shape of the chest is that of a very deep inspiration; the upper ribs are raised to the fullest possible extent, and the diaphragm has descended towards the abdomen, so that the area of pulmonary resonance extends considerably lower than natural.* During inspiration the sterno-mastoidei and the scaleni are brought into action, but there is scarcely any advance in the degree of expansion; during expiration there is but little recession, although the rigid abdominal muscles can be seen and felt to be doing their utmost to expel air from the lungs. Percussion shows much less than the natural amount of difference between inspiration and expiration in the relation of the lungs to the heart and liver; and the percussion-note of the whole chest is over-resonant. On auscultation, the inspiratory murmur is found to be almost inaudible; or it is replaced by sibilus or rhonchus. With expiration there is heard the same loud wheezing sound already mentioned as audible at a distance.

So entirely occupied is the patient with the mere act of breathing that he can scarcely utter a word, or turn his head, or even stop to cough; but after a time a slight cough comes on, leading to the expectoration of a few greyish-white pellets of mucus (cf. p. 101). Expectoration generally indicates that the symptoms are about to subside.

The temperature is not raised, and the pulse little quickened, if at all. Ringer finds the urine after a fit is deficient in both urea and salines.

The duration of a paroxysm of asthma is very variable; usually it lasts from one to three hours, sometimes only a quarter of an hour, and occasionally with but slight remissions for a whole night or a day. As it passes off the patient falls asleep, and when he wakes in the morning his breathing may be quite easy. In some cases, however, the disease continues for several days in succession with scarcely any abatement, except that there is some increase in its severity at night, and some lessening during the early part of the day. Even in such cases, however alarming, a fatal termination scarcely ever occurs.

When asthma passes off in the usual way it is apt to return during the following night; and the paroxysms may recur for several successive nights, and then cease, leaving the patient free for weeks or months together.

* Some good observers (Riegel, Wilson Fox, Goodhart) have occasionally found the opposite condition during the paroxysm;—the chest in a state of expiration,—but they admit that this condition is a rare exception.

There are other cases in which the disease shows itself night after night for years. Thus a friend of Dr Fagge, who was liable to asthma for the last twenty-five or thirty years of his life, was never able to lie down to sleep; when night came on he dressed himself in a flannel suit, and seated himself in a large chair, where he remained till morning.

Death as the direct result of a fit of asthma is almost unknown; but the following case, which occurred some years ago at Guy's Hospital, came perilously near it.

One of our students, a healthy and athletic young man, devoted to football, was admitted into the Clinical Ward one evening, labouring under a severe attack of asthma, to which he was subject. A few hours later, the house physician, who was sleeping in an adjoining room, was hastily summoned on account of an alarming failure of the patient's breathing. His respirations became more and more shallow, and at last they ceased altogether; he fell forwards in a state of insensibility, and remained unconscious for several minutes. Artificial respiration was resorted to at once, and with success; but if medical aid had been less prompt there is little doubt that he would have died.

Diagnosis.—From the above description of asthma it will be apparent that the disease ought never to be mistaken for those tracheal or laryngeal affections (such as bilateral paralysis of the abductors of the cords) in which the dyspnoea is mainly inspiratory.

But it is often only by the history that one can tell whether a patient is suffering from asthma or from bronchitis and emphysema. According to Trousseau, a child (or even an adult) may be seized with what appears to be an acute and dangerous attack of broncho-pneumonia, with abundant moist sounds over the chest; and yet the subsidence of the symptoms in the course of a day or two, and the recurrence of like attacks on future occasions, ultimately shows that the affection is really asthma.

The relation of bronchitis and emphysema to asthma is twofold. On the one hand, patients who have chronic bronchitis may suffer from time to time from paroxysms of dyspnoea, which cannot be accounted for by any increase of the bronchitis, and seem referable to a spasm of the air-tubes; if such spasm is the essential condition in asthma, it may be said that in these cases *secondary* asthma is present as a complication of the bronchitis. "Bronchitic asthma" was recognised by Salter; it is constantly worse in the winter than in the summer, which is not generally the case with primary asthma.

On the other hand, if a person with healthy thoracic viscera becomes subject to frequently recurring attacks of asthma, his lungs soon or later become emphysematous. We have seen that during the paroxysm of asthma the ribs are raised, and the diaphragm at a lower level than natural; in other words, the lungs are in a state of over-distension. When the symptoms quickly pass off, as is usually the case, the chest walls return in a few hours to their normal position and the lungs to their normal size. But if similar attacks recur again and again at short intervals, the inevitable result is that the elasticity of the pulmonary tissue is impaired, and thus the alveoli become permanently over-stretched and emphysematous; ultimately the right side of the heart dilates, dropsy sets in, and there follow the other mechanical results of general venous congestion, as in the last stage of chronic bronchitis.

Patients with confirmed asthma gradually acquire a characteristic aspect, which was well described by Salter. They are round-backed, high-shouldered, and stooping; the chest is rigid and almost motionless, and

from it the arms hang suspended, but inclined rather backwards and bent at the elbows. They are wasted, sometimes to emaciation, with prominent thoracic veins, cold thin hands, and a dusky complexion. The cheeks are hollow, the eyeballs watery, the mouth half-open, and the jaw hanging. The voice is feeble and somewhat hoarse.

Diagnosis must rest on the natural history of the disease, its early origin, its exciting causes, its paroxysmal attacks, and the condition of the patient between them.

The real difficulty is to distinguish idiopathic or primary asthma from the paroxysmal dyspnoea which is only a symptom of angina pectoris, or aneurysm, of uræmia or bronchitis, of emphysema or heart-disease, of laryngismus or of hysteria.

There is none of the pain of angina, and the paroxysms last much longer; the heart and great vessels, and the urine are found unaffected; the dyspnoea is expiratory; and the respiration is not nearly so frequent, though it is quite as shallow, as in the false dyspnoea of hysteria.

Ætiology.—With respect to the causes of asthma two questions arise: first, What are the *predisposing conditions* which render certain persons susceptible of the disease, whereas others seem to be immune? and secondly, What are the *exciting causes* which are found to bring on the paroxysms in those who are predisposed?

The answer to the first of these questions is at present incomplete; while to a third question of the *causa efficiens*, the invariable antecedent of asthma, either regarded as a paroxysm or as a diathesis, we are quite unable to reply.

Inheritance has a share in the ætiology of asthma. Salter gives many striking instances of the transmission of asthma from generation to generation; and also mentions cases in which several brothers and sisters in a family were asthmatic without the parents being so. Walshe likewise gives instances of three or four brothers or sisters being asthmatic without the disease having appeared in the preceding generation. This kind of connection, which is not hereditary so much as consanguineous, we have already noticed in the case of certain undoubtedly nervous maladies, *e.g.* Thomsen's disease (vol. i, p. 872). Burkart found a history of parental asthma in only 16 out of 100 patients, Goodhart in 25 out of 123.

In early life many cases appear to be directly traceable either to measles, to whooping-cough, or to an attack of ordinary bronchitis; at the time, the child seems to recover perfectly, but it becomes for the future liable to asthma.

Salter relates the case of an epileptic patient whose fits, after the usual premonitory symptoms, were sometimes replaced by asthmatic paroxysms. This is, however, very exceptional; nor is it pretended that there is any connection between asthma and hysteria.

In another of Salter's patients a violent attack of asthma was twice suddenly excited by fear; and Walshe tells how an asthmatic patient, who had forgotten to take his cigarettes out with him, was so alarmed on discovering his omission that the dreaded attack at once came on.

Asthma, like most other obscure ailments, is said in England to be related to gout, and in the tropics to malaria. It has also been supposed to alternate with eczema, or even psoriasis, becoming worse when the skin has got better; and Sir Andrew Clark called attention to the co-existence or alternation of asthma and urticaria, and believed that the swelling of

the bronchial mucous membrane and the cutaneous wheals may be the result of sudden vaso-motor disturbance.*

The *age* at which patients first become affected with asthma is much earlier than was formerly supposed. Salter found that in a fourth of his cases (71 out of 225) it had begun before the tenth year, and he saw two cases in infants, one fourteen and the other twenty-eight days old. The change of opinion as to the age at which asthma appears does not affect the facts but the terms of pathology. While asthma meant dyspnoea, it was true that most asthmatics were middle-aged or old; now that it means paroxysmal spasmodic attacks, it is true that it usually begins in youth and is rare in old age.

More *males* than females suffer from asthma—probably twice as many. This agrees with the liability of males, and particularly male children, to infantile palsy and other nervous diseases (excepting chorea).

Asthma is a disease more common in the well-to-do than in the poor, though cases are not wanting in the out-patient room of a London hospital.

The *exciting causes* of the asthmatic paroxysm vary widely in different cases.

Certain kinds of weather, certain winds, cold air, the confined air of crowded rooms or railway carriages, act as exciting causes in some cases. Or the attack may follow the inhalation of dust, or smoke, even the smoke of an extinguished candle or of a lucifer match.

Some patients are sure to be seized with asthma if they come near certain kinds of animals: cats, rabbits, dogs, horses, guinea-pigs, or the smaller Carnivora of a managerie. Salter relates many remarkable cases of this kind, and what is especially noteworthy is that years have often passed before the patient discovered to what his sufferings were due. One man, a livery-stable keeper, was continually asthmatic until he retired from business, and then became almost entirely free; but whenever he went back among the horses the disease returned, and so at last he found out what was for him the special exciting cause. Dr Fagge knew a lady who was attacked with asthma whenever she was in the same room with a cat; she could at once discover its presence by a painful sense of constriction in the air-passages.

The asthma produced by hay, or rather by the pollen of grasses, is one form of the disease (v. *infra*, p. 102). Some patients never have asthma unless they are exposed to the influence of the pollen; with others this is only one of many exciting causes. Many persons are sure to suffer if they inhale the powder of ipecacuanha, and odours of various kinds bring on asthma in particular patients.†

Diet plays an important part in setting up the paroxysms in almost all asthmatic patients. Heavy suppers and late dinners are very injurious; many asthmatic persons are unable to eat any solid food for several hours before bedtime. Articles of food particularly apt to provoke the disease are cheese, nuts, coffee, bottled stout, and wine.

Sometimes the attacks are clearly traceable to uterine irritation, as when they return with each catamenial period, or come on only during pregnancy or parturition.

Another occasional cause of asthma is the presence of polypi in the nose. This fact was first noticed by Voltolini, and has since been confirmed by

* See a case in point related by Dr T. D. Pryce in the 'Lancet' for May, 1886.

† See the graphic narratives of such cases of asthma given by Watson and Trousseau.

Haenisch and many other observers. In such cases, removal of the nasal growths frees the patient from the liability to asthma.

Of late years varicose veins and swollen mucous membrane have been detected in many cases of asthma where no polypi were present, and the inferior turbinated bone has been removed as a curative measure. But though the connection between this form of reflex irritation and asthma is important, it is far from being constant.

Of all the exciting causes of asthma the most remarkable is locality. In certain places the patient is sure to be attacked; in others he is as sure to escape. As a rule, the places most favourable for asthmatic subjects are large, crowded, smoky towns, like London, Glasgow, and Manchester. The most extraordinary stories are related by Salter of the effects of London air. Persons whose lives had been rendered miserable for years have become entirely free from asthma after moving to London. He thought that three fourths or more of all cases of asthma might be cured in this way. Some asthmatic patients breathe comfortably in a London fog or in the underground railway, and one of Walshe's found that he could only live in perfect freedom from his asthma in the Seven Dials. Nay, the influence of locality may neutralise the ill effects of some exciting cause of the paroxysms; the patient may be able while in London to eat what he pleases and at whatever hours, whereas in the country the strictest dieting may be required to keep off the disease. On the other hand, there are a few cases in which the air of the sea-side or of a bracing hilly district is found to be the best.

Pathology.—Various explanations have been given of the paroxysms of asthma, but even now the pathology of the disease is not fully known. There is clearly obstruction to the passage of the air in expiration,* and it is generally supposed that the cause is spasm of the muscular fibres of the bronchial tubes. That these fibres are capable of contracting, so as to narrow to some extent the calibre of the tubes, was long ago established experimentally by the late Dr C. J. B. Williams, and by Paul Bert, and others since. The suddenness of the onset of the asthmatic paroxysm, the equally sudden way in which it sometimes subsides under the influence of a violent mental shock or emotion, the marked effect of such remedies as chloral, belladonna, and stramonium in bringing it to an end, all favour the view that it is essentially spasmodic in nature. On the other hand, it is by no means clear that spasm alone can produce such extreme narrowing of the tubes as must be present in asthma; or that spasm can be kept up so long as a protracted paroxysm of asthma.

A more recent hypothesis is that the mucous membrane of the tubes becomes rapidly swollen by what German writers term a "fluxionary hyperæmia," or (as Weber put it) by "a dilatation of its blood-vessels through the influence of the vaso-motor nerves." The fact that the catarrhal form of hay-fever is attended with an obvious swelling of the mucous membrane of the nose is a strong point in favour of this view, since it seems unlikely for the asthmatic form of the same disease to be altogether different in pathology. Sir Andrew Clark regarded this supposed swelling as analogous to that of urticaria, others have compared it to erysipelas. If it is inflammatory it may as well be called catarrhal bronchitis at once; if not, it must

* During the act of expiration the very small bronchial tubes are as much exposed to pressure as the pulmonary alveoli themselves, and it is not difficult to suppose that when from any cause they are partially obstructed, they may admit air into the lungs in inspiration, and yet refuse to allow it to pass out in expiration.—C. H. F.

depend on vaso-motor nerves, and asthma may still be regarded as a paroxysmal neurosis. Störk actually observed with the laryngeal mirror that during an asthmatic attack the whole length of the trachea and part of the right bronchus were deeply congested. But may not this venous stasis have been the result, not the cause, of the dyspnoea?

It is no doubt possible that the small bronchial tubes may be affected both with hyperæmia and with spasm; but a double explanation is seldom true.

In 1871 Leyden discovered in the sputa certain pointed octahedral crystals, identical with those which are found in the blood and viscera in cases of leuchæmia, and known as Charcot's crystals, after their first description by him. Leyden supposed that these crystals might perhaps constitute the starting-point of the asthmatic paroxysm, by irritating the peripheral ends of the branches of the vagi in the bronchial mucous membrane, and so setting up a reflex spasm of the muscular fibres beneath. But they have been observed in the sputa of patients not suffering from asthma, although less constantly and in less numbers.

Elongated spiral plugs of mucus have been recognised in the scanty sputa of asthma by Lefèvre, by Curschmann, and by subsequent observers. These, like Charcot's crystals, are found in the mucous expectoration of other diseases, and cannot therefore be regarded as specific or pathognomonic of asthma; and the same remarks seem to be true of the presence of eosinophil cells, which were discovered in asthmatic sputa by Adolph Schmidt.

The microbes found in the expectoration after an attack of asthma are none of them constant or peculiar. The late Dr Kanthack, in a paper before the Cambridge Medical Society (April 1st, 1898), described streptococci, diplococci with a capsule, staphylococci, and minute bacteria, some resembling that of Löffler, but none of them specific.

On the whole the evidence at present is that, as Dr Edward Liveing put it, asthma is a paroxysmal reflex neurosis without any constant local, toxic, or bacterial origin.

Prognosis.—When asthma occurs in childhood, it often subsides about the age of puberty, leaving the patient free for the rest of his life. On the other hand, persons above the age of forty or forty-five seldom if ever get rid of a liability to it. The longer and the more frequent the paroxysms the more serious is the case. It is important to notice whether in the intervals between the attacks there is any shortness of breath, or cough with expectoration. For such symptoms indicate that the asthma is complicated with chronic bronchitis, or emphysema; and the presence of any permanent organic lesion of the lungs seriously adds to the gravity of the disease. The popular notion that "asthma is a lease of long life" is quite mistaken. It only points to the fact that death in the fits is extremely rare, and that so apparently severe a disease does not kill rapidly. All life insurance offices know that asthma leads in time to emphysema and its consequences, and reject or heavily "load" such cases.

Asthma does not tend to develop phthisis; although there are cases which prove that it does not prevent its supervention.

Treatment.—Our object is twofold: first, to prevent the recurrence of the asthma; and secondly, to relieve the paroxysms and, if possible, cut them short.

In endeavouring to prevent the attacks, the most important thing is to

study the exciting causes in the individual patient, and as far as possible to remove them. The digestive organs and the diet next demand attention; and then the place of residence. A drug which is in some cases serviceable is iodide of potassium; but it often fails. Arsenic is, in the writer's experience, more often successful, and bromides are another valuable medicine; but phosphorus is probably useless.

For the paroxysms of asthma, different modes of treatment are useful, some in one case, some in another. Many patients are at once relieved when they are made faint and sick by an emetic dose of ipecacuanha or by smoking tobacco. The latter is said to be the best remedy for hay-asthma; unfortunately, those who smoke habitually get no benefit from it; and with others the remedy is worse than the disease. In some cases nothing does so much good as strong hot coffee taken on an empty stomach, or hot whisky and water, or a subcutaneous injection of morphia. But the last two remedies are dangerous when successful. In other cases the inhalation of chloroform gives very rapid, but generally only temporary relief. Smoking the leaves of stramonium (or of the other species of *Datura*) is often very effectual. Or the patient may try stramonium as a tincture or an extract, or the ethereal tincture of lobelia in full doses, or tincture of belladonna, or chloral hydrate, or grindelia. Some patients derive great relief from the fumes of nitre-paper, burnt so as to fill the room with white smoke. In other cases they benefit by inhaling a green powder, sold in the United States as Himrod's cure for asthma, and said to consist of nitre, stramonium, and aniseed. Chloroform and nitrite of amyl are uncertain in their effects; and galvanism is probably useless. When the paroxysms can be traced to the nasal mucous membrane, surgical interference is indicated.

For details in the management of cases of asthma the reader should consult the late Dr Hyde Salter's work, based as it was upon a large experience, as well as upon his own sufferings from the disease.

HAY-FEVER.*—Some people are liable every summer to a most troublesome complaint, which in many cases assumes the characters of asthma, and is probably allied in its pathology. Attention was first directed to it in England by Dr Bostock in a paper read before the Royal Medical and Chirurgical Society in 1819.

Symptoms.—It usually sets in with a feeling of irritation in the nose, throat, and eyes. Then the patient begins to sneeze—perhaps twenty times in succession; a thin watery secretion runs from his nostrils, and the nasal submucous tissue swells until no air can be drawn through the nose. If, however, he lies down and turns on his side, the nostril which is now uppermost becomes in a short time free, apparently as the result of gravitation of the oedematous fluid. The swelling affects the lachrymal passages also, so that the tears run down over the cheeks. The eyes are often inflamed, and the eyelids swell. These distressing symptoms vary in severity and duration, but often last three or four weeks, or sometimes longer still. Paroxysms of sneezing recur from time to time, and the nasal discharge becomes thicker and more purulent. If left to itself, the malady gradually passes off, leaving more or less weakness behind.

* *Synonyms.*—Hay-asthma—Periodic vaso-motor coryza—Specific or nervous coryza—Catarrhus æstivus—Catarrhus ex fœnisicio—Rose-cold and autumnal catarrh (U.S.A.).—*Fr.* Fièvre, asthme, ou catarrhe de foin, asthme d'été.—*Germ.* Heu-Asthma, Heufieber, Bostock'sher Katarrh.

In many cases there is, along with catarrh and cough, disproportioned dyspnœa occurring in paroxysms also; and in other cases the sneezing and coryza are but slight and the asthmatic symptoms severe. The connection between these several forms lies in their causation.

Exciting cause.—This “summer catarrh” or summer asthma occurs in the beginning of the hay season, and often immediately after being in a hayfield. In 1873 Dr Blackley, of Manchester, discovered by careful observations and experiments upon himself that the true cause of the affection is the diffusion in the air of the pollen of grasses, which settles on the mucous membrane of the eyes and nose.* He found that by introducing a small quantity of pollen into the nostrils he could bring on all the symptoms of the disease. During the summers of 1866 and 1867 he made daily observations on the amount of pollen deposited on glass slides moistened with glycerine and exposed to the air; and he found that there was a pretty close relation between the quantities collected and the severity of the symptoms of his hay-fever from day to day. Blackley showed that when one might imagine that there can be no pollen in the air, it really may be present in abundance. Once he was suddenly seized with asthma while on the shore, with a sea breeze blowing; between him and the sea there was but a narrow belt of land, but upon this he found a field of wheat in full bloom.

Predisposing causes.—Hay-fever frequently appears for the first time about the age of puberty, but sometimes it is observed in children four or five years old. Persons who have reached the age of forty without being affected with it are probably never attacked afterwards. It is more common in men than in women.

It has been noticed that those who suffer from this singular complaint belong, as a rule, to the educated classes. It is rare among gardeners or farm labourers. It may be that predisposition to the disease is the result of an indoors life in towns and cities, where the exciting cause is scarce. If so, one can understand why hay-fever has become more common of late years. In addition to the personal peculiarity, which we may call an idiosyncrasy or neurotic disposition or diathesis, there is a local peculiarity in those subject to this curious malady,—the nasal mucous membrane readily swells and becomes vascular, as has been proved by Voltolini and Hack in Germany, and by Daly and several other physicians in the United States.

Hay-fever is supposed to prevail in England more than on the Continent, but it is also very common in the United States, where it has been carefully investigated by Dr Mackenzie, of Baltimore. In most cases the susceptibility to the disease increases with each successive year. At first the patient may be attacked only when he is actually in a meadow where the grass is in full bloom; ultimately he suffers as soon as he attempts to go into the country during the hay season. Sometimes it assumes the asthmatic form after it has for several years recurred as a catarrh.

The treatment is unsatisfactory. Neither quinine nor arsenic, nor any other medicine appears to have the power of enabling those who are liable to the disease to bear exposure to its exciting cause without being at once attacked by it. The local application of quinine in solution was first used

* Some writers suppose that the irritant acts not mechanically but chemically from the presence of coumarin or coumaric anhydride ($C_9H_6O_2$), an aromatic compound belonging to the Benzene group. It gives its scent to the grass *Anthoxanthum odoratum* and to *Asperula odorata*.

in his own case by Helmholtz, but it has not proved so successful with meaner sufferers. Sir Andrew Clark (after trying and discarding every kind of internal remedy, including aconite and belladonna) found that a solution of eucaine (5·15 per cent.) applied locally with a brush, or as a spray, is sometimes successful, though it often fails. As a more effectual plan, the same physician recommended the thorough local application of a solution of carbolic acid to the interior of the nostril (see the formula and method described in the 'Brit. Med. Journ.,' June 11th, 1887, p. 1256). Some physicians report good results in themselves or in their patients from the use of a solution inhaled into the nostrils from a vaporizer which contains cocaine hydrochlor., quiniæ sulph., and phenazone, with hydrochloric acid and camphor.

When the symptoms begin, considerable relief is afforded by the use of a smelling-bottle containing ammonia, iodine, and carbolic acid, made into a paste with wood-charcoal and compound tincture of benzoin.

The most thoroughgoing treatment is to destroy the secreting parts of the Schneiderian membrane by caustics or galvano-cautery. This severe treatment has been carried out in the United States, and it is said with complete and permanent success.

For those who suffer severely, the only course (apart from local treatment) is to remain in a large town through the whole of the summer months, or else to go to the sea-side, choosing some narrow peninsula or island, or to take a sea voyage. Staying indoors during the middle of the day, even in a country house, often mitigates the symptoms.

The continuance of hay-fever during several weeks—generally from some time in May until the middle of July—depends upon the continued exposure of the patient to the irritant. If he can get away to a place where there is no pollen in the air, the attack quickly passes off.

AFFECTIONS OF THE LARYNX

"Sputaque per fauces raucas vix edita tussi."—LUCRETIVS.

The laryngoscope and its use.

LARYNGEAL PARALYSIS—*Of one or both recurrent nerves—Of the abductor muscles alone—Of the adductors—Aphonia and other disorders of the voice.*

LARYNGISMUS STRIDULUS—*Nomenclature—Symptoms and Diagnosis—Pathology—Ætiology—Prognosis and treatment—Spasm of the glottis in adults.*

LARYNGITIS—*Acute catarrh of the larynx—Plastic laryngitis—Croup—Chronic catarrh of the larynx—Tuberculous laryngitis—Lupus—Syphilis and leprosy of the larynx—Edematous submucous laryngitis—Perichondritis.*

TUMOURS OF THE LARYNX—*Papilloma—Polypus, etc.—Sarcoma and Carcinoma—Laryngeal malformations.*

MOST of the affections of the larynx interfere with the performance of both its functions, the formation of the voice, and the passage of air into and out of the trachea; and many of them cause pain, tenderness, a peculiar cough, or dysphagia. Beside these symptoms, we have in the laryngoscope a means of observing what we may call physical signs of disease. They are, however, more than the signs of physical conditions in the lungs, inasmuch as we actually see the affected parts. We will begin with an account of this instrument, and then pass on to paralytic and spasmodic affections of the larynx, which cause loss of voice, or obstruction to the breath, and furnish important evidence of diseases of distant organs. Their proper place is among the affections of the nerves or nervous centres, but for practical convenience we will take them with other affections of the larynx. After paralytic affections of the laryngeal muscles will follow the remarkable spasmodic disorder known as Laryngismus stridulus; then the several forms of laryngitis, excluding laryngeal diphtheria, which was treated of in the first volume; and lastly, laryngeal tumours and malformations. The symptoms and diagnosis of obstruction of the larynx and trachea will be considered in the following chapter, along with mediastinal inflammation and growths.*

The laryngoscope.—The diseases of the larynx, like those of the retina, have within the last fifty years been made accessible, as they never were before, to actual inspection. This is entirely due to the invention of a special instrument—the laryngoscope—which was first introduced into medical practice in Vienna by Türck and by Czermak. Türck, in the summer of 1857, began to examine his hospital patients with a laryngeal mirror, such as had been used two years before by Manuel Garcia, a Spanish

* In preparing this chapter for the present edition I have to acknowledge the valuable help I have received from my colleague and friend, Mr Charters Symonds, particularly in the sections on chronic laryngitis and on laryngeal tumours.—P. H. P.-S.

singing-master, in London, who read a paper on the voice before the Royal Society. Even this was not the first effort to see the interior of the larynx in the living subject, for it had been attempted, though with imperfect success, by several others, including the younger Dr Babington in 1829, Mr Liston in 1840,* and Mr Avery in 1844. Türk suspended his observations in the winter of 1857-8 for want of sunlight, but lent his mirrors to Czermak; who, setting to work with artificial illumination, became convinced of the great value of laryngoscopy for clinical purposes, and after publishing a paper on the subject in March, 1858, in the '*Wiener medicinische Wochenschrift*,' travelled over Germany, France, and England, to make it more widely known. The writer witnessed Czermak give demonstrations on his own larynx by direct sunlight at Guy's Hospital in 1859.

In using the laryngoscope, the first thing is to secure a bright and steady light. Next to the sun, the best illumination is from an Argand burner, an incandescent gaslight, or an electric lamp. The patient is placed with his back to the light, and the rays pass over his shoulder. The observer seats himself opposite, and throws the light upon the lower part of the patient's face by means of a slightly concave mirror, fixed on a band round the head, or in a spectacle frame made for the purpose. The patient is next made to open his mouth and to protrude his tongue. This is gently grasped by the observer's left thumb and fore-finger in the folds of a towel, and drawn forwards and downwards. Should both hands be needed, the patient holds the tongue, placing the thumb uppermost; but in some patients the tongue is an unruly member, and the only plan may be to keep it depressed with a spatula bent at a right angle. The reflected rays are then directed into the back of the fauces, and kept steadily fixed there. In the meantime a stalked laryngeal mirror is warmed over a flame or in hot water, or it may be smeared with glycerine; the object being to prevent its surface becoming dimmed by the breath.

The patient is now instructed to go on breathing quietly and regularly, and to sound an "a" (as in fate or *fête*) on rather a high note, repeating the sound twice quickly, and then resuming the breathing. This brings the fauces into a position advantageous for the introduction of the laryngeal mirror, which is held like a pen between the finger and thumb, and gently but rapidly passed through the patient's mouth until it reaches the uvula, while the stem lies at the angle of the mouth, so as not to interfere with the entrance of light. In traversing the mouth the instrument must have its face turned downward, and it must take a curved course, being kept close to the palate and as far as possible from the tongue. As it reaches the uvula it must be tilted, so that its face looks forwards as well as downwards, and then gently pushed upwards and backwards, lifting the uvula and the velum. While this is being done, the back of the tongue, the epiglottis, and the interior of the larynx successively come into view, reflected in the mirror. It should not be carried so far back as to touch the pharynx, which in many patients is far more sensitive than the velum; although others bear the mirror well, even when it is made to rest on the pharyngeal surface. It is rarely needful to continue a single observation for more than a very short time. Should the larynx not be completely visible, it is best to withdraw the instrument and re-introduce it after a

* "A view of the parts may be sometimes obtained by means of a speculum. Such a glass is used by dentists on a long stalk previously dipped in hot water, introduced with its reflecting surface downwards, and carried well into the fauces." From the account of ulcerated glottis by Robert Liston, in his '*Practical Surgery*,' quoted by Czermak.

few minutes. When the student has mastered the instrument, he should learn to use the laryngeal mirror with the left hand, so as to have the right hand disengaged.

It may well be supposed that the skilful use of the laryngoscope is often difficult. In this, as in other manœuvres, the student finds himself at first baffled, and fails to see anything, but only needs patience and practice to succeed; and the patient who, when the mirror is first introduced cannot tolerate its presence, becomes after a few trials indifferent.

One difficulty is with the tongue, which in some persons arches upwards so as to interfere not only with the passage of the mirror, but also with the admission of light to the back of the throat. Such patients must practise before a looking-glass until they can "make a wide throat." If on sounding the *a* the tongue is elevated so as to obscure the view, the observer should, in his own case, demonstrate to the patient the possibility of making the sound without raising the tongue. If still unsuccessful after a few attempts, the tongue must be depressed with a long right-angled spatula.

The greatest difficulty arises from the patient holding the breath and closing the pharynx. This is due partly to fear, partly to his not comprehending the directions, and partly to spasm caused by the irritation of the mirror. By directing the patient to take deep breaths, and keeping his attention fully occupied with energetic directions, nearly every case can be managed by a skilled observer.

An occasional obstacle is the presence of enlarged tonsils narrowing the faucial space. This is best overcome by using a small mirror.

If, however, the slightest contact of the mirror causes choking, or retching, or cough, local anæsthesia must be produced by applying cocaine locally in 5 or 10 per cent. solution brushed over the palate, uvula, and pharynx, or, still better, applied as a spray.*

In some patients with catarrh of the throat or lungs, the introduction of the mirror is at once followed by the entrance of a quantity of mucopurulent fluid into the fauces from below, notwithstanding the repeated use of a gargle. Or the uvula may be so long and pendulous that it curls round the under edge of the mirror, interfering with the view of the larynx, or soiling the reflecting surface. The way to correct this is to use a large mirror, so as to lift up the whole of the uvula.

The most serious difficulty is caused by a large epiglottis, which hangs over the entrance of the larynx, and prevents anything else being seen. In many cases this obstacle is easily removed by making the patient sound, or attempt to sound, the vowel *i* (*e*, as in *feet*) on a high note; the sound itself cannot actually be produced while the tongue is protruded, but the effort to produce it is often sufficient to raise the epiglottis. Sometimes the interior of the larynx can be seen, in spite of a pendent epiglottis, if the mirror is placed rather lower in the fauces than usual, and with a more vertical inclination of its surface, the patient's head being at the same time thrown far back. If all these plans fail, an attempt must be made to raise the epiglottis by a curved rod brought into contact with its posterior surface. In most persons the back surface of the epiglottis is so sensitive that a choking sensation is produced as soon as it is touched. But the use of the cocaine solution enables one to draw the epiglottis forward with the blunt hook in the left hand, guided by the mirror held in the right.

* Formerly ice sucked for ten minutes or more, before the mirror was introduced caused temporary bluntness of sensation; and, in addition, the writer remembers giving a full dose of bromide before the attempt with a certain degree of success.

The parts reflected in a laryngeal mirror retain their proper positions so far as concerns the side of the body on which they seem to lie; the left vocal cord is visible upon the left side of the mirror, the right one upon the right side. But in an antero-posterior direction the image is inverted, as if one were looking at the larynx from behind instead of through the mouth; so that the base of the tongue and the epiglottis are seen at the top of the laryngoscopic image, and the arytaenoid cartilages below.

The appearance of the epiglottis varies widely in different persons; sometimes little more than its edge is seen, sometimes a large part of its posterior surface, which has normally a bright red colour, apt to be taken for morbid congestion. The rest of the laryngeal mucous membrane is of a paler tint, and the vocal cords are white and glistening.

PARALYSIS OF ALL THE MUSCLES SUPPLIED BY THE RECURRENT LARYNGEAL NERVE OR NERVES.—The most frequent paralytic affection of the larynx involves all the muscles supplied by the recurrent nerve, either on one side or both.* The paralysis is not quite universal, since the crico-thyroid muscles escape; but it does not appear that any appreciable result follows their contraction when the other laryngeal muscles are powerless. Türk once observed fatty degeneration and atrophy of the crico-thyroid muscle in a case in which the recurrent laryngeal nerve was alone affected, while the superior laryngeal nerve escaped.

Unilateral paralysis of the recurrens is characterised by complete immobility of the corresponding vocal cord, whether the patient only continues to breathe or utters a vocal sound. The cord usually occupies the position termed "cadaveric," the same as that of the vocal cords in the dead body—intermediate between phonation and inspiration. But sometimes the cord is drawn inwards to the middle line by the action of the arytaenoideus. The outline of the cord looks concave; and the summit of the arytaenoid cartilage is a little further forwards and inwards than that of the opposite cartilage, so that it looks larger from more of its hinder surface being seen. When a sound is uttered, the opposite arytaenoid cartilage moves further than usual, and the unaffected vocal cord is drawn up to and even across the middle line, until it may come close to the paralysed one, and so the chink of the glottis becomes oblique. At the same time the summit of the mobile arytaenoid cartilage crosses in front of the cartilage on the paralysed side. When paralysis has lasted a long time the affected cord is atrophied, and may oscillate backwards and forwards as the stream of air passes over it.

The voice of a patient with paralysis of one recurrent nerve is less altered than might have been expected. It is often weak and hoarse, and sometimes breaks into a falsetto as soon as an attempt is made to speak loudly. A point to which Gerhardt drew attention is that when two fingers are placed, one on each side of the thyroid cartilage, while the patient is speaking, a more distinct vibration can be felt with one finger than with the other. There is not the slightest dyspnoea.

Bilateral paralysis of all the muscles supplied by the two recurrent nerves is characterised by immobility of both vocal cords in the cadaveric

* When unilateral, this is sometimes called "hemiplegia of the larynx." If a special name is wanted, it is better to follow the analogy of Mr. Hutchinson's word, ophthalmoplegia, and to speak of "laryngoplegia" when the muscles of both sides of the larynx are affected, and of "hemi-laryngoplegia" when the paralysis is one-sided.

position. It is to be noted, however, that the paralysis is often less complete on one side than on the other.

There is complete aphonia, the voice being reduced to a whisper; and the patient is unable to cough or to expectorate forcibly; but there is no dyspnoea, at least in adults.

Diagnosis.—It must be borne in mind that immobility of the vocal cord, whether on one side or on both, is not in itself proof of paralysis. As Sir Felix Semon has shown, precisely the same laryngoscopic appearances may be the result of ankylosis of the crico-arytænoid joints, either with or without suppuration. It is of course only when the arytænoid cartilages are so fixed as to bring the cords into the cadaveric position that the case can be taken for one of recurrent paralysis, and then, as Semon admits, diagnosis is sometimes impossible.

Pathology.—The causes of paralysis of the muscles supplied by the recurrent nerve or nerves fall into two groups.

(1) There may be *central* disease of the nuclei of the eighth pair of nerves, one or both, in the pons; as in bulbar paralysis or multiple sclerosis. Such paralysis is usually bilateral and almost always secondary.

(2) The disease may be *peripheral*, interfering with the roots or trunks of the spinal accessory and pneumogastric nerves near the base of the skull, or with the trunk of the vagus after its separation from the spinal accessory or with the recurrent laryngeal branch. As a rule, the paralysis is in such cases unilateral. Indeed, by far the most frequent cause of paralysis of the muscles of one half of the larynx is aneurysm of the aorta, in which case the affection is most often on the left side, although aneurysm of the innominate artery may compress the right recurrent nerve. Again, mediastinal growths of various kinds may interfere with the left recurrent, while either the left or the right may be pressed upon by an enlarged thyroid, or by cancer of the œsophagus or the vertebræ. These last-mentioned peripheral causes may affect each recurrent nerve in succession, and produce bilateral paralysis. Mackenzie published a case in which there were two aneurysms of different parts of the aorta, one of which compressed the right and the other the left recurrent nerve; and Bäumlér recorded an interesting example of bilateral paralysis of the recurrent nerves, apparently due to the pressure of a large pericardial effusion.

It is a singular fact that peripheral interference with a single vagus sometimes causes precisely the same result. This was stated by Bäumlér in the 'Pathological Transactions' for 1872, and two years later Sir George Johnson discussed it in the fifty-eighth volume of the 'Med.-Chir. Trans.' The most probable explanation seems to be that ascending neuritis spreads to the bulb by the centripetal fibres of the affected vagus. If so a lesion involving only the recurrent nerve and not the vagus itself must be incapable of producing the same effect, as was well shown by Semon in the 'Berl. klin. Wochenschrift' for 1883. The further spread from one lateral nucleus to the other may be explained by the existence of a close physiological connection between the nuclei of the two sides. But Lockhart Clarke further showed that some of the fibres of origin of the spinal accessory nerve (which include the root-fibres of the recurrent laryngeal) actually pass across the middle line, being derived from the opposite nucleus; so that it is not inconceivable that disturbance of a single nucleus may directly cause bilateral paralysis.

The paralysed muscles become atrophied. Indeed, there is no form of

paralysis in which the resulting muscular atrophy is more obvious on dissection than paralysis of the muscles supplied by the recurrent nerve. In unilateral cases, in particular, the contrast between the whitish-yellow, shrunken crico-arytænoideus posticus on the affected side and the red fleshy muscle opposite to it is most striking. The recurrent nerve also, below the point at which it is compressed, is wasted, and grey instead of white.

The *prognosis* of recurrent paralysis depends upon its cause. In Bäumlér's case, in which it was dependent upon pericardial effusion, the patient recovered his voice quickly as the effusion underwent absorption. As a rule, however, the primary disease is incurable, and the paralysis persists until death.

Apart from possible treatment of the cause of paralysis, it is useless to prescribe strychnia or to apply galvanic or faradic currents.

PARALYSIS OF THE ABDUCTORS OF THE CORDS.—Since paralysis of the recurrent nerve is in most cases the result of a morbid process gradually destroying the fibres of the nerve or its nucleus, it is natural enough that cases should be met with in which some only of the muscles supplied by it suffer, while others escape. But it is a remarkable fact that such incomplete forms of paralysis invariably affect one particular pair of muscles, the *crico-arytænoidei postici* which widen the space between the cords on deep inspiration. Indeed, many cases have been recorded of paralysis of both recurrent laryngeal nerves, and yet no muscle has been affected except the posterior crico-arytænoid on each side. Gerhardt published the first example of this condition in 1863, and an admirable lecture on the subject by Riegel may be found in the second volume of 'German Clinical Lectures,' translated for the New Sydenham Society. The reason for this proclivity of the crico-arytænoid is still obscure. When there is a nuclear lesion in the bulb we might suppose that the nucleus for the fibres to the abductors is distinct from that for the other laryngeal muscles, just as in most cases of bulbar paralysis the lower part of the face is affected while the upper part escapes (vol. i, p. 678). But the result is the same when the disease involves the upper part of the vagus or the recurrent nerve. It would seem that the fibres to the abductors undergo destruction earlier than those to the adductors; indeed, Riegel found in one of his cases, in which both the recurrent nerves were embedded in dense connective tissue, that although most of the fibres had undergone fatty degeneration, some still remained intact. At the International Congress in London, in 1881, Rosenbach pointed out that in paralysis of the limbs there is an analogous fact in the greater liability of the extensors and abductors than of the adductors to suffer. Moreover, when the laryngeal muscles are affected with spasm, the abductors are invariably overpowered by the adductors. The greater liability to paralysis of the abductors, compared with the adductors, is also perhaps related to the remarkable fact discovered by Gad, that cooling down the recurrent laryngeal nerve causes paralysis of the crico-arytænoid muscle first; and lastly, to observations of Horsley and Semon, that after death the abductors lose their contractility before the adductors.

The laryngoscopic appearances which characterise paralysis of a *single abductor* are that the corresponding vocal cord lies more or less near the median line, and does not move outwards as it should when the patient takes a deep breath. During vocalisation it moves inwards as usual: so that unless the state of the larynx is carefully inspected while the patient

is not, as well as while he is, uttering a vocal-sound, this affection will be overlooked.

Apart from a laryngoscopic examination, there are no symptoms of paralysis of a single crico-arytænoides posticus. The patient's voice is of course perfect, and as there is plenty of room for the entrance of air, he experiences no dyspnœa, even on exertion. It is this fact which gives clinical importance to Semon's observations of the invariable occurrence of paralysis of this muscle as the result of disease of the recurrent laryngeal, or of the vagus trunk or nucleus. He has shown that there are cases of aneurysm, of mediastinal growth, of cancer of the œsophagus, and of disease at the base of the brain, on which a routine use of the laryngoscope may throw as much light as does the routine use of the ophthalmoscope on cases of cerebral tumour or chronic Bright's disease.

When *both abductor* muscles are paralysed, the two cords lie nearer one another than they do in health. In cases of long standing they may lie so close together that during inspiration it is scarcely possible to perceive the slightest chink between them; while during expiration they slightly recede from one another. Riegel argues that this extreme narrowing of the glottis is the result of a gradual contracture of the antagonists of the paralysed muscles, as in motor paralysis of the eyeball, the face, or the limbs.

On the other hand, in a case recorded by Feith it seems to have come on within a few days.

One cause of such marked stenosis of the glottis during inspiration is probably a sucking in of the cords towards one another, in consequence of the diminution of atmospheric pressure upon their lower as compared with their upper surfaces.*

The fact that during inspiration the cords are drawn close together near the middle line is not of itself proof that there is paralysis of the abductors; there may be spasm of the adductors. What marks the difference is the way in which the dyspnœa begins. A primary spasm is rapid in its development; the contraction of antagonists which obstruct the glottis in cases of paralysis of the abductors comes on slowly. At first there is difficulty of breathing only when the patient makes some effort or exertion; then it gradually becomes persistent and severe.

When both posterior crico-arytænoids are paralysed, the symptoms may be of the most urgent and dangerous character. The voice is still unimpaired; but there is often distressing inspiratory dyspnœa, the air being slowly drawn into the chest with loud stridor, and the patient becoming livid, with cold extremities, and at last, if unrelieved, dying of suffocation. In such cases the laryngoscope is needed, not so much to distinguish the case from other diseases of the larynx as from stenosis of the trachea.

With regard to the *cause* of paralysis of one or both of the abductor muscles, we have seen that it may be either central, or some lesion of the upper parts of the vagi, or of the recurrent nerves in their course.

In the 'Pathological Transactions' for 1882 a case is recorded by Dr Whipple, in which a bilateral paralysis of the abductors was dependent

* In two cases seen by Semon there was a modification of the usual laryngoscopic appearances; the cords were close together only along their anterior two thirds, and diverged posteriorly so as to leave a triangular opening with its base at the interarytænoid fold. Possibly this depends on a limitation of the paralysis to the outer fibres of the crico-arytænoides postici, their inner fibres escaping; for according to Rühlman the inner portions of these muscles draw the arytænoid cartilages downwards and outwards upon the cricoid; the outer rotate the arytænoid cartilages upon their vertical axes.

upon implication of the left pneumogastric and recurrent nerves in the walls of a thoracic aneurysm. Another local disease which may cause single or even double abductor paralysis is cancer of the œsophagus. In some cases abductor paralysis from peripheral neuritis has followed diphtheria, more frequently influenza, and occasionally it is a sequel of facial erysipelas. It is also one of the symptoms which may complicate tabes.

Prognosis and treatment.—In a few cases recovery may take place after weeks or months, apparently independent of treatment; but far more often the paralysis remains incurable. Relief of the subjective symptoms, with temporary disappearance of the attacks of dyspnœa, may be sometimes obtained by the subcutaneous injection of strychnia ($\frac{1}{30}$ grain of the sulphate gradually increased to $\frac{1}{10}$, daily), or by systematic use of faradisation. The proper method of applying the current is by a laryngeal electrode, with a flat, spade-shaped extremity, that can be laid upon the pharyngeal mucous membrane, over the paralysed crico-arytænoid muscles.

It is important not to rest content with a partial success from this or any other plan of treatment. So long as the objective signs of stenosis of the glottis continue, there is always the risk of a sudden and fatal attack of dyspnœa. In a case recorded by Semon in vols. xi and xii of the 'Clinical Society's Transactions,' the patient's life was just saved at the last moment by tracheotomy, and by artificial respiration continued for three and a half hours. The rule, therefore, seems to be that, as a precaution, the trachea should be opened in every case of paralysis of the abductors with marked dyspnœa.

PARALYSIS OF THE ADDUCTORS OF THE CORDS.—There is a great contrast between paralysis of the abductors and that of the adductors, in their causes, their symptoms, and their course. Paralysis limited to the adductors is never due to organic lesions affecting either the fibres of the vagi or of the recurrent nerves, or their nuclei of origin; while it is not infrequent as the result of conditions which seldom or never cause paralysis of the abductors.

The laryngoscopic appearances which accompany paralysis of the adductors vary somewhat according to the precise seat of the affection; for it is to be borne in mind that the adductors, instead of being (like the abductors) a single pair of muscles, consist of a group of muscles on each side of the larynx, which are classed together by Henle and Luschka under the name of the *sphincter rimæ glottidis*. The pair of muscles which in this country are known as the thyro-arytænoidei, have been supposed to relax the vocal cords, as antagonists of the crico-thyroidei; but German anatomists now divide the muscles in question into two on each side, the "thyro-arytænoidei externi," and the "thyro-arytænoidei interni." The latter are described as a pair of prism-shaped muscles, each of which has one of its edges projecting into the substance of the corresponding vocal cord. Their function is to straighten and approximate the cords in the act of vocalisation. In other words, they co-operate with, instead of being opponents of, the crico-thyroidei; and they are sometimes spoken of as the "internal tensors," the crico-thyroidei as the "external tensors" of the cords.

If, then, the *thyro-arytænoidei interni* are paralysed, the effect is that when the patient attempts to speak, the cords, instead of being straight, are both of them concave, and enclose a narrow oval space. If the affection is unilateral, the glottis is seen bounded by a straight and a curved line.

The width of the space between the cords depends partly upon the pitch of the sound which the patient is trying to utter; it is greater when the pitch is low than when it is high.

Isolated paralysis of the *arytænoideus* muscle causes the rima glottidis to gape posteriorly, between the two arytenoid cartilages, while the cords themselves meet perfectly. The laryngoscopic appearance is then that of a triangle behind, with its apex prolonged into the normal narrow chink.

Another laryngoscopic appearance occasionally observed is that the processus vocalis of each arytenoid cartilage forms an angle inwards; it is believed to indicate a combination of paralysis of the thyro-arytænoidei interni with that of the arytenoideus.

Paralysis of the two *crico-arytænoidei laterales* is said to produce in the laryngeal mirror a figure with an angle corresponding with the processus vocalis on each side, the rima being quadrilateral and lozenge-shaped; but it is doubtful whether these muscles are ever paralysed alone.

If all the muscles forming the *sphincter rimæ glottidis* are paralysed at the same time, the opening of the larynx, when the patient attempts to speak, forms an oval space—not bounded behind by the point of contact between the two processus vocales, as when the thyro-arytænoidei interni alone are affected, but extending back between the arytenoid cartilages.

Lastly, it is said that one may recognise by the laryngoscope, a paralysis limited to the *crico-thyroidei* muscles, which differ from all the rest in being supplied by the superior laryngeal nerves. The indication of this affection is that the rima glottidis presents a wavy outline.

Paralytic affections of some of the muscles forming the sphincter rimæ glottidis may occasionally be associated with spasm of others, so as to make it impossible to determine the precise character of such cases.

The most important and constant symptom in all cases of paralysis of the adductors of the cord is impairment of voice, extending from hoarseness up to the most complete aphonia, so that the patient can only speak in a whisper.* Paretic states of the various muscles give rise not only to hoarseness, but to a great sense of fatigue in speaking or singing, and to inability to maintain the voice for long, or to shout aloud.

FUNCTIONAL DISORDERS OF THE VOICE.—*Aphonia* is not only a symptom of paralysis of the adductor muscles from organic and permanent causes; it is also a functional and transitory condition. Probably in some of these cases there is no true paralysis, for many patients who have complete aphonia nevertheless continue to be able to cough, and also to sneeze; and these reflex acts are accompanied with a laryngeal sound, which clearly proves that for this performance the cords can be perfectly well brought into contact. This is particularly the case with hysterical women, who are the most frequent subjects of paralysis of the adductor muscles. Phthisis is another disease in which paralytic aphonia is not infrequent. Mackenzie in 1865 examined thirty-seven consumptive patients in whom the voice was affected, and found that in eleven of them the affection was purely functional. Sometimes paresis of the thyro-arytænoidei and transverse muscles follows an attack of laryngeal catarrh, and may continue long

* Whether such a total loss of voice is ever the result of the isolated affections of individual muscles seems to be doubtful. Ziemssen says that this effect can hardly be produced even by paralysis of the thyro-arytænoidei interni, so long as the crico-arytænoidei laterales and the arytenoideus remain in action, and bring together the processus vocales of the two arytenoid cartilages.

after the mucous membrane ceases to show congestion. In other cases paralysis of the adductors of the cords has appeared to be due to direct action of cold on the affected nerve-twigs, like paralysis of the portio dura from exposure to a draught (vol. i, p. 577).

Aphonia is often due to over-exertion of the voice, as in clergymen, opera-singers, auctioneers, and school-teachers. Dr Macdonald has described nodules the size of a pin's head on the vocal cords of governesses, and calls them Teachers' nodes. Mackenzie speaks of paralysis of the thyro-arytænoidei interni as being occasionally the result of an actual "sprain" of the muscular tissues by some great effort in the opera or concert-room.

Paralysis of the transversus (or of some of the other muscles) seems to be sometimes dependent upon gummatous or other lesions directly destroying the substance of the muscle. Mackenzie also mentions poisoning by lead or by arsenic as a possible cause of peripheral paralysis from neuritis limited to one or more laryngeal muscles; one case in a painter is described as complete loss of power of the adductor of the right vocal cord.

It is a peculiarity of hysterical aphonia that the patient is apt to regain the voice suddenly under the influence of some strong emotion, and the recovery in such cases is often permanent. Since the introduction of the laryngoscope it has become the usual practice to treat such cases by the application of the induced current to the interior of the larynx, and this often leads to brilliant success. The method is as follows:—One electrode is connected with a metal plate fastened upon a necklet, which is put round the patient's neck so that the metal plate rests on the front of the larynx. The other electrode consists of a small metal ball or sponge fixed to the end of a long curved stem, which can be passed down into the space between the vocal cords. The stem of this laryngeal electrode transmits no current until the end of it has entered the larynx; at that moment the operator with his finger presses a key by which the circuit is completed. It seems to be agreed that the only way by which a successful result can be counted on is to use on the first occasion a pretty strong current, which is painful, and makes the patient utter a loud cry; whereupon the electrode is instantly withdrawn. If less than this be attempted, electricity often fails altogether. In some cases the introduction of an electrode within the larynx is not required; it is sufficient to apply a current across the neck from one side to the other, the electrodes being placed one over each thyroid cartilage. The writer remembers a case of aphonia in a hysterical girl, who was promptly cured by static electricity, and drawing sparks from the larynx.

Alteration of the pitch of the voice.—Among functional disorders of the voice must be mentioned certain cases of alteration in its pitch. Störk relates instances of children with an unnaturally bass voice, and others of young men with an excessively high pitch, after their voice has broken at puberty. The remedy, in the former case, is to practise speaking with a falsetto voice; in the latter, with a bass voice. This is often perfectly successful, if sufficient perseverance be shown. Sometimes the desired change in the pitch of the voice is brought about very rapidly; for instance, a young man, aged eighteen, who for about a year had spoken in a falsetto voice which contrasted ridiculously with his broad and well-built figure, was told to utter the vowel *u* for an hour daily in as deep a voice as possible; on the fourth day his voice became normal, and from that time it remained so.

Spasm of the tensors of the vocal cords is another curious affection, characterised by a state of the voice so peculiar as to be at once recognised by those who are familiar with it. The following is Mackenzie's description:—"The patient is often able to produce some notes, either in his own natural voice, or in a slightly muffled tone; but, while he is speaking in this way, the current of the voice seems to be partially interrupted, and the sound conveys the idea of an arrested action of the respiratory muscles. In fact, it is very much like the straining and rather suppressed voice of a person engaged in some act requiring the prolonged and steady action of the expiratory muscles (parturition or defæcation). The patients often complain that they cannot get their voice out. After speaking a word or two, or even several sentences, in this peculiar tone, the patient may utter a few words in a comparatively healthy voice, and then may immediately relapse into the diagnostic intonation." Or there may be a complete absence of sound, the lips moving in the usual way for the utterance of words and phrases, which nevertheless are lost in silence. A clergyman is described as having been greatly distressed by the fact that while he was reading the service some of the words dropped soundless from him.

Of Mackenzie's thirteen cases, eleven were in men, ten being clergymen, and the eleventh a barrister; two were in women, both of whom had been constantly speaking to deaf relatives. Doubtless, therefore, the affection is the result of over-use of the voice; but its onset is sometimes gradual, sometimes sudden. No treatment seems to be permanently successful.

LARYNGISMUS STRIDULUS.*—Spasm of the glottis is present in many diseases, and in laryngismus there may be spasm of other muscles beside those of the larynx; hence it is both less and more than spasm of the glottis, and in its extreme forms may exhibit a regular series of phenomena, comparable to those of an epileptic fit. Whether it depends on a primary disturbance of the central nervous system, or whether it is reflex and due to peripheral irritation, its proper place is certainly among the neuroses, where it is placed in the last edition of the College of Physicians' 'Nomenclature of Diseases,' and the only reason for discussing it in this place is that clinically its symptoms must be studied in relation with those of laryngeal diseases. The name of laryngismus stridulus was invented by Mason Good, and has since been very generally adopted; that of "child-crowing" was proposed by Gooch. At one time it was called "thymic asthma," in the belief that it is always caused by enlargement of the thymus. This hypothesis was maintained by Kopp in 1830, but was disproved by Bednar in 1852, and by Friedleben in 1858.

Symptoms.—The child (for the disease is one of childhood) makes a noisy inspiration again and again at varying intervals—perhaps especially on first waking from sleep. This "catching in the breath" gradually assumes a more serious character; or sometimes it begins in a severe form. The child shows signs of great distress and alarm; its neck and back are arched, its chest and abdomen rigid, its eyes turned upwards, and its limbs tonically contracted, the thumbs being bent inwards, the fingers extended, and the wrists flexed, while the legs are thrust out, the soles turned inwards, and the toes stretched wide apart. Its face, at first pale, may turn purple, or of a ghastly leaden colour. Sometimes the fæces and the urine are dis-

* *Synonyms.*—Spasmus glottidis—Spasmodic croup—Child-crowing—Thymic asthma—Millar'sches Asthma.

charged involuntarily, sometimes there is a noisy expulsion of flatus. After a few seconds, or a minute or two at the longest, the spasm yields. While it lasts the glottis is completely closed, and as it passes off a chink is formed, through which the air slowly enters, making a loud crowing sound. This usually ends the seizure, but sometimes two or more paroxysms occur in rapid succession. Frequently a few unrhythmical and noisy expirations, and one or more whistling or crowing inspirations, precede the stoppage of the breath which is the chief feature of the attack. After the paroxysm is over the child frets for a while, and falls asleep; or it may seem as well as ever and return at once to its toys.

Fatal event.—In exceptional cases, instead of relaxing, the spasm persists until life is extinct. There is then, of course, no crowing sound, and there may be nothing whatever to indicate the cause of death. In some cases death is preceded by tremulous twitchings of muscles, or by an epileptiform convulsion, as in other forms of asphyxia.

Some years ago Dr Fagge made an autopsy on the body of an infant aged sixteen months, which had died suddenly and had been brought to the hospital by its mother. Nothing was found to account for such an occurrence, but on inquiry next day it was found that the child had previously had attacks of "child-crowing."

Steffen relates the case of an infant six months old, who was one night taking the breast when it was attacked with slight spasm of the glottis, after which it went on sucking. However, the attack returned more severely, and the child fell backwards. Within a quarter of an hour Steffen was at the spot. The child had been laid in its cot under the idea that it was sleeping. He found it livid and dead, without any signs of spasm of the limbs or of any other part than the larynx.

After death from laryngismus stridulus the brain and its membranes are found gorged with blood, but this is the effect, no doubt, not the cause of the paroxysms.*

Pathology.—It is clear that other muscles beside those of the larynx are concerned in these paroxysms. The diaphragm and the chest walls are in the inspiratory position when the pause follows the first short inspirations. The spasm of the sphincter of the glottis lasts from a quarter to half a minute, sometimes rather longer. In experiments on rabbits it is found that if the superior laryngeal nerve on one side is divided, and its central end excited by a faradic current, the result is a strong bilateral adduction of the vocal cords. Probably stimulation of the respiratory centres in the bulb affects the abductors as well as the adductors, but the former are overpowered by their antagonists (cf. p. 110).

Ætiology.—Laryngismus stridulus is closely related to *rickets*—probably in nine tenths of all cases. It is doubtless in consequence of their having all in turn been sufferers from rickets that laryngismus has been sometimes noticed in several children of the same parents. Reid mentions a family of thirteen, of whom only one escaped laryngismus, and four died of it. Children affected with laryngismus are not infrequently fat; but this is quite compatible with their being highly rachitic. The close relation of laryngismus to other rachitic spasmodic neuroses, tetany and carpo-pedal contractions, has been already noticed (vol. i, p. 870).

A curious point, noticed by Hensch, is that laryngismus stridulus is of far more common occurrence in the early part of the year than later on. Dr Gee, among sixty-three cases, observed no fewer than fifty-five from

* When the disease has been of some standing the lungs may, according to Steffen, be found emphysematous; if this is really the case, as the result of inspiratory dyspnoea, it has an important bearing on the theory of emphysema in general.—C. H. F.

February to June inclusive. He believes that children being kept indoors during the winter increases the irritability of their nervous centres.

The *age* at which laryngismus stridulus begins is generally from four months to two years. But Reid relates cases in infants only a few hours after birth; and others occur in children of various ages up to nine years.

Child crowing is much more frequent in boys than in girls; the proportion seems to be about two to one. We have seen that other nervous disorders are more common in male children than in female: meningitis and hydrocephalus, infantile palsy, tetany, and carpo-pedal contractions. In fact, chorea is almost the only exception, and that affects a later age.

Laryngismus is often excited by some irritation conveyed to the nervous centres from the periphery. Marshall Hall maintained that the cause was usually to be found in disorder of the stomach, of the teeth, or of the bowels.

A few years earlier, Dr Hugh Ley had endeavoured to prove that laryngismus was the result of mechanical irritation of the vagi nerves by enlarged bronchial or cervical glands. He did show that glandular enlargement was often present; but it is as often absent, and no morbid anatomist who has learnt how frequently in autopsies upon children the vagi nerves are found surrounded by caseating glands, will assign them any important share in its ætiology.

Another hypothesis was that pressure was exerted on the recurrent nerves by an enlarged thymus or, rather, by a thymus which had not ceased to grow after birth. The term thymic asthma was applied to laryngismus by German authors, and although there is no reason to regard this as a common and important cause of spasmodic dyspnoea, yet cases like the two quoted by Osler from Koenig and Siegel ('Berlin. klin. Wochenschrift,' No. 40, 1896) show that it is not only an occasional fact, but one to be acted on in practice.

When the nervous centres are in a morbid state, with gradually increasing irritability of their cells, explosions may be brought about by slight stimuli, but there is no reason to give the process of teething more than a subordinate share in the causation of laryngismus.

Diagnosis.—Laryngismus stridulus cannot be confounded with any inflammatory or structural change in the larynx. Its paroxysmal character, with free intervals, and the absence of cough, are decisive. At the moment of a first attack, or if the child's history were unknown, one might suppose the obstruction of the larynx to be due to a foreign body, and it would be quite right to pass one's finger to the back of the throat to settle the question; but the subsidence of the spasm would show the real nature of the disease. Some of the cases of laryngismus are no doubt the result of a rare and curious malformation of the epiglottis, described at the end of this chapter (p. 141).

Prognosis.—The natural course of laryngismus stridulus, when undisturbed by treatment, varies greatly in different cases. Sometimes the attacks continue to be slight, and occur at wide intervals; sometimes they increase in severity and in number, until there may be thirty or forty in the twenty-four hours. In either case they may after a few weeks gradually become less frequent, until at length they cease entirely. If mild and severe cases be reckoned together the prognosis is favourable. It is worse when the child is very young; and worst in the silent cases, when there is no inspiratory stridor.

Treatment.—When a paroxysm is prolonged, cold water may be dashed over the face and head, while the body is immersed in a warm bath; or a bottle containing ammonia may be held to the nostrils. The nurse should be taught beforehand how to act in an emergency. The inhalation of chloroform is recommended by some writers. In severe cases, and even if life is apparently extinct, the child may be rescued by tracheotomy, followed by artificial respiration, as was pointed out many years ago by Johnson in the fifth volume of the 'Dublin Hosp. Reports.'

The treatment in the intervals is, in the first place, that of rickets—sunlight, fresh air, good food, and cod-liver oil (cf. vol. i, p. 542). A change to the country or the sea-side is often quickly followed by the subsidence of the attacks. Ringer strongly advocates sponging with cold water twice or thrice daily. Of drugs, the most trustworthy is bromide of sodium, of which from three to five grains may be given at a dose. Many find syrup of chloral hydrate (m_x—xx) an efficient medicine; it is readily taken by young children, and is perfectly safe.

It is right to look out for any condition of distant parts that may possibly be concerned in irritating the nervous centres. If the gums are hot and tense they should be lanced. If the bowels are loaded, a few aperient or vermifuge powders should be given; but it will not often be found that great results are thus attained, either in diminishing the severity or in reducing the frequency of the seizures.

Spasm of the glottis in adults.—This is an occasional complication of other diseases, mostly organic, either foreign bodies, new growths, or inflammation of the larynx itself, or affections of the nervous centres as in hysteria, in tabes, or in hydrophobia. In the last-named condition Dr Pitt had the opportunity and the skill to make an observation with the laryngoscope, and found the abductors paralysed.

INFLAMMATORY AFFECTIONS OF THE LARYNX.—Laryngitis differs widely in its seat, its symptoms, and its course. Some forms mainly affect the mucous membrane, whereas others start in the deeper structures of the larynx. Some depend on acute infection, as laryngeal diphtheria (vol. i, p. 325), and others are secondary to general diseases, as smallpox or leprosy.

Catarrhal laryngitis.—When we have separated these secondary affections, there remain the non-membranous and non-specific cases arising, not from obstruction by membranes nor from pure spasm of the glottis, but from inflammation of the mucous membrane of the larynx.

The most distinctive features of this form of "croup" are the suddenness of its onset and the severity of its symptoms. A child who is apparently in perfect health, or who may have had a slight cold for a day or two, goes to bed without any sign of distress, and falls asleep as quietly as usual. About eleven o'clock, or later in the night, he suddenly starts up in a state of extreme excitement and terror. He coughs incessantly, making a hard, hoarse, barking noise, and a loud crowing sound is heard. His face, at first flushed, afterwards becomes pale and covered with a cold sweat.

The cough, at first harsh, and clanging, gradually grows husky, and the voice, from being hoarse, becomes whispering.

Each inspiration is attended with a loud whistling sound, and a similar sibilus or a rhonchus may accompany expiration. On uncovering the child's

chest, one sees that the parts above the sternum and clavicles, and the lower intercostal spaces, are drawn in during each inspiration.

The parents are alarmed, and the nearest medical man is sent for in haste. But, instead of the child getting worse, each paroxysm of coughing is rather less severe than the preceding one; and after half an hour, or it may be in two or three hours, he becomes calm and sleeps. In the morning, when he wakes up, his cough is still hoarse and barking, but it is not so hard; his voice has nearly regained its natural tone. During the day the child is as cheerful as before; he has but little cough, his pulse is not accelerated, and he is scarcely, if at all, feverish. On the following evening the symptoms usually return, though not often so severely as at first, and may recur for several nights in succession with gradually diminished intensity.

It is doubtful whether attacks of this kind ever prove fatal; but there is little doubt that they depend on catarrhal laryngitis, complicated with spasm of the glottis.

This complaint is apt to recur again and again. A child who has once had croup is likely to be attacked a second time if he is exposed to cold or wet; and up to the age of fourteen or fifteen every slight catarrh is apt to be accompanied with the peculiar hard barking cough. When one hears that a person suffered from croup repeatedly during childhood, one may pretty safely conclude that the disease was catarrhal, not membranous laryngitis.

Ferrier, a physician of Manchester, published an essay on the subject in 1810, in which he described under the name Croup, not only the membranous disease so called by Cheyne and Home (cf. vol. i, p. 318), but also the spasmodic or spurious croup, as he called it; but after reading Ferrier's essay it seems to the writer probable that his cases of "true croup" would now be recognised as diphtheria, and that those of "spurious croup" would be called acute spasmodic laryngitis.*

Trousseau remarked that although "stridulous laryngitis," as he terms it, is very common, he had had only one case in his wards at the Hôtel Dieu, owing to the sudden way in which it declares itself, and the rapidity with which it yields. But in Guy's Hospital, during ten years, there were admitted more than ten cases of croup in which recovery took place, and in which there was no proof of the presence of membranes.

Croup, as a clinical term, corresponding to acute laryngeal catarrh with spasm, does not always begin so suddenly as was above described. The child is sometimes feverish, refusing its food, but asking for water; its voice is hoarse, and it sneezes frequently; but it complains of no pain in swallowing, and only after some hours, or even a day or two, do characteristic symptoms begin.

It might be expected that such dyspnoea would cause albuminuria; but in the series of cases recorded by Mr Lamb in the 'Guy's Hospital Reports' for 1877, there was only one in which this symptom was detected, and here there were other grounds for regarding the disease as diphtheria.

* For a valuable summary of facts and opinions on the relation of croup to diphtheria the reader interested in the history of the subject is referred to the 'Report of a Committee appointed by the Royal Medical and Chirurgical Society to investigate the relations of Membranous Croup and Diphtheria. There are numerous cases tabulated (64 by Dr Dickinson, 63 by Dr Gee, and 88 by Dr Fagge), and replies to a series of questions put by the Committee from a large number of physicians to London hospitals, as well as from others in the country and abroad (vol. lxii, 1879).

Ætiology.—The chief exciting cause of croup is said to be exposure to cold. Alison noticed that it was often produced by a child sleeping in a room newly washed, and consequently that in Edinburgh cases frequently occurred on Saturday night. Cheyne said that in all but three of the cases of croup which he saw there had been exposure to the weather.

It is stated that croup is more frequent in the winter than in the summer months, but this is doubtful. Perhaps, like acute pneumonia, it may attack those who are exposed to a chill after a hot day, or during the cold weather which in our climate may occur at any season.

The *age* at which a child is most apt to be attacked with croup is between two and seven years; the complaint is rarely seen in infants at the breast.

Boys appear to be more often affected than girls. The experience of Guy's Hospital is perhaps too limited to decide this point, but it shows decided preponderance of males in cases of non-membranous croup, as distinguished from diphtheria.

Diagnosis.—The symptoms are those of laryngeal obstruction. The distinction between croup and other diseases of the larynx is based upon the acute character of the attack and upon the age of the patient, rather than upon anything in the symptoms themselves.

Even in children a post-pharyngeal abscess sometimes gives rise to "croupy" dyspnœa and cough, although it is not obvious why this should be the case. One day when Dr Fagge was visiting the Infirmary for Children, the house surgeon reported that he had just been called to see a case of supposed croup, in which he had felt an abscess at the back of the fauces with his finger, and that relief was afforded as soon as the matter was let out. In a little child, eighteen months old, at Waltham Abbey, the respiration was rather of a snoring character than croupy, but there was a brassy cough; here the abscess seemed to have begun in suppuration of the cervical glands, which had made its way inwards. According to Steiner post-pharyngeal abscess occurs chiefly in infants at the breast, except when it follows caries of the spine, and is more insidious in onset than croup.

Jenner regarded abscess at the side of the larynx as another affection which may cause great distress in breathing by compressing the tube, and as not being always easy of recognition. Laryngeal papillomata or adenoids are too slow in their effects to be mistaken for croup, and laryngismus stridulus occurs at an earlier age; but the possible presence of a foreign body in the air-passages must never be forgotten.

Treatment.—The prognosis of acute catarrhal laryngitis with croupous symptoms is good, and it requires little treatment. It is usual to give an emetic of two to five grains of powdered ipecacuanha, with or without one sixth of a grain of tartarised antimony, repeated at intervals of ten minutes until free vomiting takes place. Jenner says that besides removing from the stomach any source of reflex irritation, and relaxing spasm by the nausea and faintness to which they give rise, these drugs also promote secretion from the laryngeal and bronchial mucous membrane. He observed that cases left to themselves last for two or three days, whereas, as soon as an emetic has acted, the child generally falls asleep at once. One must always warn the relatives of a child who has had one attack of croup that it is likely to have others if it is exposed to cold. Such children must therefore be warmly clothed; but they should be accustomed to have the neck and chest sponged every day with cold water.

Membranous laryngitis in the adult.—Although diphtheria is common to all ages, membranous laryngitis apart from diphtheria is exceedingly rare. Nevertheless, there are undoubted cases in proof that the adult larynx and trachea may become the seat of acute inflammation, which leads, not to pus and catarrhal products, but to a fibrinous exudation and a false membrane.

The most conclusive one known to the writer is the following:—A pregnant woman was admitted under his care into Guy's Hospital, in December, 1879, with severe dyspnœa from laryngitis. She was taken ill with shivering, after exposure to cold, and spat up membrane on the following day; two days later a complete cast of the trachea with its bronchial ramifications was got rid of. She died after a week's illness, having previously miscarried; and there was found laryngitis, tracheitis, and bronchitis, with lobular broncho-pneumonia and pleurisy. The pyrexia had been moderate; there was no hæmoptysis, no albuminuria, and, *post mortem*, all the other organs were sound. The fauces had been free throughout. Histologically the false membrane consisted of leucocytes and scanty fibres without blood-discs or epithelium ('Path. Trans.,' vol. xxxi, p. 30).

Subacute laryngitis with aphonia.—Some persons whenever they catch a cold become hoarse or lose their voice, and this condition lasts for several days. It is particularly apt to occur in those who are intemperate, or who habitually use the voice much, particularly in the open air. It is common in those who are exposed to smoke and irritating gases, as firemen and workers in chemical factories. It may also arise as a complication of some acute disease, as enteric fever, and especially measles. The chief symptom, beside the impairment of the voice, is expectoration of a little tough mucus, which comes away with a short hawking effort, hardly amounting to a cough. With the laryngoscope one may find reddening and slight swelling of the larynx, especially the inter-arytænoid space, or the "false cords." During attempted phonation the cords may leave an oval space between them, exactly as though the internal tensors were paralysed. In more severe cases the cords look red, dry, and lustreless, and occasionally ecchymoses or superficial excoriations may be seen.

The *prognosis* is generally favourable, but the condition, if neglected, may become chronic, and may then be exceedingly intractable.

In the *treatment* one of the most important points is that the patient should entirely abstain from using the voice. He should remain in a room of which the temperature is kept uniform. Steam inhalations should be frequently employed; and Mackenzie advised the addition of tinct. benzoin. comp. (ʒss—ʒj), or of succus conii (ʒij with gr. xx of sodæ carb. exsicc.), or of lupulin (ʒss) to the water used for inhalation, at a temperature of 140° to 160°. The patient should drink freely of demulcent liquids, such as barley water, linseed tea, and decoction of cetraria or althæa. Warm milk or *lac cum sevo* is often a useful beverage. If there is great irritability of the larynx, with cough and tickling or pricking in the throat, it should be kept in check by opium or morphia lozenges. A mustard-leaf applied over the larynx and trachea often gives relief.

The best way to overcome the liability to acute laryngeal catarrh, in those who are subject to frequent attacks of it, is to make the patient accustom himself to daily sponging with cold water, and to exposing the throat in the open air without wraps, even in the winter. No one can avoid

draughts, and the more protected he is, the more surely will he suffer when exposed. It is also important to live in dry, cool, well-ventilated rooms. Warm, light clothing should be worn. A long stay by the sea-side in the autumn, or in the bracing air of Scotland or Switzerland, often does much towards diminishing the susceptibility of catarrh in the following winter. But the most important precaution of all is to learn to breathe through the nostrils only.

Chronic laryngeal catarrh with aphonia often arises out of the acute affection, especially in persons who, in spite of hoarseness of voice, persist in using their voice out of doors. Such cases are common in clergymen, schoolmasters, auctioneers, newspaper-vendors, and costermongers. Another frequent cause is extension downwards from a granular pharyngitis. As a rule, chronic laryngitis occurs in middle life; it is more common in men than in women.

A single attack of laryngitis may determine a recurrence under certain conditions, in a person otherwise healthy. Talking in a moving train, while facing the wind, has often caused such a liability; so that for years great care had to be exercised to prevent a fresh attack. In all recurrent cases attention must be directed to the condition of the nose and nasopharynx. Any degree of obstruction which leads to breathing through the mouth, whether due to septal irregularities, or hypertrophy of the inferior turbinals, or post-nasal growths, may continue the liability.

Again, the presence of nasal polypi, so high up as not seriously to interfere with respiration, but causing abundant secretion and reflex irritation, may keep up the morbid tendency. In a word, it may be said that free nasal respiration is essential for a healthy condition of larynx. If this be established, without destroying the inferior turbinal bones, and if talking out of doors be avoided, the liability to recurrence will be in a great measure prevented.

The persistence and recurrence of hoarseness in public speakers will frequently be found to depend on some degree of nasal obstruction. The vocal cords seem to bear the entrance of cold air for some years, but when once an attack of laryngitis with hoarseness and aphonia occurs, they resent the passage of air that has not passed through the nasal passages; and if the patient begins to use his voice before the attack has quite subsided, the affection becomes chronic.

Hoarseness may be kept up by a comparatively small degree of obstruction, such as slight lateral deviation or enchondrosis of the septum, hypertrophy of the inferior turbinal, or unobserved polypi.

The air is warmed, moistened, and sifted of dust as it passes through the vascular, secreting, and narrow passages from the nostril to the pharynx.*

The principal *symptom* of this affection is hoarseness of voice, which may pass on to complete aphonia. The degree to which the voice is impaired may vary much at different periods of the day. It is often greater when the patient first begins to speak than after he has been speaking for a time. The attempt to use the vocal cords gives rise to a painful sense

* The warming of the air as it passes through the nasal passages was actually measured by Dr Greville Macdonald with an ingenious apparatus contrived for the purpose. He found that air from a freezing chamber at 7° C. was raised to 28° C., air at either 12° C. or 20° C. was alike raised to 35° C., while air inspired at 45° C. was cooled in its passage through the nose down to 33·6° C.

of fatigue, with a feeling of dryness or tickling, and a constant desire to hawk or to cough.

The laryngoscope shows all gradations of change, from a slight local injection and swelling, up to the most diffused redness of the whole interior of the larynx. Mackenzie remarked that one vocal cord may be bright red while the other is white, or the congestion may be limited to the outer or attached side of the cord. Flakes of mucus are often seen adhering to the mucous membrane, or threads of mucus may cross from one cord to the other. During vocalisation the cords in many cases fail to meet, and this may be due to swelling of the interarytænoid mucous membrane, which is sometimes so great as to form a convex projection; but paresis of the muscles also is often present, and if this is unilateral, the opposite cord may pass across the middle line to meet the affected one.

In chronic cases the mucosa of the larynx is greatly thickened. In a case recorded by Störk, of fifteen years' standing, the whole of the interior of the larynx was hypertrophied, so that there was great dyspnoea, rendering tracheotomy inevitable. Every part of the interior of the larynx was thrown into thick folds and ridges. This condition is known as *Pachydermia laryngis*, and resembles that of chronic hypertrophic dermatitis. The same affection has sometimes been observed below the glottis, reducing the channel of the trachea to a narrow ring, so that it has been necessary to open the trachea. The vocal cords themselves sometimes become granular on the surface, a condition which Türck designated "*chorditis tuberosa*."

Enlargement of the mucous glands is not infrequent in chronic laryngeal catarrh. Mackenzie describes their enlarged orifices on the epiglottis and the posterior parts of the cords as pale specks on a congested surface, or as small red circles on a pale surface. Another complication is dilatation of the veins of the mucous membrane, especially on the epiglottis or the cords.

Those most qualified to judge seem to be now agreed that catarrhal laryngitis does not lead to ulceration. Laryngeal hæmorrhage in sufficient quantity to suggest phthisis certainly points to a tuberculous ulcer.

Störk speaks of a vertical fissure in the interarytænoid mucous membrane as being extremely frequent, though not peculiar to cases of chronic catarrh. The fissure, as the result of lateral traction, assumes a rhombic form; so that the upper part, which is alone visible in the laryngeal mirror, appears triangular. Its detection is often very difficult; the patient must be placed in the position required for inspection of the trachea with the laryngeal mirror. Störk knew a singer of reputation, whose voice remained perfect after such a fissure had existed for many years; but, as a rule, there are symptoms like those of chronic laryngeal catarrh; and sometimes the subjacent arytænoideus muscle is so enfeebled that the patient is distressed by mucus running down into the larynx whenever he attempts to swallow.

The course of chronic laryngitis is generally very tedious and protracted; for patients will seldom carry out treatment with perseverance, imagining that they ought to be well in two or three weeks, and neglecting precautions as soon as they begin to improve. Another reason was that until lately the condition of the naso-pharynx was not systematically investigated.

In all cases of laryngeal catarrh with aphonia, the first thing is to ascertain whether the patient can breathe comfortably with his mouth shut. If not, there is sure to be some obstruction in the nasal passages or the

pharynx. In children the narrow jaw, crowded teeth, and high-arched palate, with, in some cases, an ill-developed or rickety chest and deafness, lead one at once to suspect enlarged tonsils or adenoid growths. In older patients asymmetry of the nasal septum, affections of the turbinated bone, varicosity of the mucous membrane, or the presence of a small polypus, will produce the same effect. The nostrils and naso-pharynx must therefore be examined, and after local obstructions have been removed by the surgeon, in many cases the cure is complete.

When there is no such permanent obstruction, the patient may breathe through the mouth owing to the passage through the nose being impeded by temporary swelling of the mucous membrane, and secretion of mucus which accompany a "cold in the head." In many cases a constantly recurrent catarrh during the winter leads to fresh sore throat, and so to increased post-nasal obstruction; but even then some overgrowth of the tonsils is often the starting-point of the vicious circle.

When there is no obstruction to breathing through the nose, or when such obstruction is removed, if there still remain its effects on the throat and voice, they will disappear entirely under careful management. Necessary rest must be given to the voice, and the patient must speak as little as possible, or only in a whisper. He must abstain from smoking, and he must take little or no alcohol.

Locally, if the larynx is irritable, the patient should use a spray containing bromide of potassium or carbonate of soda with a little morphia, or inhale steam impregnated with turpentine, terebene, or eucalyptol. In more chronic and obstinate cases the most effectual treatment is the systematic application of astringent solutions to the interior of the larynx by means of a brush. Mackenzie used to employ a solution of chloride of zinc (gr. xv ad ʒj), applied daily during the first week, on alternate days during the second and third weeks, and afterwards less frequently.

In more chronic and severe cases, one of the oldest applications is still found the most effectual, namely, silver nitrate. It is best applied with a sponge on a curved laryngeal probang in aqueous solution, gradually increased in strength from gr. xv to the ounce, up to ʒj or even ʒiiss in the ounce. When a decided effect is produced, the strength should be again diminished.*

In many cases of chronic catarrh, interrupted galvanism applied locally aids in restoring the voice; and some patients believe they find benefit from drinking the waters at Aix-les-Bains or at Ems.

In obstinate cases, with much thickening of the mucous membrane, particularly when the condition described by Virchow in 1877 as *pachydermia laryngis* is reached, lactic acid is now frequently applied in 25 to 50 per cent. solution with great benefit.

TUBERCULOUS DISEASE OF THE LARYNX AND TRACHEA.—Towards the end of the eighteenth century we first hear of *laryngeal phthisis*. This is not a good name, because it implies that an affection of the larynx may give rise to wasting and other symptoms of pulmonary phthisis, without there

* Ziemssen recommended the topical use of solid nitrate of silver fused on the end of a laryngeal probe, and repeated at intervals of a week or a fortnight. It causes violent spasm, which, however, is at once relieved by filling the throat with cold water. This severe treatment he found particularly efficacious when there is an interarytænoid fissure; but he warns that if the healthy mucous membrane is touched, the patient's condition will be aggravated instead of relieved.

being any lesion of the lungs. The opinion that such is sometimes the case was, indeed, held by Trousseau, and by some other writers since, and it often seems to be clinically supported; but the experience of autopsies is that when tuberculous ulceration of the larynx is found after death the lungs contain tubercles also; and if laryngeal tubercle is seen in cases of acute general tuberculosis, old as well as recent disease of the lungs is also present. If it ever occurs, primary tuberculous disease of the larynx is so rare that we need not consider it in practice.

Pathology.—That the laryngeal affection which accompanies phthisis is itself tuberculous, was taught by Laennec, and although disputed by Louis, the general belief of English pathologists has always been that the laryngeal ulceration is really tuberculous, not merely because it occurs in cases of phthisis, and along with tuberculous ulceration of the intestine, but also because many laryngeal ulcers have caseating edges. Heinze, of Leipzig, published a monograph on the disease in 1879, based upon microscopical examination in fifty cases of phthisis, complicated with disease of the larynx or trachea. He found that in forty among his fifty cases, tubercles were plainly recognisable in the larynx; in thirty-nine of these forty there was ulceration, and in one tuberculous infiltration without ulceration; in eleven of the forty there was also tuberculous ulceration in the trachea; and in eight the trachea contained ulcers not so clearly tuberculous. Of the remaining ten cases, in eight there were laryngeal ulcers not certainly tuberculous, but in five of these the trachea showed tuberculous ulcers, and in two there were no lesions in the larynx, but tuberculous ulcers in the trachea. In other words, there were only three out of the fifty cases in which tubercles were not detected either in the larynx or in the trachea, or in both. The tubercles were plainly visible to the naked eye in hardened sections. They lay partly in the mucous membrane, partly in the sub-mucous tissue, but always superficial to the laryngeal mucous glands. They had often undergone more or less complete caseation.*

Laryngeal tubercles, as a rule, are very small; but tubercles, both grey and caseating, have frequently been seen by the naked eye. Koch's tubercle-bacilli are sometimes found in the granulations of a laryngeal ulcer, but not abundantly; and even when the secretion covering it contains them, they may be pulmonary in origin.

The fact that tuberculosis of the larynx scarcely ever occurs independently of phthisis, and that probably it never precedes the latter, naturally suggests that the upper air-passages become infected by the passage of tuberculous sputum.†

Sex and age.—Tuberculous disease of the larynx and trachea is more frequent in men than in women, and the preponderance is far greater than is the case with pulmonary phthisis. According to Heinze, 33·6 per cent. of male phthisical patients have ulceration of the larynx, but only 21·6 per cent. of female phthisical patients.

The age at which tuberculous laryngeal affections are absolutely most

* With regard to the non-tubercular ulcers of the larynx and trachea, Heinze saw nothing to the naked eye that distinguished them from those that were tubercular. It is, therefore, quite possible that they were the result of tubercles which had softened and been cast off by ulceration. In the trachea, on the other hand, there seems to be no doubt that ulcers occur which are really non-tubercular; they appear as minute depressions of yellow colour surrounding the mouths of the tracheal glands.—C. H. F.

† Ziemssen once traced a continuous tract of ulceration from a vomica in the upper lobe of one lung along the corresponding bronchial tube, through the trachea to the larynx.

frequent is between twenty-one and thirty ; but among fatal cases of phthisis the proportion in which the larynx is found diseased is largest at a later age,—between forty-one and fifty for males, between thirty-one and forty for females. During childhood tuberculous disease of the larynx is not common ; among nearly 400 cases Heinze found only nine in patients under the age of fourteen, but one of these was an infant of eleven months.

Symptoms.—Tuberculous ulcers are often found in cases of phthisis, when during life no decided symptoms have pointed to the larynx ; but sometimes there is much dysphagia, every attempt to swallow causing pain and distress. The effort to speak is often painful, the voice becomes hoarse and weak, and ultimately is reduced to a whisper ; while the cough becomes weak and toneless, as well as the voice. In some cases, however, in which the true cords are entirely destroyed by ulceration, it is believed that the false cords may vibrate so as to produce harsh and deep tones.

Pain, cough, and aphonia are, however, common to other forms of common laryngitis, and experience in the deadhouse teaches that extensive superficial catarrh of the mucous membrane causes more symptoms than does the presence of small, deep, tuberculous ulcers.

Diagnosis.—The first character shown in the laryngeal mirror is an extremely *anæmic* state of the mucous membrane of the larynx.

In twenty-one of Heinze's forty cases, there was infiltration of the false cords and aryteno-epiglottidean folds, causing a smooth tense swelling, of greyish colour, often presenting pale yellow granules, or spotted with points of hæmorrhage. Over the tips of the arytenoid cartilages are swellings which Heinze compares to two rounded sugar-loaves, and Mackenzie describes as pyriform. If the epiglottis is affected, it becomes rounded and thickened. The false cords are much swollen, while the entrance into the ventricle of Morgagni is narrowed. Thickening of the interarytenoid mucous membrane causes bulging at the back of the glottis, which, as Störk stated, is characteristic of tuberculous disease. The vocal cords very rarely exhibit this tuberculous infiltration, and in the trachea it only occurs in the posterior membranous part.

Tuberculous *ulcers* are usually situated at the attachment of the vocal cords to the arytenoid cartilages. They are rarely deep or hæmorrhagic.

Among Heinze's fifty cases, ulceration of the cords was present in forty ; on both sides in twenty-seven, on one or the other side separately in ten, at the anterior commissure in three. In the laryngeal mirror an ulcer on one of the cords may appear either as a narrow linear fissure, or as an excavation of its edge.

The false cords are not so often ulcerated ; only in fourteen of Heinze's fifty cases, but in eight of these on both sides.

Over the arytenoid cartilages ulcers were present in twenty-three of the fifty cases, sometimes towards the bases of the cartilages, sometimes on their summits or towards the pharyngeal surface. Those which lie towards the bases of the cartilages are almost always bilateral. They penetrate deeply into the tissues, so that they often reach the perichondrium and lead to necrosis of the cartilages. They are seldom, if ever, visible in the laryngeal mirror, but in the dead body their outline is seen to be irregular, their edges smooth or fringed with papillary outgrowths, their surface uneven and dirty grey.

The epiglottis was ulcerated in twenty-six of Heinze's cases, generally upon its laryngeal surface, sometimes upon its border, never on its lingual

surface. Often there are many small round shallow ulcers, which sometimes run together. In severe cases the epiglottis is eaten away from its margin inwards, so as to cause deep fissures; or it may be reduced to a mere stump.

The detection of ulcers in the laryngeal mirror is not always easy, even when they come fully into view. They are often recognised only by a defective lustre of the mucous membrane, and unskilled observers may mistake patches of puriform mucus for ulcers.

Non-tuberculous ulcers.—Apart from either tubercle or syphilis, ulcers over the arytaenoid cartilages are not infrequent in pneumonia; also in cases of double pleurisy with pericarditis, probably pneumococcal in origin, in cases of erysipelas, and in cases of Bright's disease. The most common of secondary laryngeal ulcers, however, are those found in cases of Enteric fever.

Prognosis and treatment.—Dr Fagge noted three cases of phthisis in which the larynx after death presented cicatrices of ulcers which had healed; Ziemssen and Heinze recorded similar instances. At the International Congress of London, in 1881, Rossbach and some other observers spoke positively of the curability of laryngeal phthisis, although Krishaber doubted it. Certainly, very few patients with tuberculous disease of the larynx recover; and laryngeal symptoms have long been recognised as of ill omen in the outlook of phthisis.

For the relief of the distressing symptoms we may use the local *insufflation of morphia* as a powder, in a dose of $\frac{1}{8}$ — $\frac{1}{2}$ a grain mixed with half a grain of starch.* Mackenzie found that the sensitiveness of the affected parts is least in rather less than an hour, and therefore when there was much pain in swallowing, introduced the morphia at about that interval before a meal. It may be repeated twice daily.

Cocaine lozenges, taken before food, are of the utmost value in these cases, and proved so particularly in a severe one lately under the writer's care, when all other means failed. If distress is caused by liquids entering the air-passages, all fluid nourishment should be thickened with arrowroot or cornflour; and the patient is more likely to swallow well in taking off a good draught than in sipping. Sometimes it is necessary to administer food through an œsophageal tube for weeks together. Dr Wolfenden recommends the patient, in extreme cases, to drink only when lying in the prone position, or supporting himself on his hands and knees. Tracheotomy is now and then required when there is extreme dyspnoea; but this operation seems in no way to retard the further progress of the laryngeal affection.

If we attempt more than palliative treatment of laryngeal phthisis, the application most useful appears to be a 50 per cent. solution of lactic acid. This treatment was introduced by Dr Heryng, of Warsaw, and has been adopted by Krause and other specialists in Germany and England. Mr Symonds reports well of its results, especially if the lactic acid is applied

* A special instrument called an "insufflator" is made for the purpose: it consists of a hollow vulcanite tube, one end of which is bent downwards so that it can be directed towards the orifice of the larynx; in the middle of the tube is a hole, through which the powder is introduced, and which is then closed by a moveable covering; the other end of the tube is connected with a piece of elastic tubing. In using this instrument the operator places the elastic tubing in his mouth, and introduces the vulcanite tube into the mouth of the patient. He then blows the powder into the larynx, just when the patient is drawing his breath.

after the tuberculous ulcer has been scraped with a curette. He finds it most useful when the disease is most localised.*

The duration of life in cases of tuberculous disease of the larynx is seldom long, because it occurs in an advanced stage of a dangerous disease; but it does not often directly shorten the patient's life, and if the pulmonary disease assumes a chronic form, laryngeal ulcers may exist for years.

LUPUS OF THE LARYNX.—Within the last few years several cases have been recorded, in which lupus of the skin has been associated with a like disease of the epiglottis and the larynx. Mr Symonds believes that secondary laryngeal lupus occurs in from 3 to 5 per cent. of cases of cutaneous lupus. Primary lupus of the larynx is very rare.

The lesions visible are nodules—some like pin-heads, some as large as peas—and ulcers, with probably some cicatrisation. The ulcerated epiglottis often looks as though a heart-shaped piece were taken out of its edge. Otherwise there is little to distinguish lupus of the larynx from syphilis; nor are the symptoms peculiar:—hoarseness, sore throat, dysphagia, and perhaps dyspnoea; while pain is remarkably absent. The patients are usually girls.

The diagnostic characters are the youth of the patient, the slightness of the symptoms, and the presence of cicatrices; negatively, the absence of signs of lues or tuberculosis elsewhere. Pharyngeal lupus is a frequent complication, and also lupus of the fauces or palate. The best treatment seems to be the systematic administration of cod-liver oil, and cauterisation with nitrate of silver. Scraping with a curette and the electric cautery have also been employed with success. The most serious result is sclerosis, leading to stenosis of the larynx.

A remarkable case, which was regarded by the writer as lupus of the larynx, occurred in a young man who was long under his care in Philip Ward in 1887. There was no lupus of the skin, and no signs or history of syphilis, but the epiglottis was seen to be ulcerated, and there was no appearance of tuberculous ulceration of the cords, nor of phthisis. He was therefore put under iodide of potassium and mercurial inunction, but with no good result. Emaciation gradually came on, and he died after about a year's illness. Deglutition had been only possible for some time by the local use of cocaine, and the immediate cause of death was gangrenous pneumonia from particles of food gaining entrance to the air-passages. There was no proof of syphilis discovered after death, and only a trace of obsolete phthisis at the apex of one lung. The epiglottis was entirely destroyed; the cords were much less affected. The appearance of the larynx was quite unlike that of tuberculous disease. The essential nature of the two conditions is identical; but the same remarkable difference between the pathological and clinical characters is observed in lupus and tuberculous ulcers of the skin.

LEPROSY, as well as lupus, may affect the larynx, and in a far greater proportion of cases. The disease usually begins in the soft palate, and then spreads to the epiglottis. There is as great anæmia of the fauces and larynx as in cases of tuberculosis, and the swollen parts look (as Mackenzie put it)

* Dr Clifford Beale has written with judicious reservations, but decidedly in its favour, in an excellent paper on "Laryngeal Affections in Phthisical Persons" ('Birmingham Medical Review,' April, 1890).

as if infiltrated with tallow. Characteristic leprous nodules next appear, and perhaps after several years ulceration follows; the voice, which before was characteristically hoarse, harsh, and nasal, is now completely lost, and foetor of the breath, hæmorrhage, and extensive destruction of tissues follow. The anæsthetic form of *Lepra* very seldom affects the larynx, and only late in its course. The most striking peculiarity is the absence of pain or even irritation, and more marked anæsthesia than in syphilis, in complete contrast with the sensitiveness of tuberculosis and with the severe pains of cancer. (Guy's Hospital Museum, 58, 59, 60.)

LARYNGEAL SYPHILIS.—Syphilis of the larynx is by no means rare, whether compared with other effects of lues, or with other diseases of the larynx; its occurrence seems to some extent to be determined by local irritation. Most patients are, as might be expected, between twenty and forty; but late symptoms are met with in patients much older. The earlier laryngeal manifestations of acquired syphilis may occur within two or three months after infection; its more remote effects may appear five, ten, twenty, or even thirty years afterwards, when all other indications of the disease have long disappeared, and when the patient has almost forgotten it.

In some cases, usually recent, the larynx shows mere catarrh, with nothing characteristic of its origin. Neither the colour, nor the protracted duration, nor the recurrence of a laryngeal catarrh are evidence of lues; and Störk speaks of syphilitic catarrh of the larynx as so transitory that patients are seldom treated for it.

More characteristic are *mucous patches*, or “flat condylomata” (vol. i, p. 343). Their chief seats are the vocal cords, the posterior laryngeal wall, the false cords, and the epiglottis. According to Mackenzie they differ from pharyngeal condylomata in being yellow rather than white, and in being accompanied by less marked congestion. They seldom ulcerate, and often disappear quickly, even without treatment.

At a later period *gummata* not infrequently occur in the laryngeal mucous membrane and submucous tissue. They form small rounded nodules, from the size of a pin's head to that of a small pea, of the same colour with the rest of the laryngeal surface, isolated or collected into larger masses. They may be seated upon the epiglottis, the posterior wall of the larynx, the false cords, or the surface below the glottis. In 1874 Mr Norton showed the Pathological Society a gumma larger than a pigeon's egg, which occupied the right arytaeno-epiglottidean fold, and reduced the air-passage to a mere chink.

Syphilitic *ulcers* in the larynx are often observed. During the earlier stages of the disease they are generally superficial; afterwards they are gummatous, and eat deeply into the tissues. They are usually “punched out,” and coated with a whitish-yellow material. As Störk remarks, a patient with extensive destruction of the epiglottis from syphilis may still retain a fresh, healthy appearance, which is never the case when the condition is tuberculous. Tuberculous ulcers are smaller than syphilitic, except when several have coalesced; they are often numerous, whereas syphilitic ulcers are usually solitary; and the surface around is pale and anæmic instead of being red.

Cicatrices frequently form within the larynx, and produce remarkable deformity. Sometimes a web is formed between the cords, as in several cases recorded by Elsberg, of New York. In other cases the epiglottis is

dragged down and fixed to the side of the pharynx; or the parts forming the entrance of the larynx may be puckered together, so as to reduce it to a small round hole. Papillary growths of considerable size are occasionally developed in the neighbourhood of syphilitic cicatrices, and increase the obstruction to the passage of air.

Symptoms.—Syphilitic disease of the larynx produces hoarseness and aphonia. Pain is often entirely absent; but if the epiglottis is affected, swallowing may be exquisitely painful, although even then there may be freedom from pain at other times. It is wonderful how well some patients manage to swallow, even when they have lost a large part of the epiglottis; the base of the tongue is carried backwards, and keeps even fluid from passing into the larynx. Cough is often troublesome, and if there is deep ulceration, blood-stained mucus or pus may be expectorated. In one case at Guy's Hospital hæmoptysis occurred so freely that the patient was thought to have phthisis; and Türk met with an instance of fatal hæmorrhage from an extensive syphilitic ulcer of the larynx, exposing the cricoid cartilage. This is very rare in cases of tuberculous ulcers of the larynx.

Diagnosis.—The course of tubercle is painful and ingravescens; that of syphilis is painless, but rapid and destructive. In the tertiary stage the lesion most frequently seen is ulceration, and particularly a solitary ulcer, situated in the aryæno-epiglottic fold or the ventricular borders. It is well-defined and deep, with grey or yellowish surface. Particularly noticeable is the absence of the œdematous infiltration of tubercle, the solitary character, the little pain, and the free movement of the cords. In tubercle it is exceptional to find a solitary large lesion; there are usually other ulcers, or at least infiltration, much pain, and dysphagia.

The course of lupus is slow; a case recorded by McBride was in good health six years after being first seen. The ulceration of syphilis is usually more localised than that of lupus, more rapid, and exhibits a deep ulcer, with much attendant œdema. It gives rise to dysphagia, and occurs in older subjects. When the epiglottis has been partially destroyed by syphilis, the truncated remnant, though nodular, is yet smooth as a whole, and does not exhibit the thick, hard margin seen in lupus.

Tuberculous ulcers do not readily cicatrise, and are attended with great distress; there is œdema and much secretion, while in lupus there is little or none.

In malignant disease, the ulcer is not so well seen owing to surrounding infiltration, and there is greater fixation of the cord.

The ulceration of leprosy is never seen in the larynx unless there are also signs of the disease in the skin, hence the diagnosis is easy. There is less pain and reflex irritability than even in syphilis.

In severe syphilitic disease there is wide infiltration of the submucosa. The laryngoscope shows irregular œdematous masses of mucous membrane: a view of the cords is impossible, and there is much secretion. In this stage there is a close resemblance to malignant disease, or the severer form of tubercle. Help will be obtained from the history, which will be longer than in cases of cancer; with periods of comparative recovery, followed by relapses. There is not the same foul odour as in necrosing growth, nor the same emaciation or distress.

It is in these severe cases that necrosis of the cartilages occurs: or a gummatous mass may project externally through the ala of the thyroid cartilage, and cause a fluctuating swelling. As a result of this destructive

process, extensive cicatrisation follows, with fixation of one or both cords, and the epiglottis may be reduced to a mere stump.

The *treatment* of syphilitic disease of the larynx consists mainly in the administration of mercury; but sometimes it is advisable to brush over the affected parts with dilute tincture of iodine, or with a mixture of two parts of iodine, two of iodide of potassium, and ten of glycerine. When mercury has already been used, full doses of iodide of potassium may be prescribed.

The local application of calomel in powder is of great service; or the inhalation of calomel vapour. Absolute rest of voice should be enjoined, and, if possible, confinement in bed for a time. In the case of a professional singer seen for hoarseness and loss of voice, and in whom extensive syphilitic infiltration was observed, these methods resulted in complete restoration of the singing voice.

When the stenosis from swelling is great, and dyspnoea is becoming dangerous; or, again, when the disease is resisting treatment, tracheotomy should be performed. The immediate improvement which follows this operation, is the best justification for its employment. As much advance has been noted in the first three weeks after operation, as in the previous three months. The operation, deliberately performed, is free from risk, and may be performed under cocaine, or the ethyl chloride spray, should any reason prevent the use of chloroform.

After successful treatment of the ulceration and arrest of the syphilitic process, the cicatrisation may cause such a degree of stenosis as to require the permanent use of a tracheal cannula. Fitted with a pea-valve, and concealed by the collar, the wearer can discharge all the ordinary duties of life, and even undertake public speaking. No risk attends the permanent use of the tube, even for years; in one instance a patient of Mr Symonds has worn a tube thirty years.*

Congenital syphilitic laryngitis is not uncommon, but is usually of only symptomatic importance, by causing aphonia. Cases, however, have occurred of deep and severe ulceration leading to death. Two, in brothers aged between six and three, were recorded by Semon in the 'Pathological Transactions' for 1880, and a third, in a girl eleven months old, by Barlow, in the same volume.

Fränkel recorded an instance in which a syphilitic infant, less than three months old, died from laryngeal stenosis, as the result of perichondritis with exfoliation of the cricoid and of the left arytenoid cartilages.

CEDEMATOUS OR PHLEGMONOUS LARYNGITIS.—This is the condition commonly but inaccurately termed *œdema glottidis*, the name originally given it by Bayle. The part affected is not the glottis, for (except in very rare instances) the vocal cords are free from swelling; the seat of the obstruction is in the entrance of the larynx above. Moreover the condition is not a passive œdema, but inflammatory exudation of serum loaded with leucocytes into the submucous tissue, and ends in diffuse purulent infiltration, or the formation of an abscess. In some cases the term *œdematous laryn-*

* Schröter introduced a successful treatment of such cases by mechanical dilatation, at first with vulcanite tubes, and afterwards with pewter plugs, about an inch and a quarter in length, which can be left in the larynx for several hours at a time. Having been introduced through the mouth, the plug is held *in situ* by being bolted into the convex surface of the cannula which the patient is wearing.

gitis, in others phlegmonous laryngitis, is more applicable. Dr Fagge proposed as a better name than either "submucous laryngitis."*

Passive oedema of the larynx is no doubt often seen after death from Bright's disease, or heart disease, when the epiglottidean folds form watery swellings, often of considerable size; but such cases do not appear to present special symptoms during life.

On the other hand, true oedematous or submucous laryngitis is one of the most rapidly fatal of diseases. When the epiglottis is involved, it forms a turgid round mass, or two lateral halves pressed closely together, so as to leave only a narrow gap between them. This may be felt by the finger passed into the fauces, or may be seen in the laryngeal mirror; occasionally it is directly visible when the tongue is depressed with a good spatula. When the parts most affected are the epiglottidean folds, they form two globular swellings, as tense and resisting as swollen tonsils. The mucous membrane covering the cornicula laryngis and between the arytaenoid cartilages may share in the swelling, so that the movements of the cartilages are much impeded. Within the larynx the inflammation usually affects the false cords, which bulge downwards as well as inwards, so as to overhang and conceal the true cords. The latter are very seldom involved in the swelling, but Risch once recorded a case in which, having actually removed the larynx within ten minutes of the patient's death, he found the true cords swollen to the breadth of half a centimetre and pressed against one another, so as completely to close the glottis. A similar instance occurred at Guy's Hospital in 1873; the patient was a woman who was found moaning on the ground in the street, and who died before she reached the ward.

Lastly, the exudation may in some cases be limited to the parts below the cords. Mackenzie speaks of such "subglottic oedema," as generally chronic rather than acute; but Ziemssen cites five instances, one of them observed by himself, and all verified by the laryngoscope, in which the symptoms were urgent and rapidly developed.

The colour of the affected parts, as seen during life, is generally a bright red. After death they look much paler, the arytaeno-epiglottidean folds in particular appearing gelatinous, often with a yellowish-green tint from infiltration of pus. When, however, they are incised after death, it is often found that no fluid escapes from them, even under pressure. Sometimes the inflammation extends to the laryngeal muscles, and after death they are found full of suppurating points.

Among the *symptoms* of submucous laryngitis the most important is dyspnoea, which may increase with extraordinary rapidity until it destroys life by suffocation. Inspiration is attended with a loud whistling sound, there is some pain in the throat, increased by speaking, and the larynx is tender when handled in the neck. The voice is not always much altered, but as a rule it becomes first hoarse and then extinct, while the cough is toneless. If the epiglottis is inflamed, there is intense pain in swallowing, and in all cases much distress is occasioned by the accumulation of mucus and saliva in the mouth and pharynx.

Dysphagia is sometimes the first indication of oedematous laryngitis; hence the importance of using the laryngoscope whenever there is difficulty of swallowing. Often, however, the swelling can be felt by the finger.

* A typical case was described by Drs Carrington and Hale White in 1885 ('Clin. Trans.,' vol. xviii, p. 1885). In 1888 it was described as a new disease by Senator under the title acute infectious phlegmon of the larynx. Kuttner adopted Fagge's title submucous laryngitis, in a monograph published in 1895.

Ætiology.—Acute submucous laryngitis occurs chiefly in young adults between the ages of eighteen and thirty-five, and in men more often than in women. It is rarely seen in children.

As a primary affection, it appears to be generally due to some septic cause. Sometimes it follows exposure to cold, as in Trousseau's case of a man who, having drunk too freely at a wine-shop, was turned out into the street on a cold night, and fell asleep there, to wake with a violent sore-throat, which in an hour or two produced the most extreme dyspnœa. It arises by direct extension from faucial erysipelas, as in the case at Avignon referred to above (vol. i, p. 312); and sometimes it occurs in the course of smallpox, or of enteric fever. It is frequently the immediate cause of death in *angina Ludovici*,* a diffuse inflammation of the connective tissue of the neck, with brawny infiltration, which, as a rule, ends in suppuration.

In a case at Guy's Hospital in 1863, it was secondary to chronic suppuration in the fibrous tissues about the hyoid bone. It is often developed by extension from perichondritis of the laryngeal cartilages, or from other suppurative foci. In all cases it is septic in origin, acute and erysipelatous in clinical characters; but there does not appear to be any constant suppurating microbe. The *streptococcus* of erysipelas, the *Streptococcus pyogenes* (if it is not identical), and the *Staphylococcus pyogenes aureus*, have each been present in cases apparently similar.

Distinct from this septic laryngitis must be regarded *traumatic* cases of acute laryngitis from scalds, corrosive poisons, or impaction of foreign bodies. Distinct also is the mere passive œdema of the larynx in cases of general dropsy. But in cases of Bright's disease the condition is frequently not mere œdema, but exudation of a shreddy lymph-like material in the mucous membrane of the larynx, either above or below the cords.

A case at Guy's Hospital of acute inflammation of the submucous tissue of the larynx occurred in a man, aged twenty-seven, who died in 1878 of epileptiform convulsions. He had complained of sore-throat and of shortness of breath, but no symptoms had been observed indicative of laryngeal mischief. Both epiglottidean folds were found infiltrated with pus, but especially the left one. There was also much exudation of puriform lymph round the pharynx and at the base of the tongue.

In the following year a man, aged thirty-four, who was lying in the hospital with cirrhosis of the liver, was attacked one day, at 11 a.m., with sore-throat, followed at 4 p.m. by severe laryngeal symptoms, and by rapidly fatal dyspnœa at 10 p.m. After death the left epiglottidean fold was found moderately swollen with an effusion of serum, the right one much swollen, smooth, and shining, and infiltrated with thick purulent material.

Ziemssen relates the case of a young man who was attacked with extreme distress of breathing after eating some bread, and who ran off to the surgeon, thinking there must have been a needle in it, and that it was sticking in his throat. The laryngoscope showed that the right sinus pyriformis contained a pointed splinter of wood, which was at once removed with a pair of forceps. Only a quarter of an hour altogether had passed, yet there was considerable œdema of the right epiglottidean fold.

In cases of acute laryngitis from hot water, poison or corrosives, suffocative symptoms, after setting in suddenly, so that tracheotomy appears imminent, may sometimes subside with no less rapidity.

Treatment.—It is often well, at the commencement, to apply leeches to the neck on each side of the larynx. The patient should constantly suck

* There does not seem any reason to regard this condition (named after Ludwig in 1836) as a primary lesion. It is the result of many local or general causes. See Semon's paper in the 'Med.-Chir. Trans.' for 1895.

ice; and if the entrance of the larynx is found to be already swollen, the tissues should at once be scarified, so as to allow the exudation to escape. Numerous shallow parallel incisions should be made, with a "laryngeal lancet," or small double-edged knife mounted on a curved stem; but in an emergency an ordinary bistoury, covered up with adhesive plaster to within a quarter of an inch of its extremity, answers very well. If relief does not speedily follow, tracheotomy must be performed. Indeed the patient must not be left, even for a few minutes, until an opening into the trachea has been made, for a paroxysm of dyspnœa may set in at any moment, and may end fatally before there is time to fetch a surgeon.

Internally alcohol, quinine in full doses, or tincture of steel are indicated.

LARYNGEAL PERICHONDritis.—Several writers in the latter part of the eighteenth century recorded cases of suppurative laryngitis, with destruction of one or more of the laryngeal cartilages, and supposed that the disease began in the cartilages. But it is now generally admitted that the starting-point is in the perichondrium. Indeed, as far back as 1850, Dittrich showed in the '*Prager Vierteljahrschrift*,' that a small portion of the cricoid, lying in an abscess cavity, was converted into a dirty yellow, calcified mass, while the rest of it and the other laryngeal cartilages were healthy. Frequently disease of the laryngeal cartilages and perichondrium is secondary to ulceration of the mucous membrane, particularly in the course of phthisis, enteric fever, smallpox, or syphilis.

Dr Fagge met with a case in Guy's Hospital in 1859, where the right ala of the thyroid and the right half of the cricoid were alike necrosed, whereas the left halves of their cartilages had escaped.

In the case of a child, eighteen months old, who died with "croupy" symptoms, he once found that part of the left half of the cricoid, which was bathed in pus, had undergone absorption, so that there was a gap in it, with thin smooth edges.

A year previously, in 1874, in the body of a man aged thirty-three, the back part of the cricoid was necrosed, lying loose in an abscess cavity, while its anterior part was represented by a narrow edge of healthy cartilage, thinning off into fibrous tissue.

Dittrich suggested that in bedridden patients, necrosis of the cricoid may be an indirect result of its pressure against the vertebral column. He supposed that the pressure first caused ulceration and sloughing in the two opposed surfaces of the pharynx, which then spread from the anterior pharyngeal wall to the perichondrium. The process would thus resemble that of ordinary bedsores; and Dittrich gave one case in which the patient, a phthisical man, aged thirty-one, also suffered from bedsores. He also recorded in detail two out of several cases in which, in bedridden patients, he found that both surfaces of the pharynx showed local patches of ulceration, without the cricoid cartilage having as yet become involved. Störk satisfied himself that in severe enteric fever necrosis of the thyroid cartilage may arise in a similar way from pressure against the spinal column; and Ziemssen says that in old people, in whom the cricoid is ossified, perichondritis (or periostitis) may follow the repeated introduction of œsophageal bougies.

Laryngeal perichondritis is much more common in males than in females, and appears to occur most often between twenty and thirty years of age—the period of tuberculosis and of enteric fever. As a primary affection, however, it has been more frequent from thirty to fifty at Guy's Hospital. One patient was a girl under two years old, one a boy of nine, and one a man of sixty-three.

When necrosis occurs, the cartilage is sometimes extruded from the abscess-cavity. An arytaenoid is often expectorated entire, and the larger cartilages break up and come away piecemeal. Suppuration may go on for years; but when the necrosed material is completely got rid of, the cavity may be closed up by fibrous tissue.

Adhesive perichondritis without suppuration or necrosis leads to ankylosis of the crico-arytaenoid joints. Ziemssen records the case of a young man, in whom, during the course of enteric fever, a dark red flat projection appeared over one processus vocalis, causing hoarseness and severe pain. During convalescence this gradually diminished; but he insisted on going out, and after three days' exposure to weather and indulgence in alcohol returned in such severe distress, that tracheotomy was performed.

The *symptoms* of laryngeal perichondritis are not very distinctive at first. The patient usually complains of hoarseness, or of aphonia; there may be dysphagia, cough, and more or less localised pain and tenderness. Before long dyspnoea sets in, which may rapidly increase until it threatens suffocation; but sometimes the spontaneous evacuation of the contents of an abscess-cavity affords relief. Enlargement of the cervical glands is frequently present. The putrid discharge from the necrosed sequestrum is a direct cause of danger, by dropping into the air-passages and setting up pneumonia that may pass into gangrene; two patients in Guy's Hospital died from this cause.

Perichondritis of the *thyroid* cartilage sometimes shows itself on the outer, sometimes on the inner surface of the cartilage. In the former case there is a swelling, oedema, and at length fluctuation over one of the alæ or over the pomum Adami; and the affected part is very tender when pressed upon. In the latter case a swelling usually appears in the position of one sinus pyriformis within the arytaeno-epiglottidean fold on one side, or even below the vocal cord, as in an instance recorded by Störk, in which it was mistaken for a polypus. Not infrequently both surfaces of the thyroid are affected in succession, so that when the abscesses have discharged themselves, milk or any coloured fluid can be injected through a sinus in the neck and run into the larynx, or a probe can be passed from without inwards until it is visible in the laryngeal mirror.

Perichondritis of the *cricoid* cartilage affects its posterior more often than its anterior wall. It causes marked dysphagia, and sometimes paralysis of the crico-arytaenoidei postici muscles, so that the cords appear fixed near the median line. The symptoms may develop themselves with extreme rapidity; Ziemssen cites a case of Pitha's which ended fatally in a week. When suppuration occurs, the abscess may discharge itself into the pharynx or the larynx.

Perichondritis of an *arytaenoid* cartilage leads to swelling and oedema of the soft parts, which may be visible in the laryngeal mirror. The mobility of the corresponding vocal cord is more or less impaired. Dr Fagge's experience was that neither aphonia nor any marked alteration of the voice is at all a constant symptom of disease of an arytaenoid cartilage. Cases of phthisis, in which complete exfoliation was found after death, had sometimes been free from laryngeal symptoms during life. When an arytaenoid has been exfoliated, an obvious falling in of the soft structures is often to be seen with the laryngoscope.

More than one of the laryngeal cartilages not infrequently suffer; one or both of the arytaenoids, together with a part or the whole of the cricoid.

The *treatment* of perichondritis, if the disease is detected early enough, may begin with leeches, the application of an ice-bag to the throat, and other antiphlogistic measures. When an abscess is recognised, whether outside or inside the larynx, it ought at once to be incised.

In almost all cases, however, tracheotomy is sooner or later required; and when dyspnœa has once set in there is great risk in delaying it. Many surgeons prefer intubation, the ingenious alternative introduced by O'Dwyer, of New York. The immediate result is, in either case, almost always successful.

LARYNGEAL TUMOURS.—New growths in the larynx are by no means rare. Benign growths are most seen in persons who use the voice much. They may occur at any age, and are more common in males than in females, and, according to Causit, this is true of children as well as adults.

Papilloma.—This is the commonest of all laryngeal tumours. It consists of one or more pointed or bulbous papillary excrescences, sometimes of small size, sometimes forming a large mass, which may almost fill the cavity of the larynx. Their most frequent starting-point is from one of the vocal cords; but sometimes they arise from the false cords, or from the epiglottis, seldom or never from the mucous membrane covering the arytaenoid cartilages or the parts adjacent. Their colour may be either pale or red. They cause more or less alteration of voice, up to complete aphonia, cough, which may torment the patient greatly, and dyspnœa, which sometimes is severe and fatal. It now and then happens that the fragments of papillary growths become detached in the act of coughing, and are expectorated. Otherwise it is only with the aid of the laryngeal mirror that their presence can be diagnosed. When removed by operation they are very apt to return, sometimes within a few months.

In cases of chronic laryngitis with papillary growths and general thickening, Virchow observed that the lesion is usually situated towards the anterior ends of the cords. This *pachydermia verrucosa* is apt to be recurrent, but is not malignant. We may regard cases as simple, local, and only superficially hyperplastic, in which we find a sharp line of distinction at the base of the epithelial growth separating it from the fibrous tissue beneath. If there is any trace of epithelium in the fibrous tissue the case is serious; but if there is no epithelium beneath this border-line, then, notwithstanding papillary growths, the disease is local and benign.

Dr Percy Kidd described a case of papillary tuberculous tumours growing from the interarytaenoid fold of mucous membrane in a man aged fifty, who died of phthisis with subsequent tuberculous ulceration of the larynx and also of the colon. No giant-cells were discovered, but the other histological characters were those of tubercle, and characteristic bacilli were found ('Clin. Trans.,' 1884, vol. xvii, p. 156). Dr Kidd quotes only one other case, of a young man from whose larynx several tuberculous tumours were successfully removed by Schnitzler ('Wiener med. Presse,' April 8th, 1883).

Fibroma, or fibrous polypus of the larynx.—This forms a round or pear-shaped swelling, generally pedunculated, but sometimes sessile, smooth or lobulated, hard, or more rarely soft in consistence, very vascular, whitish or bright red in colour, varying in size from a pea to a hazel-nut. It is a solitary growth, its development is exceedingly slow, and it never recurs when it has once been completely removed. Its most frequent starting-point is from one of the vocal cords, but sometimes it is attached to some other part

of the larynx. Ziemssen figures one as large as a walnut, which arose from the mucous membrane covering the posterior surface of the cricoid cartilage. Growths of this kind most frequently occur in adult or middle-aged patients. In some few cases a fibrous polypus has become detached spontaneously and has been expectorated. With the laryngoscope the existence and the seat of this sort of tumour are, as a rule, easily recognised.

Almost the only other possibility is eversion of the sacculus laryngis—a curious condition which Dr Lefferts, of New York, has diagnosed in the living subject. Such a specimen, taken from the body of a man who had no laryngeal symptoms during life, was shown to the Pathological Society in 1868 by Dr Moxon, and is now in the Museum of Guy's Hospital; it appeared like a semi-elliptical tumour hanging down in front of one of the cords, and could easily be replaced. The writer had a case in a woman of seventy in Mary Ward, which presented this difficulty, but Mr Symonds found that it was not possible to push the projection into the tube of the larynx, as can be done when the mucous membrane is everted. He therefore removed the tumour, which proved to be a myxo-fibroma.

The symptoms produced by a fibroma of the larynx vary with its seat. Unless it is at a distance from the glottis, there is almost always more or less affection of the voice. When a polypus has a pedicle of some length it may rise between the cords during phonation, and rest upon their upper surface, falling down again between them during inspiration. Dyspnoea is not a constant symptom. In a case of laryngeal fibroma recorded by Lieutaud about a century ago, the patient died of sudden suffocation on stooping out of bed to pick up a book which had fallen on to the floor; he had been conscious some time of the presence in the larynx of something which he could not get rid of by coughing.

Mucous cysts have sometimes been found on the epiglottis, as in a case which Durham recorded in vol. xlvii of the 'Med.-Chir. Trans.' The patient was a boy, aged eleven, who had suffered for some months from dysphagia, from hoarseness and feebleness of voice, and from attacks of dyspnoea, which came on especially during sleep. The cyst, which was situated on the laryngeal surface of the epiglottis, was incised, and gave exit to a glairy, thick, muco-purulent matter; it is therefore evident that the cyst was inflamed, and, indeed, the epiglottidean folds themselves were swollen and cedematous. In other cases a similar cyst has been found in the ventricle of Morgagni.

Other cysts connected with the larynx or the hyoid bone contain sebaceous material, and sometimes cholesterine crystals: they are probably congenital, though they may enlarge afterwards. They do not appear to produce symptoms as a rule; but Dr Abercrombie showed to the Pathological Society in 1881 a remarkable case of congenital cyst of the crico-thyroid membrane, which blocked the glottis so much that the child (a female infant who died on the fourteenth day after birth) had never been able to cry or to breathe properly ('Path. Trans.,' xxxii, p. 33).

In some rare cases a laryngeal tumour has been a *myxoma*, a *lipoma*, an *angioma* (Mackenzie), or an outgrowth of the thyroid body penetrating the crico-thyroid membrane. Cases of enchondroma projecting inwards from the cricoid cartilage are also recorded.

Treatment of benign tumours.—Mackenzie advises that small growths on the epiglottis or on the false cords should be left alone if they give rise

to no inconvenience; he has observed several cases in which small "warts," after reaching a certain size, have ceased to grow.

Various instruments have been devised for the purpose of removing laryngeal tumours through the natural passages by the "endo-laryngeal method;" and knives (guarded or unguarded), cutting or crushing forceps, guillotines, *écraseurs*, the galvano-cautery, have all found their advocates. Their choice depends on the surgeon's judgment in each case. It is not advisable to use an anæsthetic unless tracheotomy has previously been performed; but the local use of cocaine in solution or as spray has superseded other kinds of anæsthesia. Both in this country and abroad an extraordinary degree of skill has now been attained in the performance of endo-laryngeal operations; and, as a rule, more skill is required for the removal of a very small laryngeal growth than of a larger one.

In cases in which it is difficult or impossible to operate through the natural passages, recourse may be had to "thyrotomy," or the division of the thyroid cartilage in the median line, with separation of its halves, enabling the surgeon to seize the growth or growths and to clear out the whole cavity of the larynx at once. This procedure, which had been adopted for the removal of foreign bodies nearly a century ago, was advocated by Durham in a paper read before the Royal Medical and Chirurgical Society in 1871. But subsequent experience seems to have much limited the range of the operation, and at the International Congress in 1881 opinions were almost unanimous against it. It was urged that the operation is attended with considerable danger from hæmorrhage, or from consecutive evils, as pneumonia and necrosis of cartilages. Further, it was shown that a permanent impairment or loss of voice is a not infrequent result of thyrotomy, though there are many recorded cases in which the voice has been perfectly restored. Lastly, it was stated that in some patients there is great difficulty in getting the *alæ* of the thyroid cartilage wide enough apart. Even in young children (in whom multiple papillomata are common) thyrotomy is seldom necessary. Krishaber related the case of a child, aged six, in whom he succeeded in rapidly removing several tumours without a laryngoscope by sliding a pair of forceps along his index finger into the larynx. In children this may be more easily accomplished under chloroform, or with the aid of cocaine. Mr Symonds told the writer of a successful case where many small papillomata were thus removed from a girl of seven, who had a gruff voice, but no dyspnoea. The recovery was complete, and after six years the child remains well with a clear voice.

When, however, endo-laryngeal attempts have failed, or when frequent recurrence has taken place, the question arises of obtaining direct access into the larynx by dividing the thyroid cartilage.

The vast improvement in the treatment of wounds, and the recognised necessity for preventing the entrance of blood into the trachea, have robbed this operation of most of its risks. Thyrotomy in competent hands may be looked upon as carrying no greater risk to life than an exploratory tracheotomy. The main points are, to exclude the blood by a deliberate tracheotomy, all vessels being secured before opening the trachea; to insert Home's sponge tampon; to divide the thyroid exactly in the median line between the cords; to apply cocaine in order to prevent the laryngeal spasm; to close the thyroid with deep and superficial sutures; and at once to remove the tracheotomy tube.

Though this operation carries a comparatively small risk, yet it must only be recommended where other means have failed, and where, in addition, the symptoms demand operation. It would, for instance, be a serious step to advise it for a small growth causing only hoarseness, as, for example, a subglottic pedunculated fibroma.—C. J. S.

A mucous cyst in the larynx requires only to be incised and to have its

interior rubbed with caustic. Contrary to what might have been expected, it seems seldom or never to fill again.

Of malignant growths in the larynx only a few are *Sarcoma*, usually of the spindle-cell kind; its seat is usually on or near one of the vocal cords, but Mackenzie figured a sarcoma growing from the cricoid cartilage.

Carcinoma of the larynx is far more common, and is almost always of the keratoid variety, still often called epithelioma (vol. i, p. 93). It is rare in comparison with cancers of other parts, but, according to Ziemssen, not uncommon compared with other laryngeal growths; he collected 147 cases, of which thirteen had come under his own observation. It attacks many more men than women, and many more patients of sixty and upwards than of a younger age.

The larynx may become affected with cancer by extension from the pharynx or from the base of the tongue; but often the starting-point of the affection is in the laryngeal mucous membrane, its original seat being generally one of the cords, one of the ventricles of Morgagni, or one of the false cords. The amount of the growth at the time of the patient's death is sometimes remarkably small; so that until the microscope reveals the structure of a carcinoma, the fact of a new growth being present may be doubtful. The first appearance of cancer is often that of a papilloma, and removal of part of the growth may give only negative, and therefore misleading, results—as in a well-known case of great public importance in 1888. As the disease spreads, ulceration occurs, and the structures outside the larynx become infiltrated with the growth. It may intrude into the pharynx, or form a tumour in the neck; while the ulcerated surface may pour out a thin discharge mixed with blood. In such cases the breath becomes horribly foetid. Perichondritis, with suppuration, hæmorrhage, and necrosis of cartilages, often occurs as a complication. Death may be due to submucous laryngitis, or to pneumonia or empyema.

Of the general symptoms hoarseness, seldom amounting to complete aphonia, is the most constant, and generally the earliest. Next to hoarseness, pain, which may either be seated in some one spot within the larynx, or referred deeply to the pharynx, is the most conspicuous symptom; and Ziemssen lays stress on the frequent radiation of pain into one or other ear, conducted perhaps by the auricular branch of the vagus. As a rule, dyspnœa occurs sooner or later, especially when the patient is lying down. There may also be dysphagia. Careful search must be made for enlarged cervical glands; but they can seldom be detected within the first six months, and often not for a year; so that, as with cancer of the tongue, the diagnosis would come too late. Secondary cancer of the viscera is very rare.

The laryngoscopic diagnosis of carcinoma is not always easy, and when an ulcer has formed it is often very difficult to distinguish between syphilis and cancer. According to Mr Symonds, it may appear as a pale or pink-coloured wart attached to a vocal cord; with this there is congestion of the cord, and very early impaired mobility. This immobility is one of the most valuable signs, as distinguishing malignant disease from the early stages of tubercle and benign growths; it is caused by infiltration of the muscular structures. But a papillomatous mass attached to one cord in an elderly patient is always suspicious.

Carcinoma sometimes appears as a dark red infiltration, with an irregular depression, or a papillary projection; while in later stages a fung-

ating mass may be seen. A diagnosis can often be made by removing a fragment for microscopical examination; but in warty growths an uncertain result may be obtained, if only the surface of a growth with a malignant base is examined.

When the hoarseness persists, when no amelioration takes place on treatment, and when no positive evidence can be obtained by endo-laryngeal removal of a fragment, the question of an exploratory thyrotomy must be considered. This proceeding is, if properly carried out, attended with little risk. Should the malignant nature be confirmed, then, if the case be otherwise suitable, a partial or complete extirpation can be carried out.

The medical treatment of carcinoma of the larynx can be only palliative; but whenever there can be a doubt as to the nature of the disease, the patient should have the chance of a course of iodide of potassium. Tracheotomy is generally required sooner or later; and the duration of life after this is seldom more than a year.

The only effectual treatment is the total extirpation of the larynx. This operation, first performed for syphilitic stenosis by Dr Heron Watson, of Edinburgh, in 1866, was carried out in 1873 by Billroth with temporary success in a case of cancer. In a paper read in 1881 by Dr Foulis, of Glasgow, before the International Congress, reports of thirty-two cases of the operation were collected, in twenty-five of which the disease was carcinoma. In fourteen out of the twenty-five death occurred within sixteen days after the operation; and in not one of the remainder was life known to have been prolonged more than nine months. Since then results have greatly improved. Bottini, of Turin, had an early successful case, in which the larynx was extirpated for sarcoma; in 1881, six years after the operation, the patient was well, and had been able to work in the fields and as a postman. In a more recent case, the affected half of the larynx was removed by Dr E. Hahn, of Berlin, and not only was life prolonged for several years, but the voice was restored by the use of the artificial larynx, originally invented by Störk, so as to enable the patient to continue his duties as the magistrate in a London police court.

In an abstract in the 'Brit. Med. Journ.' of June 16th, 1888, from Dr Max Scheier's paper in the 'Deutsche med. Wochenschr.' of June 7th in the same year, statistics of sixty cases of removal of the larynx for cancer were given. Of these, about 20 died from the effects of the operation, 16 within a year from return of the disease, and 9 remained free from cancer sixteen months after the operation.

During the last five or ten years further improvement has taken place. In 103 cases of carcinoma, Semon reports 16 of extirpation of the half or the whole of the larynx, and of these eleven ended in recovery. Mr Butlin has also met with remarkable success in this operation, and it may now be regarded as offering a good prospect—and the only one possible—of recovery, if performed at an early date.

MALFORMATIONS OF THE LARYNX.—Certain congenital abnormalities of the larynx occasionally give rise to clinical symptoms. One such appears to be almost confined to female infants; it causes the act of inspiration to be attended with a loud crowing noise, which is nearly constant, continuing even during sleep and after the administration of chloroform, though it is louder during the day. It is sometimes increased by exposure of the child to cold, or in other ways. The noisy state of the breathing is present from

the time of birth, but disappears entirely at the end of about a year. Dr Lees has had an opportunity of making an autopsy in a case of this kind, in which death was due to diphtheria; and he found ('Path. Trans.,' 1883) that the epiglottis was folded on itself, like a leaf on its midrib, the arytaeno-epiglottic folds being almost in contact. This condition had been seen in the laryngeal mirror during life, and it is probably not uncommon, for Lees had seen three other cases, and it has also been recorded by Gee and by Barlow. It is important as simulating laryngismus stridulus.

Two cases of the same malformation were shown to the Laryngological Society in December, 1897 (also 'Lancet,' September of same year) by Dr L. Lack, who described the clinical condition as *Congenital laryngeal stridor*. He and Dr Sutherland found the epiglottis "folded laterally so sharply that its lateral halves come very close together, or even into actual contact," while the arytaeno-epiglottic folds flap inwards at each inspiration so as to constrict or even close the entrance to the larynx. As in Dr Lees' case, the stridor and obstruction passed off before the close of the second year owing to the natural growth of the parts.

Another and a much more serious malformation, described by Mackenzie, consists in a longitudinal bifurcation of the epiglottis, forming two flaps which (in a case that he saw) fell into the larynx, and caused constant laryngismus from the first week and death at the end of four months.

A congenital band of mucous membrane sometimes connects together the anterior parts of the cords. Mackenzie recorded a case in vol. xxv of the 'Pathological Transactions.' The patient was a young lady of twenty-three, who had suffered from complete aphonia all her life, apparently without dyspnoea. With the laryngoscope the web was seen as a flat membrane during inspiration, but on attempted phonation it folded up, and protruded so as to resemble a tumour, of red colour, and of about the size of a haricot bean. It was excised, and the patient immediately afterwards spoke, and soon acquired a perfectly natural voice. In a similar case which Dr Poore exhibited at the International Congress in 1881, the patient, a girl of thirteen, could speak, but with a peculiar falsetto tone of voice; she had been liable to attacks of dyspnoea from infancy.

OBSTRUCTION OF THE TRACHEA AND BRONCHI

MEDIASTINAL GROWTHS, ETC.

“Doubtful his death ; he suffocated seems.”

DRYDEN'S *Ovid*.

Pressure on the trachea from enlarged thyroid, from aneurysm, mediastinal abscess or growth, from enlarged lymph-glands, cancer of the œsophagus, or dilated auricle—Stricture of the trachea—Impaction of trachea or bronchus by foreign bodies—Symptoms of tracheal obstruction—Sequelæ—Diagnosis, duration and treatment.

Inflammation of the mediastinum, acute and chronic.

Sarcoma and cancer of the mediastinum and root of the lung.

New growths in the pulmonary tissues, primary and secondary.

Pulmonary embolism—its causes, symptoms and results.

IN this supplementary chapter we will deal with several morbid conditions, which differ in pathology, but agree in leading to obstruction of the air-passages between the Lungs and the Larynx ; and also with affections of the mediastinum, whether they cause such obstruction or no.

Inflammation of the trachea is scarcely an independent disease ; it usually accompanies inflammation either of the larynx or of the bronchi, and clinical importance naturally belongs to affections of the narrower rather than to the wider parts of the air-passages. Tracheitis has been described under diphtheria, tuberculous laryngitis, and catarrhal or plastic bronchitis ; but many conditions not yet considered may narrow the calibre of the trachea or of the bronchi, and produce a characteristic group of symptoms. Of these, some compress the air-passages from without ; others affect the walls of the passages themselves ; and others, as foreign bodies, obstruct their channel from within.

It is often an accident whether in a given case the part narrowed is the lower end of the trachea or one of the primary bronchi, and both are frequently involved at the same time.

Stenosis from compression—by an enlarged Thyroid.—A bronchocele may compress the trachea in the neck, flattening it from side to side, so as to make it “scabbard-shaped,” and often pushing it out of the straight line. It is not always the largest goitres which act thus ; much depends

on the exact situation of the growth, and on the strength of the overlying muscles. Moreover, as Virchow taught, the middle lobe of the thyroid, when enlarged, may pass down behind the sternum so as to compress the trachea backwards against the spine. Ross, of Zürich, in vol. xxii of the 'Arch. f. klin. Chirurgie,' drew attention to a peculiar change in the tracheal cartilages which occurs as the result of a goitre, and renders them soft and flaccid. This may be recognised after death by dissecting out the larynx and trachea and placing them upright; the tube at some one point bends sharply by its own weight, so that its channel becomes completely closed. A like condition is probably the cause of sudden attacks of dyspnoea in patients suffering from goitre; they instinctively maintain the head in a safe position, but the muscles become relaxed during fainting, or sleep, or chloroform narcosis, or from mere exhaustion.

Dr Bristowe recorded, in the 'St Thomas's Hospital Reports,' vol. iii, the case of a woman who was one day suddenly attacked with intense dyspnoea, followed by blackness of the face and insensibility. Fortunately, he was close at hand, and finding a cystic tumour in front of the neck, he had this punctured; two or three ounces of a reddish-brown fluid were removed, and she was saved from imminent suffocation. In other cases a hydatid or a malignant growth of the thyroid may press upon or perforate the trachea, and thus cause death.

Aneurysm.—Among twenty-seven cases of thoracic aneurysm taken without selection from the *post-mortem* records of Guy's Hospital, Dr Fagge reported that there was pressure on the trachea or a main bronchus in every one. In fourteen the sac arose from the arch and pressed backwards upon the lower end of the trachea, flattening it, and often adhering to its walls. Probably in several of these cases the pressure extended also to one or both of the bronchi; but in seven cases the aneurysm seems to have pressed solely upon the *left* bronchus: in three of these the sac arose from the summit of the arch on its left side, and pressed mainly on the upper or the anterior aspect of the bronchus; in the other four it arose from the descending part of the arch and pressed forwards upon the posterior surface of the same bronchus. In only two cases the sac, arising from the right side of the arch, compressed only the *right* bronchus. The remaining four cases were aneurysms of the root of the innominate artery, and the sac pressed upon the trachea behind the upper part of the sternum.

In three of the twenty-seven cases the aneurysm was of only moderate size. One, which flattened the trachea, was the size of a walnut; another, which compressed and opened into the left bronchus, was "no bigger than a marble;" and the third, which likewise pressed on the left bronchus, was "the size of a small plum."

Mediastinal tumour.—In the same period during which these twenty-seven cases of aneurysm were observed in the deadhouse at Guy's Hospital, there occurred nearly an equal number of cases in which the great air-passages were narrowed by mediastinal growths. Among twenty-four in which details are given as to the exact seat of the lesion, there appear to have been eight in which the obstruction affected the lower end of the trachea or both bronchi (sometimes one much more than the other), six in which it was limited to the right bronchus, and ten in which it was limited to the left. In every instance the new growth invaded the walls of the air-passages, and did not merely press on them. In two other cases, beside the twenty-four already mentioned, although the bronchus was penetrated

by the tumour, there was no narrowing of its calibre. Mediastinal tumours appear seldom to invade the trachea or the bronchi in their early growth; but we must remember that they are not likely to be seen in the deadhouse at this stage, since, unlike aneurysms, they do not often destroy life suddenly by hæmorrhage (cf. *infra*, p. 153, *et seqq.*).

In a case of lympho-sarcoma of the mediastinal glands, recorded by Weil in the 'Deutsches Archiv' for 1874, all the symptoms and signs of tracheal obstruction disappeared suddenly eight days before death; at the autopsy it was found that this was due to the giving way of the softened mass, which must have escaped into the air-passages, although the sputa had shown no fresh appearance even under the microscope.

Abscesses of various origin may compress the trachea or bronchus. Of this, a striking case was recorded by Schnitzler, in the 'Wiener Klinik' for 1877: the patient was four years old; an abscess as large as the child's fist pushed the trachea forwards and to the right; its starting-point was caries of the second and third dorsal vertebræ.

Caseous disease of the bronchial glands.—This is supposed to be one of the causes of obstruction of the trachea or of a bronchus, especially in children; but such cases are not numerous, and Vogel doubts whether the slight flattening frequently observed ever goes on to actual stenosis. Widerhofer, in Gerhardt's 'Handbuch,' cites instances in which, after prolonged dyspnœa, abscesses of the bronchial glands discharged into the air-passages with relief to the urgent symptoms.

Carcinoma of the œsophagus is an occasional cause of stenosis of the trachea; but although it frequently invades the air-passages, it seldom causes interference with the entrance of air. As already remarked (p. 113), it may cause a bilateral paralysis of the abductors of the glottis, and so render the performance of tracheotomy necessary.

A dilated left auricle, secondary to mitral stenosis, may compress the left bronchus, as was first pointed out by Mr Wilkinson King in 1838 ('Guy's Hosp. Rep.,' iii, p. 175). Three of his preparations are in the museum of Guy's Hospital (No. 188). Friedreich recorded an instance in which pressure on the bronchus from this cause was actually diagnosed by physical signs four years before the patient's death. At the autopsy, made by Virchow, it was found that only a very narrow channel was left.

Stenosis from disease of the walls of the trachea.—Langhans, in 'Virchow's Archiv' (vol. liii, pl. xiii), recorded a case of primary carcinoma, having its origin in the mucous glands of the lower end of the trachea and right bronchus, which destroyed the life of the patient, a man of forty; it appeared as a warty affection of the lining membrane, infiltrating the muscular and fibrous coats. Whether a local inflammation can produce such thickening of walls of the trachea as to obstruct its calibre is doubtful. Wilks is quoted by Riegel as having reported such cases; but his observations refer to syphilitic stenosis only. He also quotes Demarquay for a case of stenosis from ulceration in the course of glanders.

Syphilis is, indeed, by far the most important cause of obstruction of the lower air-passages, apart from impaction by foreign bodies or compression from without. Gerhardt analysed, in vol. ii of the 'Deutsches Archiv,' twenty-two examples; and seven instances presented themselves among the autopsies at Guy's Hospital in thirteen consecutive years. Occasionally the disease is limited to a single spot in the trachea, as in a specimen taken from a patient of Dr Bright, and now in the museum of

Guy's Hospital, which shows opposite the second ring a contraction, as if produced by a ligature; but more often it extends along the trachea, and may be prolonged into one or both of the bronchi.

The bronchi are seldom affected when the trachea escapes; but Wilks recorded in the 'Guy's Hospital Reports' for 1863, a case in which the right bronchus alone was narrowed; and in another case, observed at the hospital in 1875, the lesion was found to affect only the left bronchus and the upper branch of the right; in a third case, both bronchi were narrowed, while the trachea was free ('Path. Trans.,' vol. xxviii, p. 336). The mucous membrane is often raised into a series of irregular bands and ridges, which Wilks taught us to regard as the cicatrices of former ulcers. Gerhardt recorded a case of chronic cirrhosis of the lung, in which there was an ulcer with raised edges found occupying the right bronchus and one of its branches for about an inch; but more often we meet with a diffused thickening of the whole tracheal wall, raising its lining membrane into folds and prominences to which ulceration, though not infrequent, is only secondary. Whether primary or not, such specific ulcers may spread deeply, setting up perichondritis, and leading to ossification and necrosis of the tracheal or bronchial cartilages, which may be exfoliated and appear in the sputa, just as we saw in the case of the arytaenoid cartilages (p. 136); or they may penetrate to the tissues outside the air-passages, and form an external abscess. In a case that occurred at Guy's Hospital in 1865 there was perforation of the aorta, so that the patient died of sudden hæmorrhage. In other instances the tracheal rings, instead of being exposed and detached, become atrophied and bent on themselves, or dragged one over the other.

Whether syphilitic stenosis affecting a bronchus ever leads to its complete obliteration is doubtful, and such a condition when found has been supposed to be congenital. Ratjen, in vol. xxxviii of 'Virchow's Archiv,' thus regarded a case occurring in a man aged forty-nine, whose left bronchus was converted into a fibrous cord for an inch and a half of its length, the corresponding lung being quite airless, while the right lung was enormously enlarged, apparently by true hypertrophy, for its air-cells were of normal size. But, as Cohnheim observes of this case, the presence of pigment in the collapsed left lung seems to show that it had at one time been functionally active.

Syphilitic stenosis of the trachea, judged by the fatal cases, is most common between forty and fifty, and occurs in men far more often than in women; at least this has been the experience at Guy's Hospital. Gerhardt found one case recorded in a patient under ten, and another under twenty years old. Probably these were due to inherited syphilis, like two other instances in children twelve years old given by Widerhofer.

Obstruction by a foreign body.—Usually the patient coughs, or laughs, or sneezes, with food* or some small article, like a button or a coin, in his mouth, and a deep inspiration carries it into the glottis. Here it may excite a violent cough, and be expelled at once or after more than one effort; or it so completely and firmly obstructs the larynx that the patient dies at once without symptoms of dyspnoea; or it slips down below the glottis. The last is the case with all except very large objects, or those

* Not only food received into the pharynx from the mouth, but also that which is vomited up from the stomach may be sucked into the larynx and excite spasm of the vocal cords and all the symptoms of suffocation.

with sharp jagged edges, which stick in the mucous membrane. Thus beans, peas, pebbles, small coins, or fragments of bone, most often fall into the trachea. The violent efforts at relief, the cyanosis and sweating and extreme distress are then immediately relieved, for the larger calibre of the trachea allows air to pass beside anything which has fallen through the chink of the glottis. Sometimes the foreign body remains free for a time, moving up and down as the patient coughs; and we may then be able to feel its impact against the side of the trachea with the fingers placed on each side the patient's neck, as was observed by Mr Lucas in the case of a child with a pebble in its air-passages ('Clin. Soc. Trans.,' xv). In that case there were physical signs which rendered it probable that the pebble lay in the right bronchus in the intervals between the fits of coughing.

As a rule, such bodies soon become fixed in the right bronchus or in one of its main divisions, because the fork between the two is slightly to the left of the middle line, so that the passage into the right bronchus is rather the more direct. Sometimes, however, the left bronchus is the one into which a foreign body passes; and sometimes each bronchus in turn, the offending substance being dislodged by cough, and falling now into one, now into the other. In certain cases the cause of obstruction is "foreign" only in its locality; it may be a tooth, or a pharyngeal polypus separated by the hand of the surgeon; or it may be a necrosed laryngeal cartilage, a concretion from a bronchial gland, or a hydatid from the liver. An accident which has several times happened after tracheotomy is that a portion of the tube has become detached and has dropped into the trachea. Altogether the variety of foreign bodies in the air-passages is very extensive, and 374 recorded cases were collected and analysed by Kühn.

As may be supposed, foreign bodies are most frequently found in the air-passages of children and of lunatics. But other patients also, when attacked by sudden and violent symptoms as the result of this accident, may not be able to explain the cause. Hamburger (cited by Riegel) recorded the case of an old man aged seventy, who fainted after a journey, and was found in a state of dyspnœa, with evident obstruction of the right bronchus. An emetic was given, which led to the expectoration of a green pea swollen to the size of a bean; and afterwards it was learnt that when he was eating peas one day something had gone the wrong way.

Symptoms of obstruction of the lower air-passages.—Of these the most important is dyspnœa; as contrasted with laryngeal stenosis, that of the trachea is characterised by difficulty of breathing without loss of voice. It is not, however, conversely true that the power of speaking proves that the cause of dyspnœa is not in the larynx; for in bilateral paralysis of the abductors of the vocal cords precisely this combination of symptoms is met with (p. 112). Moreover it frequently happens that the voice in cases of tracheal obstruction is weak and thin, from deficiency in the stream of air reaching the larynx from below. Again, a patient may have lost his voice as the result of syphilitic ulceration of the larynx, but the dyspnœa from which he suffers may depend on the same ulceration lower down, so that tracheotomy would prove a failure. Moreover, in many cases of aneurysm or of mediastinal growth, tracheal stenosis is accompanied by paralysis of laryngeal muscles, as the result of pressure upon one or both of the recurrent laryngeal nerves.

Whether or not the voice is affected, it is therefore essential to make a

thorough laryngoscopic examination in all cases of suspected stenosis of the trachea or of the main bronchi. Moreover, it is sometimes possible, especially if the larynx is healthy, to ascertain the nature of a tracheal lesion by examination with the mirror. The lower part of the windpipe, with its bifurcation and the orifices of the two bronchi, is said to have been first seen in the person of Czermak himself by Elfinger. Türck has described the conditions most favourable to a successful view of these parts. The patient should be seated with the body and the neck upright and the head bent slightly forward, the object being to bring the axis of the larynx and that of the trachea into a straight line. The mirror must be placed against the soft palate, rather further forwards than usual, and with its surface nearly horizontal. The observer should sit at a lower level than the patient. The illumination must be very bright, and the light should be thrown into the mouth horizontally, or rather from below. An aneurysm may sometimes be seen bulging into the trachea, as in a case of innominate aneurysm, which was discovered by Mr Lane when house-physician at Guy's Hospital. It must not, however, be supposed that a slight pulsation of the lower end of the trachea always indicates a morbid condition, for Gerhardt and Schrötter have shown that such pulsation transmitted from the great arteries is present in many healthy persons.

The dyspnoea from stenosis of the lower air-passages is chiefly inspiratory, like that in laryngeal stenosis (cf. *supra*, p. 134). It is less often extreme, on account of the greater calibre of the trachea compared with that of the glottis. Hence the breathing is not usually much altered in frequency; nor are the lower ribs and the other unsupported parts of the chest walls much sucked in. But in the most severe cases of the disease, all these symptoms are as marked as they can be.

There is generally noisy stridor, heard not only through a stethoscope over the trachea, but also more or less on auscultation over every part of the chest, so that it drowns the normal breath-sounds. Indeed, it is usually heard by every one standing near the patient. When there is stenosis of the lower part of the trachea, the sound is often audible with greater intensity over the larynx than over the sternum; but sometimes a râle is constantly discoverable over the seat of obstruction. A sign to which Demme has drawn attention is that in prolonged cases of constriction of the lower air-passages the circumference of the upper part of the thorax is diminished.

In most cases of stenosis of the trachea, the dyspnoea undergoes aggravation from time to time; there are paroxysms of extreme distress attended with cyanosis, and generally one of them at length proves fatal. It was formerly supposed that the cause of such attacks was paralysis, or spasm of the vocal cords, from implication of the recurrent laryngeal nerves; but Bristowe, in the third volume of the 'St Thomas's Hospital Reports,' showed that this is not the fact. The cause is probably either acute swelling of the mucous membrane at the seat of pressure, or accumulation of mucus there which cannot be dislodged, or possibly spasm of the muscular tissue of the trachea itself.

The patient complains more or less of oppression of the chest, of soreness behind the sternum, or of actual pain. There may or may not be cough, with expectoration of mucus, perhaps tinged with blood, according to the nature of the disease which produces the stenosis.

Local diagnosis.—An important distinction between stenosis of the

lower air-passages and that of the larynx was first pointed out by Gerhardt. It is that in the former case the larynx does not during inspiration make the rapid and extensive vertical movements which occur when the larynx is itself the seat of obstruction to the entrance of air. According to him, if with severe stenosis the range of descent of the larynx is not more than one centimetre, one may confidently assert that the disease is either in the trachea or possibly in both bronchi, but not in the larynx. He also remarks that the position of the patient's head differs in the two sets of cases. When the obstruction is laryngeal, the head is thrown backwards as far as possible. When it is tracheal, the head is stretched forwards, and the chin slightly depressed, so as to relax the trachea.

In contrasting the physical signs of obstruction of one bronchus with those of stenosis of the entire lower air-passages, we find that much depends on whether the obstruction is complete or partial. In the former case there is absence of vesicular murmur over the corresponding side of the chest, with impaired movement of the ribs and of the diaphragm, deficient vocal fremitus, and a normal percussion-sound. After a time the side may be found to have fallen in, and to measure less than the other side. In the latter case a snoring, whistling, or humming sound may be heard over the root of the lung between the scapula and the vertebræ, or large rattles may be audible. A thrill may sometimes be felt with the hand placed upon the surface of the chest.

One clinical peculiarity of the obstruction of a main bronchus caused by a foreign body is that it is far more sudden, as well as more complete, than that due to any other cause. Its effects may be supposed to approach more closely than in any other morbid condition likely to be observed in man to those of plugging a bronchus by wedges of laminaria, as recorded by Lichtheim in a series of experiments on rabbits ('Arch. f. exp. Pathologie,' vol. x). The opposite lung in these experiments became distended, and often gave way, so that pneumothorax resulted; and even when this did not occur the animal usually died within twenty-four hours. What proved that the rapidly fatal issue depended on the state of this lung and not the one deprived of air, was that death did not follow when the pleura was laid open on the side of the obstructed bronchus. A bean or a pea is capable of swelling, like the laminaria plugs used by Lichtheim, although more slowly; and his results are worth bearing in mind, because in the failure of all attempts to extract a foreign body from a bronchus, it might sometimes be justifiable to admit air into the pleura; even if the operation did not prolong life it might greatly relieve the dyspnoea.

Sequelæ.—Every form of disease producing obstruction of the lower air-passages is liable to be followed by changes in the mucous membrane below the obstruction, and ultimately in the pulmonary tissue. Thus when an aneurysm has pressed upon the trachea, or upon a bronchus, the mucous membrane is often seen ulcerated, and some of the cartilages exposed and partially detached. Stenosis of a bronchus, from whatever cause, is often followed by dilatation of its branches in the lung. Purulent fluid is apt to accumulate in them, and the result is foetid bronchitis or gangrene. A foreign body fixed in a bronchus often sets up ulceration and sloughing of the part of the tube against which it presses. Sometimes this ends in perforation of the pleura, with pneumothorax, and the foreign body may become loosened and fall into the serous cavity. Sometimes it leads to septic pneumonia, which may spread from the root of the lung far into its

substance. The occurrence of foetid expectoration, and the development of the physical signs of hepatisation may reveal these various changes, but in some cases they are first detected at the autopsy. Even after expulsion or removal of the foreign body, the case has occasionally ended fatally from the secondary lesion in the lung.

In attempting a *diagnosis* between the several affections that may cause obstruction of the lower air-passages, it is worth remembering that the two diseases in the course of which stenosis of the trachea is most apt to occur without other symptoms are *syphilis* and *aneurysm*. When the obstruction is limited to a bronchus, especially perhaps on the left side, aneurysm is still the most probable diagnosis; and a mediastinal growth is more likely than a syphilitic stricture. The possible presence of a foreign body must never be forgotten, even if the symptoms have not come on suddenly.

The *duration* of syphilitic stenosis of the trachea, after symptoms have set in, ranges, according to Gerhardt, from two months to four years. That of stenosis from compression would probably be found confined within much narrower limits. Foreign bodies sometimes remain in the lower air-passages for a very long period—for weeks or months—and yet are after all expectorated.

As to the *treatment* of the various affections that may cause stenosis of the trachea or of the bronchi, there is little to be said. Whenever there is a possibility that it may be due to syphilis, mercury and iodide of potassium should be actively employed. Unfortunately, however, the cases which are usually seen, where cicatricial bands and ridges have already been formed, appear not to be amenable to anti-syphilitic remedies.

Gerhardt relates, in vol. ii of the 'Deutsches Archiv,' the case of a man, aged thirty-six, who had had constitutional symptoms after a hard chancre eight years before, and who consulted him on account of cough with scanty muco-purulent expectoration, a tickling sensation behind the sternum, a little alteration of voice, and slight interference with the breathing. These symptoms had been present for about six months. The patient had lost flesh, and his face was somewhat puffy and livid. Nothing could be discovered with the laryngoscope, and only râles behind the manubrium with the stethoscope. A permanent cure was effected by full doses of iodide of potassium during several weeks.

When there is a foreign body in the air-passages, the only proper course is to perform tracheotomy at once. Until this has been done, it is not safe to place the patient head downwards in the hope of expelling the obstructing object; but after tracheotomy this may succeed, as it did in the case of Mr Brunnel, so graphically narrated by Watson. The same consideration applies to the use of an emetic; for the body, if dislodged from its position in a bronchus, might become impacted in the glottis and cause suffocation.

MEDIASTINAL INFLAMMATION.—The mediastinum is not a space at all, and the loose connective tissue which fills the irregular series of subserous intervals between the thoracic viscera is not an anatomical structure in itself, and is not peculiar to the thorax; but answers to the subserous tissue of the parietal pleura and the subperitoneal and post-peritoneal connective tissue of the abdomen. It is liable to two kinds of inflammation—one acute in course, suppurative in exudation, and septic in origin; the other chronic in course, interstitial and sometimes hypertrophic in character, and irritative rather than decidedly inflammatory in origin. Both are secondary; the suppurative to streptococci and other pyogenic organisms, the fibrous to some primary lesion of the thoracic walls or viscera.

Mediastinal suppuration is met with occasionally in the form of one

or more circumscribed abscesses behind the sternum or in front of the vertebræ, sometimes encapsuled in dense fibrous tissue. The source of the suppuration is most often caries of the sternum, ribs, or vertebræ, sometimes infection of the mediastinal lymph-glands by general pyæmia, and sometimes extension of local suppuration by a fistulous communication from the liver or kidney—a suppurating hydatid or actinomycosis, or a tropical abscess of the liver, or a calculous pyelitis, or extension downwards from the cervical fascia (as in one case where a tracheotomy wound was the seat of origin), or upwards from the post-peritoneal connective tissue. Again, it may be secondary to empyema, or to suppurative pericarditis, or to abscess of the thymus; or it may be of a sloughing character, as when it follows perforation by an œsophageal cancer, or by a foreign body in the gullet or windpipe.

Lastly, it is frequently caseous in character, and forms part of tuberculous disease, starting in the sternum or mediastinal lymph-glands.

The writer had many years ago a woman about fifty years old under his care in Addison Ward. She suffered from tuberculous disease of the cervical lymph-glands, from what might be called *tabes mesenterica*, and also from a series of caseous abscesses behind the sternum. She died after being several weeks in hospital, and at the autopsy, while extensive caseous tubercle of the thoracic and abdominal lymph-glands and of the anterior mediastinum was found, there was no tuberculosis of the lungs or other viscera.

Dr Goodhart described two cases of acute mediastinal suppuration in 1877 ('*Path. Trans.*,' xxviii, p. 37).

One was a man of 57, a patient of Sir Samuel Wilks. There was a history of a severe blow on the chest a few days before. The symptoms were marked dyspnœa and local pain and tenderness in the left mammary region. He died the day after admission and there was found double pleurisy, purulent on one side, plastic on the other, and also plastic pericarditis with purulent infiltration through the anterior mediastinum.

The other was a baker æt. 44. For some months he had felt solid food stop in its passage to the stomach, though he could swallow liquids easily. Beside this dysphagia, there was urgent and increasing dyspnœa without evidence of disease of the lungs by auscultation, nor did the laryngoscope explain his symptoms. He died suffocated on the fifth day after admission. At the autopsy there was acute double pleurisy with purulent infiltration of the lymphatic vessels of both pleura and lung. A large abscess occupied the bifurcation of the trachea and surrounding connective tissue. There was no visible obstruction of the gullet, and it was thought that the dysphagia was due to reflex spasm.

In the 24th and 27th volumes of the same '*Transactions*,' Dr Moxon described two cases of acute mediastinal suppuration, both of which seemed to be of pleuritic origin. The pyrexia in these and in Dr Goodhart's cases was moderate—less than in cases of pyæmia or phlegmonous erysipelas; the dyspnœa was severe, but not paroxysmal.

The writer once collected fourteen cases of mediastinal inflammation which had occurred during three consecutive years at Guy's Hospital (1873-4-5). Five of these were chronic, two purely hypertrophic and fibrous, and three tuberculous, probably originating in the mediastinal glands. Two acute cases secondary to pneumonia showed œdema and softening of the tissues, but no actual suppuration. Seven others were acute and purulent, one occurring in the course of Bright's disease, four spreading down from the neck, one up from the abdomen, and one by suppurative phlebitis of the jugular vein from caries of the mastoid process.

Dr Sidney Phillips published a remarkable case in the '*Med.-Chir. Transactions*' for 1898, in which left pleurisy and empyema was complicated by secondary inflammation of the mediastinal glands. After apparent recovery, death at last ensued by repeated hæmorrhage from a calcareous lymph-gland opening into the arch of the aorta.

Occasionally an acute inflammation in the mediastinum ends in gan-

grene; this appears to be due to contamination with saprophytes conveyed by direct communication from the air-passages or the œsophagus.

The *chronic interstitial* form of inflammation may start from a carious or tuberculous abscess, but more often it is quite free from suppuration, and begins by extension from hypertrophic pericarditis or thickening of the pleura after pleurisy. Occasionally its origin is explained by finding gummata in the adjacent lung, or periosteal nodes on the back of the sternum. Neither the acute nor this chronic form of disease are ever traumatic in origin.

Lastly, it may form part of a chronic hypertrophic inflammation of the whole chest, accompanying pleurisy and pericarditis, or, in some cases, forming part of a process affecting the entire cœlom or mesoblastic space in the thorax, abdomen, and tunica vaginalis, such as has been described in the chapter on pleurisy (vol. i, p. 1084). In such cases the lungs, heart, and abdominal viscera cannot be removed separately, but form part of a vast fibrous mass which binds them in almost inextricable union. The majority of these cases are probably tuberculous in origin, but certainly not all.

The chronic indurative form of inflammation was described in connection with pericarditis by Wilks in 1871 ('Guy's Hosp. Reports,' 3rd series, vol. xvi, p. 201), more fully by Kussmaul in 1873, and, among recent authors, by Dr Harris, of Manchester, in 1895, and by Dr Frederick Roberts in 'Allbutt's System' in 1899. The disease is, according to Dr Harris's collection of twenty-two cases, more common in men than in women; and it is not unfrequent in children.

Dr Fowler has recorded a case of chronic inflammation of the mediastinum complicating aortic aneurysm, which led by fibrous contraction to stenosis, and at last to complete obliteration of the superior cava.

The *symptoms* of mediastinal inflammation are few and uncertain. In the suppurative cases the temperature is irregularly high, as in other septic cases. Pain is sometimes referred to the back of the sternum, and there may be tenderness on pressure of that bone or of a rib or cartilage, but more often pain is varying in seat or absent. If the suppuration points towards the neck or abdomen, œdema, swelling, and perhaps fluctuation may be detected over the manubrium or below the ensiform cartilage.

The symptoms of chronic interstitial mediastinal inflammation are also very uncertain. Most characteristic are the results of the pressure and contraction of the new-formed fibrous tissue upon the tubular organs passing through the chest, on the trachea and œsophagus and arteries, and above all on the great venous trunks, which offer scarcely any resistance to the strangulating effects of the surrounding tissue. According to Kussmaul, the pulmonary veins frequently suffer. The effect is to obstruct the passage of blood through the internal jugular or the innominate veins, the superior cava, or the azygos vein.

When this is the case, we have such obvious symptoms as œdema of the head and neck, or of one arm, and dilatation of the anastomosing veins under the skin. Most of the cases we meet with of greatly dilated and tortuous superficial mammary and superficial epigastric veins are probably due to mediastinal obstruction.

Owing to the resistance offered by their thicker coats, the great arteries are very seldom constricted under these conditions, and the trachea still more seldom. When dyspnœa is present, it probably depends upon the

expansion of the lungs or the movements of the heart being hampered. Occasionally the recurrent laryngeal nerve has been found paralysed by pressure in the root of the neck.

The physical signs consist of extension of the cardiac dulness, faintness of the impulse and of the cardiac sounds, with indications of thickening of the pleura on one or both sides.

The occurrence of the *pulsus paradoxus* (*i. e.* of the radial pulse ceasing during inspiration) may also indicate chronic mediastinal constrictions. In one case of the writer's, Dr Perez, of the Canary Islands, directed his attention to a new sign afforded by listening to the chest while the patient gently moves one arm; in this case the creaking sound was distinctly audible.

MEDIASTINAL NEW GROWTHS.—These intra-thoracic tumours differ in their starting-point and in their histology; but they form a single clinical group, and produce similar effects.*

The anatomical starting-point of a mediastinal growth is often difficult to decide at an autopsy, when it has generally reached a great size and involved various tissues. A frequent origin is from the bronchial and other lymph-glands, especially when the thoracic affection is only part of Hodgkin's disease or general lympho-sarcoma, or when it is secondary to a tumour seated in some other part of the body. Some of the glands are infiltrated with the new growth, while others, though embedded in it, retain their natural structure. In other cases the origin of the tumour is probably the thymus† or the mediastinal connective tissue and fat, or perhaps the periosteum of the sternum or vertebræ, or the cartilages of the ribs or trachea.

The greater number of mediastinal tumours are made up of small round-cells, and are classified either as lymphoma or as round-celled sarcoma. But some specimens contain a large proportion of spindle-cells, others consist of little but fibrous tissue, and some have been described as having an alveolar structure, and styled either alveolar sarcoma, or carcinoma. Another group of mediastinal tumours consists of gummata embedded in a dense fibrous material. Endothelioma and osteo-chondroma have also been recorded. Lastly, some have been dermoid cysts, containing hair, bone, and occasionally teeth, beside a large quantity of fat.‡

Symptoms.—The most important symptoms of mediastinal tumours depend on their relation to the other structures in the thorax, particularly the great systemic veins and the main air-passages.

Obstruction of the great veins—a symptom almost peculiar to mediastinal inflammation or tumour—either affects the superior cava, or one or other of the innominate veins, or all three. In one case a growth in the lower part of the thorax reduced the orifice of the inferior vena cava to a mere slit, through which the finger could scarcely be passed. As we shall see, aortic aneurysms occasionally, but only occasionally, compress the veins; and almost the only other cases in which the flow of blood through the superior cava is retarded or prevented are certain cases of heart disease.

* In his valuable monograph on Mediastinal Tumours (1892) Dr J. L. Steven quotes a case recorded by the great Dr Thomas Willis in 1679.

† Dr Rolleston has lately (1897) published a remarkable case of a hypertrophied thymus, causing death by compression of the trachea, in a boy six years old ('Path. Trans.,' vol. xlviii, p. 200).

‡ A case of congenital dermoid cyst was recorded by Dr Hale White, in which it was adherent to the pericardium ('Path. Trans.,' vol. xli, p. 283).

in which, during an acute attack of pericarditis, there has been inflammation of the mediastinum with thrombosis of one or both of the innominate veins, ending in obliteration of the affected vessels. But when there is a mediastinal new growth, venous obstruction is a very frequent result. Sometimes the growth penetrates the coats of the vena cava, and forms a soft smooth mass, encroaching on its lumen, in accordance with Wilks' rule, that malignant growths readily invade veins, but only perforate arteries when they are complicated by sloughing. Sometimes it surrounds the superior cava or one of the innominate veins, and causes extreme narrowing or even obliteration of the vessel. In either case there may be a consecutive thrombosis of the jugular and other tributary veins.

The clinical effects of these lesions are sometimes very marked. There may be great œdema of the arm and hand on one side or on both. The neck and the face are often enormously swollen and of a deep red colour, with obvious distension of the veins. When the obstruction is limited to the superior cava, a collateral channel for the passage of the blood is afforded by the azygos vein; but when both innominate veins are blocked, the intrathoracic vessels can do little towards carrying on the circulation, which then depends chiefly on anastomoses between the superficial veins of the chest and back with those of the lower part of the trunk. The consequence is that the body becomes covered with dilated and tortuous vessels, and may acquire a deep purple colour. We can make out in which direction the blood flows, for when one of the veins is emptied by pressure along its course it fills far more rapidly from above downwards than from below upwards; or, by passing a piece of string round the chest, we may see at once that the vessels above it remain full, whereas those below it become empty. In many cases, however, the appearance of the affected parts is in itself a sufficient indication; for in consequence of the presence of valves in their interior, large and obstructed veins become far more tortuous when the blood-stream runs through them in the reverse, than when it is in the natural direction. Thus, when the superior cava is blocked, the veins may be zigzagged and varicose all over the chest and upper part of the back, whereas those over the lower part of the body may be little larger than natural, and almost straight. If there is obliteration of the inferior cava, on the other hand, the effect is often exactly the converse. At the end of Sir Thomas Watson's sixty-third lecture, he recorded two cases in which this distinction was very manifest.

Obstruction of the trachea or bronchi is another effect of compression by mediastinal growths. The records of necropsies at Guy's Hospital contain very few cases in which these parts were found untouched by the disease; but it is sometimes noted that although their walls, even to the mucous membrane, were infiltrated, there was yet no narrowing of their calibre.

In most cases the growth extends along the bronchi into the pulmonary tissue. Sometimes it fills up a great part of the lung, forming large masses in its interior, and even reaching its surface. This pulmonary invasion is either confined to one side, or at least more marked on one side than on the other. Douglas Powell says that the left lung is more often invaded than the right, but among twenty-six cases taken from the records of autopsies at Guy's Hospital the numbers on each side are nearly equal.

There is generally more or less *dyspnœa* from the time when the patient first notices that anything is amiss with him. Very often he is obliged to

sit up, even at night; sometimes the only position in which he can sleep is leaning forwards, or lying over on his face. The breathing is generally quickened to twenty-four or thirty in the minute.

Pain is often an early symptom, and occasionally, though not as a rule, it is very severe. It may be referred either to the side, or to the shoulder, or to the front of the chest. Sir Risdon Bennett speaks of it as sometimes sudden and transitory on physical exertion (Lumleian Lectures on "Intra-thoracic Growths," p. 179), particularly when the patient refers to the attack as his earliest symptom. The same author says, with reference to the later symptoms, "the amount of mere pain is seldom such as to call for the use of any large quantity of opium, but the distress is often very great."

Most patients have more or less *cough*, which is often constant and distressing. In most cases it is "ineffectual," giving rise to little or no expectoration; but sometimes a viscid mucus is brought up, and occasionally this contains blood so intimately mixed with it as to give the appearance of red currant jelly. The spitting of pure blood is much less frequent, but it may occur at the very commencement of the clinical history of the case, and more than a year before the fatal termination. Profuse hæmoptysis is seldom observed, but Sir William Church recorded in the 'Pathological Transactions' (vol. xix, p. 64) one instance in which four pints of blood were brought up immediately before death; the bleeding probably came from broken-down lung tissue in the neighbourhood of the growth, rather than from the growth itself. In many cases there is no hæmoptysis from first to last.

Cachexia is by no means a constant symptom. The patient often looks well long after he comes under treatment, and even at the last there is not often extreme emaciation. *Pyrexia* is generally absent, but Risdon Bennett relates a case (*loc. cit.*, p. 121) in which the temperature had varied from 100° to 101·4°, but in which Dr Sutton, who made the autopsy, could detect no appreciable inflammatory changes, so that the only way of accounting for the febrile disturbance was by referring it to the active cell-growth that had been going on, not only in the lungs, but also in other organs of the body.

Among occasional symptoms of mediastinal tumours, it may be noted that in two cases of intra-thoracic dermoid cysts the patient expectorated hairs, in one of them for twelve years before death.

It may be that no symptoms are present at any stage of the case, which is cut short by disease of some other part of the body, most frequently by a similar tumour of the brain. Of this, several instances have occurred at Guy's Hospital, and they show how little dependence can be placed upon the duration of pulmonary symptoms as an indication for prognosis.

Physical signs.—These depend on the size of the mediastinal tumour, and the degree of its pressure on the air-passages and the lungs. The bulk is sometimes enormous; in one case recorded by Dr Fagge, the solid mass weighed ten pounds, and in another the length, breadth, and thickness, were ten inches, seven inches, and five inches respectively. Such tumours naturally cause enlargement of that side of the chest which they principally occupy. The intercostal spaces may be widened, and the movements of the ribs much impaired. On the other hand, if the growth leads to shrinking of the lung, the measurements may be less than those of the opposite side. In some cases the tumour protrudes above the clavicle, so as to be felt at

the root of the neck ; in others it bulges through one or more of the intercostal spaces. It may lead to some absorption of bone, but it does not appear as a rounded swelling, projecting far beyond the natural level of the ribs, as aneurysm does. It may, however, pulsate more or less forcibly without true expansion, and a systolic murmur may be transmitted through it from the heart or the aorta.

As may be supposed, such large growths as these cause great dullness on percussion. This is usually most marked over the sternum, and for a greater or less distance on each side of it. But it may extend over the whole of the front of the chest on one side up to the clavicle, or it may be apparent only over one scapula, or between the scapulæ, or over one base. Occasionally the whole of one half of the chest from apex to base, including the axillary region, is as uniformly dull as if there were hydrothorax. Not only is the percussion-sound absolutely toneless, but the sense of resistance to the finger may be extreme. The condition may therefore be exactly like that which would be produced by liquid effusion ; and as a matter of fact such an error of diagnosis has been often committed, and repeated exploratory punctures have been made, which have led to no good result. The best way to avoid this blunder is to map out carefully and mark with ink or an aniline pencil the area of dullness in all directions, when it will be found to extend beyond the bounds of pleural effusion. This is one of the cases in which it is sometimes useful to employ auscultatory percussion—*i. e.* for an assistant to percuss the chest while the auscultator listens to the sound conveyed through a stethoscope.

It must, however, be remembered, that when a thoracic tumour is present, effusion as a rule follows, and sometimes in large quantity. Hence the success of a paracentesis is no warrant for resting satisfied with the diagnosis of pleurisy, for the effusion may be merely a complication of a far more grave disease. If there is altered blood in the liquid, giving to it a reddish tint, the presence of a tumour is almost certain. But in some cases of mediastinal tumour the pleural effusion is straw-coloured, and exactly like that of ordinary hydrothorax.

It must also be borne in mind that there are cases, especially where the growth is limited to the root of the lung, in which percussion yields negative results.

The other physical signs of mediastinal tumours are very uncertain. Tactile vibration is sometimes increased, but more often lessened or abolished. In the latter case the disease is likely to be mistaken for pleural effusion. There may be more or less loud bronchial breathing, or a faint vesicular murmur may be audible when the patient inspires deeply, or there may be absolute silence. Stridor and sibilant rhonchi are frequently heard, produced by narrowing of the lower air-passages by the pressure of a tumour. When the bronchial tubes are invaded, there may be not only bronchitis but secondary bronchiectasis produced, and this may cause rattles and the other signs described above (vol. i, p. 1071).

Effects on other organs.—In many cases mediastinal growths invade the pericardium, generally from the base of the heart ; and may spread downwards along the parietal layer or in the walls of one or both of the auricles. The pericardial sac may either become closed by adhesions, or distended with liquid effusion, which is often sanguineous ; or inflammation may ensue, with the exudation of lymph or of lymph and pus.

Of the great vessels, the vena cava superior is often narrowed, as has

already been stated, and the main divisions of the pulmonary artery or of the pulmonary veins may be pressed upon, so that their calibre is much reduced. It does not appear that these cases are marked by more intense dyspnoea than others.

The heart is often much displaced by a mediastinal growth; generally it is pushed downwards and to the right or the left; but sometimes, when there is shrinking and contraction of the left lung, it is dragged up so as to be felt pulsating not far below the clavicle. Although the aorta itself resists the pressure of the growth, it may happen that some of its branches are more or less narrowed, causing the radial pulse to be weaker and smaller on one side than on the other.

The œsophagus does not seem to be often occluded by mediastinal growths; at least in the reports of autopsies at Guy's Hospital there are very few instances in which this was observed, or in which dysphagia was noted among the symptoms. One patient brought up his food almost directly after attempting to swallow it; and at the autopsy the œsophagus was found pushed aside, but not invaded.

In some rare instances a mediastinal malignant growth extends into the spinal canal, and causes paraplegia by compression of the cord.

One curious effect of a mediastinal growth observed many years ago in Guy's Hospital, was apparently due to pressure upon vaso-motor nerves. A woman, aged twenty-six, was admitted, in the summer of 1866, for a defective state of the circulation in one hand, the fingers being blue, cold, shrunken, and also very painful and tender. After a few weeks she was discharged, but she again came in the following April, and died some months later. At the autopsy it was found that a growth infiltrated the fibrous tissue in front of the spine, and involved the first dorsal nerve.

In other cases, when there is pressure upon the sympathetic on one side at the root of the neck, the third nerve is no longer antagonised, and the corresponding pupil is smaller than the opposite one, as is best seen when but little light reaches the eyes. This was the case in a patient of the writer's in 1898, and, like the inequality of the pulses, may lead to a wrong diagnosis of aneurysm. In three cases of mediastinal growth, Rossbach observed that both pupils dilated regularly with each inspiration; in two of these cases, in which there was a swelling above the clavicle, pressure upon this tumour caused the pupils to become widely dilated, while the frequency of the pulse was also altered, being retarded in one instance and quickened in the other.

Diagnosis of mediastinal tumours.—As in other instances, the recognition of this group of diseases must be based, not upon supposed pathognomonic signs or symptoms, but on careful weighing of the probabilities of each case in the light of pathology and experience. Thus we must remember that acute mediastinal inflammation goes with local or general sepsis, and that the chronic form must be distinguished from the pleurisy or pericarditis with which it is often associated. We must think of new growths and enlarged lymph-glands in cases of pleuritic effusion and of thickened pleura, in cases of tuberculosis, of Hodgkin's disease, of cirrhosis of the lung, of bronchiectasis, of pericarditis, of syphilis, and of Bright's disease. The distinction between intra-thoracic tumours and aortic aneurysms will be considered again under the latter head; but it may here be noted that a younger age, the female sex, the presence of pyrexia, the

absence of syphilis, and above all the presence of pleural effusion or enlarged lymph-glands, are in favour of tumour and against aneurysm.

Of the secondary affections, the most important are enlargement of the lymph-glands, particularly in the posterior triangle and in the axilla, bronchiectasis, and secondary hydrothorax or more active pleuritic effusion—which must be carefully looked for, or it may be easily overlooked.

The physical signs of a mediastinal tumour are not very distinctive, and care is needed to distinguish it from pericardial effusion, from aneurysm, and from the chronic hypertrophic or acute suppurating forms of inflammation in this region.

Dulness on percussion over the upper or lower parts of the sternum, extending laterally to the cardiac dulness on the left and the hepatic dulness on the right and downwards, is accompanied by absence of breath-sounds, and damping or obliteration of cardiac sounds over the same area. Vocal resonance also is abolished where the dulness encroaches on the left lung. Auscultatory signs are usually less marked; most often there is in the dull region mere loss of those normally heard; but occasionally bronchial breathing may be detected. If a tumour or enlarged gland is compressing the trachea or one of the main bronchi, marked tubular or amphoric breathing may be heard over the sternum or between the scapulæ; if the aorta or one of its primary branches is involved, a systolic murmur is produced and conducted to the surface by the arterial tunics from just beyond the point of compression, *i. e.* it will be audible in the neck beneath the right clavicle, or in the back opposite the third to the fifth dorsal spine.

Pressure on the cava or azygos veins does not give rise to murmurs; but the most characteristic, perhaps, of the signs of mediastinal tumour is oedema of the face, of one arm, or of the lower limbs, without renal or cardiac dropsy; or in the more chronic cases the *caput Medusæ* formed by tortuous and dilated veins over the chest and abdomen (p. 152). The tumour may cause caries of the sternum, ribs, or vertebræ, stenosis of the trachea and bronchi or the great veins, displacement of the heart or the aorta, and occasionally paralysis of the sympathetic trunk or the recurrent laryngeal branch of the vagus.

In the most chronic cases also, as in chronic inflammation of the same region, the fingers are often clubbed and the finger-nails convex and incurved. Cyanosis is another diagnostic sign of value.

Cough and dyspnoea seem to be the most constant symptoms, and pleurisy the most frequent complication. Hæmoptysis is less common, and when present, is seldom profuse.

Ætiology.—With regard to the *causes* of mediastinal growths very little is known. In a few recorded instances they have been attributed to injuries, such as blows upon the sternum; but it may well be doubted whether this is more than a coincidence.

In a remarkable paper by Hesse in the 'Archiv der Heilkunde' for 1878, it was stated that in the mines of the Schneeberg 75 per cent. of all the miners—from twenty-one to twenty-four each year—die, generally about the age of forty, from "cancer of the lungs," spreading from the root.* Microscopically examined, the growth proved to be a lympho-sarcoma. One can only suppose that there was a tendency inherited and transmitted from generation to generation, for the miners of the Schnee-

* Nothing in the minerals worked (bismuth, cobalt, and nickel, with some arsenic and sulphur) nor in the miners' surroundings appeared to explain this mortality.

berg are for the most part sons of former miners. Walshe mentions the cases of two brothers who were each affected with intra-thoracic growths, and a parallel instance will presently be mentioned; but certainly most cases of malignant disease of the lungs and thorax, as seen in England, are not hereditary.

It is believed that more men than women die of mediastinal growths, and this was confirmed by thirty-three cases collected from the *post-mortem* records of Guy's Hospital by Dr Fagge, in which the proportion was more than two to one. As regards age, the numbers for each decennial period from twenty to sixty were almost exactly the same. It is generally stated that the disease is more frequent in persons between twenty and forty than in those who are older. A few cases have been observed in children. In eleven unpublished cases under the writer's care in which an autopsy was obtained, there were 8 men to 3 women: 1 was a boy of twelve, 4 between thirty and forty, 2 between forty and fifty, 2 between fifty and sixty, and 1 sixty-nine.

Prognosis.—In cases of mediastinal tumour the forecast is always very grave. If recovery should take place, one would suppose that a mistake had been made, and that the disease had really been chronic inflammation of the mediastinum, or syphilitic thickening of the lung or pleura. But the following case goes some way towards establishing a different conclusion.

A man named John Bullions was admitted into Guy's Hospital under Dr Habershon on February 1st, 1867, with loss of voice, stridulous breathing, great swelling of the neck, œdema of the chest, and fulness of the veins. There was also slight deficiency of resonance on percussion over the right apex and over the root of the right lung behind. Under the administration of iodide of potassium he rapidly improved, and left the hospital on March 2nd, after which he returned to work. But on May 22nd he was readmitted with what was apparently an attack of erysipelas of the face and neck. This also quickly subsided, and from that time we lost sight of him. However, in 1871 another man, named Thomas Bullions, aged nineteen, came into the hospital and died of a mediastinal new growth, as was proved by the autopsy. Struck by his peculiar name, we inquired and found that the former patient was his elder brother, and was then in good health, though still rather short of breath. Were both cases of the same nature, like those of the Schneeberg miners, and of the two brothers recorded by Walshe, or were they different?

In nine cases the duration of the symptoms varied from two and a half to eleven months. The extremes in each direction are probably afforded by two cases cited by Hertz, in 'Ziemssen's Handbuch:' one which proved fatal in a week from the first appearance of symptoms; the other in which life was prolonged for at least seven years.

In the *treatment* of this disease a faint hope of cure is afforded by the administration of iodide of potassium, mercury, or arsenic. When there is great venous obstruction, marked relief is often afforded by venesection, cupping, or leeches. To ease pain recourse may be had to blisters or belladonna, and also to various internal anodynes; for the cough, Risdon Bennett recommended antimony in small doses, with a sedative.

MALIGNANT DISEASE OF THE LUNGS.—We have seen in other cases, and shall see again, that it is a mistake to suppose that the same diseases affect all the organs and tissues of the body indiscriminately.

Even a common cause, such as the contagium of scarlatina, produces very different results upon the skin, the throat, and the kidneys. Even tubercle causes very dissimilar effects, as seen in the lungs, the lymph-glands, the skin, the bones, and the adrenals. Moreover, while each organ

and tissue impresses its own character on the results of a common cause—traumatic, degenerative, or infective—there are some diseases to which certain parts of the body are prone, and to which others are rebellious or even immune.

It was natural when morbid anatomy was first systematically studied by Laennec, Cruveilhier, and the other masters of the French school, and afterwards by Rokitansky in Vienna, that diseases should be described as certain morbid changes observed in each of the several organs. Thus we find in the great work of the German pathologist, hypertrophy and atrophy, degenerations, inflammation, suppuration, and the several varieties of malignant disease appearing in identical order in the successive chapters. But many of the compartments thus provided are now known to be empty. There is no primary "idiopathic" inflammation of the brain, no fatty degeneration of the lungs, no hypertrophy of the testis, no cancer of muscles. And even acute inflammation or tubercle or carcinoma is so different a thing when it affects the lungs and the spinal cord, the intestines and the meninges, the mammæ and the tongue, that we cannot but regard the organ affected as at least of equal importance with the "disease."

In all treatises on morbid anatomy, a section on cancer appears in the chapter on diseases of the lung; and in clinical works there has been a tendency to describe the symptoms of cancer of the lung, not so much by unprejudiced observation as by combining the well-known characters of carcinoma as it affects the mammæ or the uterus, with the well-known symptoms of bronchitis, pneumonia, and phthisis.

It is, however, certain that malignant disease of the lung is rarely met with, and that the great majority of cases are secondary, consisting of invasion of both lungs by numberless nodules, which have been transported to the pulmonary capillaries from a primary cancer of the breast or stomach or rectum. These cases have no special symptoms; the infection of the lung only occurs towards the end of the pathological scene, and only hastens the inevitable result. Such disseminated pulmonary cancer may be compared with secondary pyæmic abscesses of the lung—of much pathological interest, but small clinical importance.

The primary cases are almost confined to Sarcoma, and even these seldom take their origin in the pulmonary tissue—they begin for the most part in the mediastinal lymph-glands or thymus, or in the bony walls of the thorax.

So also the symptoms of malignant disease of the lungs are neither constant nor characteristic. Very rarely is there severe local pain, extensive destruction of tissue, hæmorrhage, foul sputa, or discharge of any distinctive histological elements.

Hence the clinical alliance of pulmonary, and other intra-thoracic tumours is more close with saccular aneurysm than with cancer of the testis or liver.

The mode of death varies. In several cases pneumonia of the unaffected lung has ended the disease; in some others gangrene; and in one case of the writer's, the immediate cause of death was acute and severe œdema of the glottis, or rather of the false vocal cords and aryteno-epiglottidean folds of the larynx. In one case the aorta was perforated (Dr Hale White, 'Path. Trans.,' May, 1893); in another (Dr Taylor, 'Guy's Hosp. Rep.,' 1892, xxxiv, p. 130) the malignant growth suppurated, and thus the case closely simulated empyema.

The following is a case of primary growth of the lung which came under the writer's care.

George P—, æt. 57, was admitted into Philip Ward June 14th, 1898. "He has suffered from a winter cough for several years, but only four months ago began to suffer from shortness of breath. This was followed by pain in the chest and marked aggravation of his cough, particularly when he lay on his left side. In the last five months he has lost about twenty pounds. He has only occasionally brought up a speck of dark blood with the muco-purulent sputa, and no blood was seen after admission. Tubercle bacilli were absent.

There was no fever; nor dyspnoea while at rest. The legs were swollen, the heart and urine normal. The right side of the chest scarcely moved, and was dull throughout, with deficient entry of air; the left lung hypertrophied, with displacement of the heart towards the right. Rhonchi were heard over the right lung, and at one point in the second intercostal space there was tubular breathing and pectoriloquy. There was no diminution of tactile vocal fremitus over the right side. Breath-sounds on the left side of normal quality but exaggerated, with occasional stridor heard on both sides, but probably produced on the right. The liver was enlarged, probably from passive congestion, the jugular veins were distended and pulsated with the heart. The urine was healthy. The right side of the chest had been punctured before admission with negative result. The rectum and the larynx were examined and found free from disease.

It was clear that the right lung was more or less solidified, with no pleural effusion, and that the case was not one of either phthisis or aneurysm. The diagnosis appeared to lie between cirrhosis, with thickened pleura and bronchiectasis, and primary new growth. The short duration of the illness, the character of the sputa, and absence of more significant phthisical signs were against the former view, the absence of glandular enlargement against the latter. A small tumour on the back below the left scapula looked like a sebaceous cyst, but it had no sign of a duct, and had dilated venules, so that Dr. Bitt, who had sent in the man from the out-patient room, justly regarded the presence of this little tumour as strong evidence in favour of the disease of the right lung being malignant. The diagnosis arrived at was primary intra-thoracic growth, probably sarcoma affecting the right lung.

There was some improvement of the symptoms for a time, but the patient continued to lose flesh, and his breath was sometimes very bad at night. On June 28th he was suddenly attacked with severe dyspnoea; he was bled and oxygen was inhaled, but he died in about a quarter of an hour, the heart continuing to beat for a short time after respiration ceased.

Post mortem a large tumour was found pressing on the right bronchus. It grew from the root of the lung, projected into the pericardium, and invaded the greater part of the right lung. The pleura was everywhere adherent, the tubes dilated in the upper lobe, the new growth caseous and softening in places. The mediastinal glands were involved, as also those of the mesentery, and there was one secondary nodule in the left kidney. The liver showed a nutmeg appearance on section, and weighed 77 oz., but was otherwise unaffected. The heart weighed 12 oz., and the right side was somewhat enlarged."

The patient often, up to the time of his death, shows no indication of more than general cachexia, but at the autopsy the lungs are found full of nodules of new growth. Sometimes cough, dyspnoea, or, in exceptional cases, hæmoptysis draws attention to the state of the lungs; and physical signs of a tumour are discovered, and perhaps of effusion on one or both sides.

Such pulmonary growths are, however, almost always secondary. The lungs are the most natural seat for all forms of secondary growth when infection takes place by the blood-current, except when the primary tumour lies within the area of the portal system of vessels, or when infection is through the lymphatics, or by direct extension, as from the mamma.

In most cases of pulmonary cancer, the breast or the stomach or some other part is known to be the seat of malignant disease. But, as sometimes happens in the case of the liver and of the brain, a patient may die with symptoms of cancer in the lungs, and it is discovered for the first time at the autopsy that this was secondary to some primary growth in a distant organ. Thus a young man once came into Guy's Hospital under Dr Frederick Taylor with what appeared to be acute bronchitis, and died in a

few days. At the autopsy it was found that the lungs were full of sarcomatous masses, secondary to a like affection of the testis. Multiple melanotic sarcoma of the lung is occasionally met with; in one case it was secondary to a pigmented mole of the skin (Guy's Museum, No. 334), in another to primary growth in the eyeball (*ibid.*, No. 335).

A remarkable case of multiple sarcoma of the lungs, chiefly affecting the surface, but not causing pleurisy, occurred in a woman aged sixty-four, who died in Miriam Ward, November 29th, 1886. There were malignant growths in the vertebræ, liver, and kidney, and a mass of diseased glands in the neck. These were taken for tuberculous glands, and the pulmonary symptoms ascribed to phthisis by the writer, under whose care she was for a day before her death: there was considerable caseation of the bronchial lymph-glands; but the true nature of the case was chronic lympho-sarcoma, lasting at least eighteen months, and then becoming generalised. One point worthy of note was a rise of temperature from 99° to 105·8° F.

Primary growths in the lung are among pathological curiosities, and even less can be said about them than about secondary growths. Cases in which there is only a single mass of considerable size have often been described as examples of primary malignant tumour in the organ, but even these may have started from the cartilaginous rings of the bronchi or from the perichondrium. The origin and pathology of the remarkable cases recorded under the name of disseminated or miliary cancer of the lungs is obscure; they look far more like a secondary than a primary lesion.

One such was brought by Dr Fagge before the Pathological Society in 1866. The patient was a man aged fifty, who died of an illness of two or three months' duration, but only two days after being admitted into hospital, with what appeared to be capillary bronchitis complicated with some pneumonia. At the autopsy the lungs were found full of round bodies like tubercles, but larger (some as large as hemp-seeds), and of a shining white appearance. The only growths discovered elsewhere were a few (apparently also secondary) in the heart and in the liver (Guy's Hosp. Museum, No. 324).

A still more remarkable form of pulmonary cancer occurred in 1870.

A man aged thirty-six died in Guy's Hospital after two and a half months' illness, which was attributed to damp and cold, and which appeared clinically to have been pneumonia of the right lung, accompanied with much effusion into the pleura. At the autopsy the lung was found much enlarged, nearly white in colour, but somewhat mottled, smooth, and shining in appearance, soft and cushiony to the feel, so that one might have imagined it to be generally emphysematous but for the fact that it was absolutely airless, every part of it sinking instantly when put into water. At the root of the lung there was obvious new growth, which probably was seated in the glands, but which had also involved the superior vena cava, and narrowed it considerably. Unfortunately the lung was thrown away before any microscopical examination of it was made, but probably it was an example of primary diffused carcinoma of the lung, such as described by Hertz as "bearing a striking resemblance to grey hepatitis."

In a patient of Dr Frederick Taylor's (a man of forty-three), the primary disease was an epithelioma (or squamous-celled corneous cancer), and it began in the mucous membrane of the bronchus ('Guy's Hosp. Rep.,' xxxiv, p. 130). Another case of primary squamous epithelioma of the lung is described by Dr Pitt in the 'Museum Catalogue' (No. 323). The patient was a man of sixty-eight.

Pulmonary embolism.—The most common example of this terrible event occurs in puerperal cases, when the natural coagulation in the uterine veins and sinuses gives occasion for a clot to be accidentally dislodged. But this is more likely to happen when septic absorption from the parturient tract has caused more extensive coagulation in the iliac and femoral veins. It is

usually a fortnight or three weeks after delivery, on the patient's rising from bed, that a portion of the fibrinous clot becomes detached, passes through the right auricle and ventricle into the pulmonary artery, and causes almost instant death. If the clot should be smaller, and plug only one pulmonary artery or a branch, terrible dyspnœa comes on, and death ensues in a few minutes; or the patient may recover for a time and die in a second or a third recurrent attack. In other cases the detached portions are still smaller, and not being large enough to close the trunk of the vessel, stick in some of the branches, and there produce further coagulation. This proceeds until the whole lung is blocked, and death follows in a few days. In some cases, however, the patient recovers.

Such instances of slow coagulation in the lungs with lingering symptoms are much less frequent than those of sudden death from pulmonary embolism. The two following cases were of the former kind, and occurred in men.

A medical man, fifty years of age, was seized on the 3rd of March with difficulty of breathing, and on the following day was examined by Dr Wilks. The patient was panting and breathless, the respiration was very quick, and on the slightest exertion it almost ceased, as happened more than once when he attempted to get out of bed. On examination of the chest nothing abnormal could be discovered; the sounds of the heart were healthy, its action quiet, and the pulse was 80. The patient had no difficulty in taking a full breath; indeed, he felt, he said, as if he must breathe too much. A suspicion of embolism caused an examination of the whole body, when phlebitis of one leg was found, the result of an injury six months ago. A month before, he was seized with shortness of breath, which passed off after a few hours. Dr Wilks concluded from this that he had embolism of the pulmonary artery. On March 7th he was worse, and was beginning to spit up tenacious, rusty, and bloody mucus. On March 8th he was dying, gasping for breath and with great lividity of the extremities, but the heart was regular and there was no bruit. The autopsy showed embolism of both branches of the pulmonary artery. In the left was a large clot, tolerably recent, and in the right an older one adherent to the walls of the vessel. There was also pneumonic consolidation in portions of both lungs. The right femoral vein contained a thrombus of exactly like that found in the pulmonary artery.

A young man, an officer in the army, was operated on for varicocele; he rapidly recovered from the operation, and had left his bed, but had not gone out of the house. During conversation one evening, February 9th, a fortnight after the operation, he suddenly called out, fell back, and gasped for breath. It was thought he was dying. Dr Fagge saw him soon afterwards and found him cold, pulseless, and breathing heavily. About an hour afterwards he spoke; he was very pale, and the respiration was sighing, but his pulse was just perceptible. On the following morning he had rallied, his skin was warm, the breathing tranquil, and the pulse 100. February 11th.—Some oppression of breathing; temperature normal. 12th.—Slight crepitation heard over lower part of chest on left side; heart normal. 13th.—Crepitation over lower part of left lung in front and expectoration of a little bloody mucus. A slight murmur heard at base of heart. 14th.—Bruit more audible and traced up in course of pulmonary artery. 16th.—Bruit less marked and crepitation diminished. 20th.—Bruit gone and no râles heard. He continued to improve, but spat up red, glairy, transparent mucus, and when he sat up the pulmonary bruit became audible. March 15th.—Allowed to leave his bed, all physical signs having disappeared; but he still spits a little coloured mucus. He gradually recovered.

Two cases of recovery from pulmonary embolism have come under the present writer's notice: one in a patient seen with Dr Hine, at Oxford, where it followed thrombosis of the femoral vein from an injury, and was marked by hæmoptysis with a pleuritic rub; the second during recovery from pneumonia, in a patient under the care of Dr J. H. Galton. In both cases the condition was recognised from the first, and in both the diagnosis was amply confirmed.

DISEASES OF THE HEART

FUNCTIONAL DISORDERS OF THE HEART

“Arteriarum pulsus—index morborum, stabilis aut citatus aut tardus, observatione crebri aut languidi ictûs—gubernacula vitæ temperat.”—PLIN. *Nat. Hist.*, lib. xi, cap. 88.

History and general pathology of cardiac diseases—their arrangement—physiological conditions—The pulse—The sphygmograph and other instruments—Bradycardia—Tachycardia and irritable heart—Intermittent and irregular pulse—Allorhythmic pulse—Palpitation—Cardialgia—Treatment of the above conditions—Tension of the pulse—its indications—Asphyxia and syncope—its distinction from epilepsy, collapse and sunstroke—its treatment.

IN passing from the affections of the lungs to those of the heart, we enter on a chapter of medicine which is even of more recent origin. In fact, with the exception of the spinal cord, there is no organ of which the diseases and their diagnosis were until the nineteenth century so entirely unknown.

Like the lungs, the heart is inaccessible to direct observation; and like the lungs it has been revealed to us in its disordered, as well as in its healthy, action by the invention of the arts of percussion and auscultation. Harvey's great discovery of the movements of the heart and of the blood had no direct effect upon practice in the seventeenth and eighteenth centuries. Sufficient physiological knowledge of the circulation existed, but it was almost useless for want of satisfactory methods of investigation.

The study of the pulse is of very ancient date, but its indications were scarcely better understood by European physicians ninety years ago than by those of ancient Greece or modern China.

The variations of the pulse were familiar to the ancients, as is shown by the quotation from Pliny at the head of the present chapter; but the pulse was not counted until long after the invention and common use of watches. Its changes were all referred to such general conditions as fever, and not to disease of the heart.

The chief symptoms belonging to the circulation which were recognised before 1810 were Palpitation, Syncope, and heartburn or Cardialgia, otherwise known as *passio cardiaca*, together with the rare and terrible malady *Angina pectoris*, or breast-pang, first described by Heberden in 1764. The only anatomical lesion of the heart recognised (beside Peri-

carditis with hydropericardium) was general enlargement; and this, as well as the cardiac symptoms just mentioned, was supposed to be the result of disturbed passions and mental emotions, not as we now know it to be, the direct consequence of mechanical derangement.

The most striking and universal of the effects of organic disease of the heart, dropsy, was by the best physicians at home and abroad at the period indicated, that of the reign of Napoleon, regarded as a substantive and single disease, to be distinguished from obesity and flatulence; and not as a mere symptom of antecedent lesions of the heart or kidneys or liver. Indeed, the valvular defects on which dropsy follows as a natural hydraulic result were not even suspected.

Auenbrugger in 1761 (cf. vol. i, p. 1021) only mentioned incidentally the dulness of note heard on percussion over the heart. His translator, Corvisart, in his essay on the 'Diseases of the Heart and Great Vessels,' published in 1810, made the first serious attempt to describe the morbid anatomy of these organs. Diseases of the pericardium, aneurysm of the aorta, dilatation and hypertrophy of the several cardiac chambers, and contraction of its orifices were the lesions that he described.

Laennec's discovery of auscultation began with auscultation of the heart when, in 1816, at the Neckar Hospital, he used an improvised stethoscope and first heard the sounds of the human heart (cf. vol. i, p. 1020). He laid the foundation of cardiac semeiology; but while his account of the physical signs of diseases of the lungs left little to correct, and often little to add, his investigation of the heart was far less successful.*

He recognised as the ordinary valvular lesion, narrowing and consequent obstruction of the cardiac orifices; but the leaking produced by a damaged valve was not understood until long after. Hypertrophy and dilatation he regarded as primary diseases, and their relation to valvular lesions was not apprehended. He was naturally struck by the remarkable diversity of the sounds heard on listening to a heart when diseased, and described and named the most remarkable by happy comparisons which we use to this day. But he could not know of how small importance the *bruit d'oboe* and the *bruit de scie* would prove in comparison with the rhythm of these sounds and with their locality.

Laennec supposed both sounds of the heart to be of muscular origin; the one due to contraction of the ventricles, the other to that of the auricle. He had not discovered the means of distinguishing the exact seat and nature of the lesions by means of the position and the time of the abnormal sound. When the experiments of Hope and C. J. B. Williams, and the clinical investigations of Skoda and Corrigan had laid the foundation of accurate valvular pathology, it was still believed that endocarditis, like pericarditis, produced a sound of itself; while the presence of anæmic and other functional bruits still prevented the mechanism of those produced by valvular lesions from being understood. In 1845 Dr Latham, of St Bartholomew's, taught that murmurs are distinct from the natural sounds of the heart and differently produced; he thought they resulted from unusual

* This seems to have chiefly depended upon imperfect knowledge of the mechanism of the heart in health. The action of the valves, though understood by Harvey, was scarcely appreciated until the experiments of Hope and Williams. Bouilland, Donné, and Fauvel never grasped the effect of a damaged valve in leading to regurgitation of the blood into the cavity from which it had just flowed. So late as 1864, when the present writer was a student in Paris, one member of the Faculty of Medicine taught that the first sound of the heart coincided with the diastole and the second with the systole.

vibrations communicated to the particles of the blood by obstacles which it encounters in its passage through the heart. He understood, however, the doctrine of regurgitation.

In spite of his errors, Laennec's discovery of the normal cardiac sounds, and of the fact that they became altered or replaced in disease, furnished the key to the problem. The work of associating the several abnormal sounds with the various valvular lesions was continued by his successors in France; and in this country by Corrigan (1829), Hodgkin (1829), Hope (1832), C. J. B. Williams (1835), Latham (1845), Hughes (1845), Walshe (1851), Billing (1852), Stokes (1854), and Gairdner (1861); by Austin Flint (1860) and Da Costa (1864) in America; and in Germany by many investigators, of whom Skoda, of Vienna (1850), and Traube, of Berlin (1867), were perhaps the most eminent.

In arranging this section, we will first deal with functional disorders of the circulation, and the various kinds of pulse. The remarkable disease known as *Angina pectoris* will find a place in the following chapter.

Passing next to organic diseases of the heart, we will take first those of the pericardium, then changes in the muscular substance, next those of its lining membrane, with the important subject of valvular lesions; and last will come a chapter on aneurysms of the aorta.

Of all known diseases, those of the heart are most demonstrably dependent on physical causes. The most common and typical "heart disease" depends upon damage to the valves, which interferes with the circulation exactly as it would in an artificial machine, and which can be imitated in the "schema" of the circulation used in physiological lectures. The next most frequent and important division, occurring usually as the result of the first, is dilatation of the cavities and hypertrophy of their walls. These are both the consequence of increased blood-pressure: the former its mechanical effect, the latter the physiological response to it. Degeneration of the muscular walls of the heart directly enfeebles its action as the chief motor power of the circulation. Inflammation of the pericardium, and particularly pericardial effusion, mechanically interferes with the movements of the heart.

In all these cases, speaking generally—whether the primary disease affects the orifices of the valves, the size of the cavities, or the contractions of the left ventricle—the effect is the same: to impair the action of the heart as a force-pump, to diminish the blood-pressure in the aorta, and to produce a condition throughout the whole systemic circulation of arterial anæmia and venous congestion.

There are, however, two physical conditions which influence the action of the heart, apart from mechanical changes in the left ventricle.

One of these is the state of the peripheral channels of the capillaries and arterioles. If there is narrowing of these passages by contraction of the vaso-motor nerves (or, possibly, by contraction of the protoplasmic plates of endothelium which line the capillaries), the direct hydraulic effect is to raise the pressure between the area of increased friction and the heart, so long as the ventricular contractions remain the same; whereas if the normal calibre of the systemic peripheral vessels be increased, the pressure of blood in the arteries will fall, so long as the energy of the cardiac impulse is not increased. The elasticity of the arteries stores up the energy of the left ventricle during its diastole, and makes the flow of blood more

uniform ; but it contributes nothing to the forces of the circulation. Nor does the muscular coat of the arteries : the only auxiliary forces are the pressure of the muscles of the limbs on the veins, the aspiration of the thorax, and the active diastole of the left auricle and ventricle. The calibre of the arteries is regulated by the vaso-motor nerves (and in some regions by vaso-dilator nerves as well), but these seldom, if ever, act on the whole systemic circulation at once. The great splanchnic area, the muscles, the glands, and the brain are the most important vascular territories, and when one is rendered anæmic by contraction of its arteries, some or all of the others are flooded by relaxation of theirs.

The lesser circulation is governed by the same forces, except that it is still doubtful whether there are any vaso-motor nerves distributed to the pulmonary artery and its branches.

The second regulating nervous mechanism of the circulation is the inhibitory effect upon the left ventricle of the cardiac branches of the vagus, and the stimulating effect of the cardiac excitator nerves from the sympathetic trunk, which are derived ultimately from the grey matter of the bulb.

The ultimate force of the circulation is nothing else than the energy derived from oxydisable food, used as active energy by the muscle of the heart, just as by the muscle of a man's arm working a force-pump. The immediate force is that of muscular contraction. The rhythm of the heart is certainly independent of the central nervous system, and does not, according to the latest and most trustworthy observations, depend upon reflex action, nor upon the presence of intrinsic cardiac ganglia at all, but is an inherent primary endowment of the muscular fibres. The part of the nervous system is only to regulate (or in pathological conditions to disturb) the cardiac rhythm.

DISORDERS OF THE PULSE.—The *frequency* and the *rhythm* of the radial pulse correspond with the frequency and rhythm of the cardiac contractions ; and the number of contractions of the left ventricle is, in disease as in health, the same as that of the right and of the two auricles. It can be readily increased from the usual seventy beats a minute by muscular exertion or by mental emotion. It is much less readily reduced below its wonted rate, but the horizontal posture, cold, and insufficient nourishment tend in this direction. Many healthy persons have a naturally slow pulse, not exceeding 60 in the minute. In old age, with inelastic and dilated arteries, the pulse is, as a rule, large and somewhat below 70 in rate.

In disease some of the ventricular contractions may be too weak to be felt as a pulse-wave in the radial artery, and hence there ensues an apparent difference in rate between the heart and the pulse.

The rhythm of the heart is almost as easily altered as its rate. Thus running or other exertion, at least in those who are untrained, produces an irregular as well as an accelerated cardiac action and radial pulse. This, however, is an effect which is only transient, and is thereby distinguished from a pathological condition.

The *size* or volume of the pulse depends partly on the natural size of the artery in the individual ; but also in each person upon the "fulness" of the vessel, *i. e.* on the force of the left ventricle at the time, and the degree of contraction or relaxation of the arterial tunics.

The pulse of old age and of premature arterial degeneration is a large

one, the arterial tunics having yielded to the blood-pressure (like an old glove); but we must not forget that the same pressure operates also lengthwise, and makes the senile artery longer as well as wider than before; hence the tortuosity of the inelastic artery, as of the varicose vein.

The *strength* or force of the pulse (in systole), as appreciated by the eye or by the compressing finger, is due to the energy of the ventricular contraction. The apparent strength of the pulse is, however, diminished, even with a vigorous ventricular action, if the coats of the artery are less yielding than usual, or if the peripheral resistance is unusually high; while its apparent strength is increased (even with a weak ventricular contraction) if the "tension" of the artery is low, *i. e.* if the arterial walls readily expand in systole and collapse in diastole. In other words, the finger appreciates, not the absolute force of the ventricle, but the difference between the artery when expanded by the pulse-wave which follows cardiac systole, and when more or less collapsed during cardiac diastole.

The *length* or duration of the pulse depends on the length of the cardiac systole, but also on the tension or relaxation of the artery, its walls yielding slowly or easily to the wave of increased blood-pressure.

The *tension* of the artery is the degree in which it yields to the compressing finger. If readily obliterated, it is said to be soft and of low tension; if firm and unyielding when squeezed or rolled under the finger, it is termed hard, wiry, or cord-like.

It is sometimes difficult to separate the passive strength of the artery in resisting compression from the active strength in lifting the observer's finger. The first belongs to the (cardiac) diastole, the second to systole, but it requires some experience to disentangle the sensations. A hard and strong pulse also is often called a large one.

Another difficulty is to distinguish between the firmness of a vessel due to distension with fluid contents (the artery of "high tension" or high blood-pressure), and the hardness of a thick-walled artery, which would still be felt when it was empty after death; the difficulty is not lessened by the fact that the two conditions may go together. The feeling to the finger of a thick-walled artery is like that of whipcord, but when the arterial tissues are calcified, the resistance is inelastic; it feels hard not tough, "like a tobacco-pipe," not like a tendon.

To estimate the tension of an artery it should be first rolled under two fingers, so as to judge of its size and of the character of its coats. Then the forefinger should be gradually pressed on the artery, until the middle finger just below it no longer feels pulsation. It is well for the ring finger to be pressed firmly still lower down, so as to prevent any regurgitant pulsation due to free anastomosis through the deep palmar arch.

The sphygmograph.—Since the graphic method of registering the mark made by a moving index on a recording surface was introduced into the physiological laboratory by Ludwig,* attempts have been made to register the arterial pulse, either by tambours connected by flexible tubing, or by a lever and style writing on a blackened card, which is moved rapidly past in a straight line by clockwork.† The form most generally used, and, on the

* The method was taken from an invention of the famous engineer James Watt.

† Many years ago Wilkinson King attached a bristle to the skin over an artery by means of wax, to show the movements of the pulse. If this bristle, which King called the "sphygmometer," had been made to take tracings, it would have been a rude sphygmograph.

whole, most useful, was devised by Marey.* The ingenious modification of the instrument by Dr Dudgeon, though cheap, and convenient, is more open to fallacies. Dr Waller's digital sphygmograph is a better form.

The sphygmographic tracing, when skilfully taken on a normal artery, shows a series of waves of equal height and force, recurring at equal intervals. Each wave should correspond with the changes in the diameter of the artery produced in one cardiac cycle. The upstroke shows the expansion of the vessel by the wave of pressure, from the systole of the left ventricle; while its degree of slope (supposing the rate of the moving card to be the same) denotes the tension or resistance of the artery; in other words, its rapid or more gradual expansion.

The apex of the wave is formed by the inertia of the lever carrying the writing point a little higher than the expanding artery does. It is known by its sharpness, and by a rounded shoulder following it on the downstroke. By connecting this latter with the upstroke we strike out the "percussion-wave" of inertia, and obtain more or less precisely the true "tidal" wave, as it is called, which would be traced by the expansion and relaxation of the arterial wall. The breadth, or bluntness, of this tidal wave shows the length or duration of the systolic pulse; if narrow, it points to a low tension with a compressible and probably dicrotic pulse; if broad, to high blood-pressure and a hard pulse.

In the downstroke is usually seen a "notch," followed by a second minor wave. The former has been called "*the (aortic) notch*," on the supposition that it depends on closure of the left sigmoid valves checking the fall of blood-pressure in the arteries, early in diastole. The latter is called the *dicrotic* or second pulse-wave, and of course denotes increased pressure, which at this point interrupts the diastolic collapse of the artery. The difficulty of the explanation just given leads us to accept Burdon Sanderson's explanation, which refers the second rise of the lever to a back wave of pressure from the small arteries and capillaries in front, which react in virtue of their elasticity when filled by the systolic wave.

Whatever its precise origin, this dicrotic wave is small or absent when there is high arterial tension, and, as a rule, well marked when the tension is low. Sometimes it is so large that it rivals the tidal wave. If situated high up on the downstroke, the notch and second pulse-wave show an early occurrence of the reflux, and high tension of the arterial system; if far down on the descending line, they show a relaxed artery with low tension. When the second wave is very low (*i. e.* very late) and very large, the pulse is called hyperdicrotic. In such cases the second pulse-wave may rival the first, and give a tracing which looks at first sight like alternating stronger and weaker strokes of the heart.

If we draw a line from the summit of the tidal wave to the notch we have a measure of the amplitude or breadth of the pulse-wave, and so a measure of the arterial tension. When the tidal wave is well marked and broad, the tracing will fall outside this straight line, the tension is high, and the pulse feels hard. When there is no projection to be noticed, and the tracing falls directly from the apex of the percussion-wave to the notch, the tension is low and the pulse compressible. The aortic notch will come high up on the downstroke (*i. e.* early in diastole) in the former case, and low down (*i. e.* late in diastole) in the latter.

It is often difficult to get a good sphygmographic tracing, and it is only

* Marey's sphygmograph was suggested by that of Vierordt.

by trial that we find the most suitable pressure to bring out the characteristic pulse in each case. In chronic Bright's disease, in fever, in advanced mitral incompetence, in aneurysm, and in aortic regurgitation, the tracing may carry the diagnosis with it. But on the whole, the expectations raised by Marey's instrument have not been realised; and the sphygmograph is rather useful as a corrector or confirmer of a diagnosis based on other grounds, than a discoverer of unexpected lesions, like the stethoscope or laryngoscope. Sometimes we see differences between the right and left pulse, of which we could not otherwise be sure; often the indications derived from the finger receive useful modification; but probably the chief benefit of the instrument is that it has made feeling the pulse a more intelligent procedure, our nomenclature more precise, and our conclusions less individual and arbitrary. It is possible to auscultate the chest without the aid of any instrument, but it needed the invention of the stethoscope to teach auscultation; and so also, while the practised physician is almost independent of the aid of the sphygmograph, this practice, and the true *tactus eruditus*, is only to be gained by assiduous comparison of the results of palpation with the tracings of the sphygmograph.*

Sphygmometers.—An ingenious instrument invented by Dr Rayner Batten to test the tension of the pulse consists of a piston sliding in a cylinder, and provided with a spring. This piston rests on the artery instead of the compressing forefinger next the heart, and when the middle finger no longer feels the pulse, an index-lever working on a disc (like that of a weighing machine) points to the pressure in ounces, 8, 12, or 16 being the most frequent numbers.†

Various attempts have been made to measure the blood-pressure in the human subject, by von Basch in 1831, by Roy and Adami in 1890, by Mosso in 1895, and in 1899 by Hill and Barnard. This last *sphygmometer* conveys the pulsations of the radial or other suitable artery by an air-tight india-rubber tube to a dial (see figure in Schäfer's 'Text-book of Physiology,' vol. ii, pp. 79-80).

The *pulse-pressure-gauge* invented by Dr Geo. Oliver, of Harrogate, is a much more elaborate instrument, constructed on the same principle as his *arteriometer*; but whereas the latter instrument is adapted to ascertain the exact diameter, and hence the calibre, of the radial or other artery investigated both in systole and diastole, the pressure-gauge records by the movements of a lever on a circular face, the pressure of the blood in the artery at its maximum in ventricular systole and at its minimum in ventricular diastole.‡

Tachycardia and bradycardia.—Of the Functional Disorders of the Heart, we will take first acceleration of its rhythm. We estimate this by counting the pulsations at the wrist; but sometimes the radial pulse is too weak or too frequent to be counted, and only by feeling the carotids or

* See Dr Galabin's valuable papers in the 'Guy's Hospital Reports,' 3rd series, vols. xix and xx, and the account in 'Foster's Physiology'; also Burdon Sanderson's work on the subject (published in 1870). Of later writings in this country, the most important are Sir Wm. Broadbent's 'Croonian Lectures' for 1887, and Dr Oliver's 'Pulse-gauging,' 1895.

† The writer has often found this method valuable, and the obvious objection, that the springs would weaken by use, does not appear to hold in practice. Moreover, one can always check its accuracy by applying it to a healthy pulse, and comparing the pressure with that of the patient suffering from chronic cardiac or renal disease.

‡ "A Contribution to the Study of the Blood and Blood-pressure," 1901.

applying the stethoscope to the chest can we ascertain the rate at which the heart is beating.

Almost every one speaks of the pulse as being "slow" or "quick," meaning infrequent or frequent. In strictness, a slow pulse (*Pulsus tardus*) is one in which each pulsation of the heart takes more than usual time for its completion; a quick pulse (*Pulsus celer*) is one in which the ventricular contraction is short and soon over. In the present chapter, at any rate, we will employ the terms frequent (*P. frequens v. creber*), and infrequent (*P. rarus*) when the number of beats in the minute is referred to, and short (*P. brevis*) or long (*P. longus*) when we refer to the duration of the ventricular contraction and pulse-wave.

The frequency of the pulse in health is liable to wide variations in different circumstances. It is slightly greater in the young than in the old, in women than in men, in the upright posture than in sitting or lying, and after a meal than when fasting. It is much increased by exertion, by emotion, or any mental or bodily excitement. Few persons can submit to medical examination, especially for life insurance, without the heart's beats rising to a hundred.

It has been said that the pulse may permanently stand at 100 in a healthy person; but probably a pulse habitually above 80 should be regarded as evidence of some morbid condition (Latham's 'Collected Works,' New Syd. Soc., vol. ii, p. 526).

(1) An *infrequent* pulse is natural to some persons, in whom the heart never beats oftener than fifty or even than forty times in the minute; this does not appear to affect the prospect of longevity. Among the few morbid conditions which may render the pulse infrequent are aortic stenosis, when due to atheroma, loss of arterial elasticity, fatty or fibrous degeneration of the heart (occasionally), and jaundice. A *pulsus rarus* is also observed during convalescence from febrile diseases, and—though more seldom seen in women than in men—it may follow childbirth; also as the result of cold and hunger, and in the last stages of emaciation with subnormal temperature. It does not of itself indicate danger. The same infrequency of the cardiac beat is often observed during collapse, and in some cases of cerebral tumours, compression, concussion, and apoplexy.

A very infrequent pulse has been often described, when the condition is really that of a *pulsus alternans*, or, as it is sometimes called, *bigeminus*, in which the alternate weak pulsation is not noticed. Such a pulse, with every other beat "dropped," may occur in some cases of advanced atheroma and occasionally in mitral stenosis.

Frequently the same alternate pulse is produced by digitalis; and Tripier, of Lyons, believes that a slow pulse depending on a dropped beat may be often observed in epileptics.

Probably some cases of bradycardia begin as a strong, followed by a weak, pulse, and the weak cardiac contraction becomes gradually weaker until every other pulse-wave altogether fails to reach the wrist. In other cases, what is under the stimulus of food or alcohol a *pulsus alternans*, becomes when the patient is fatigued a pulse of only half the number of beats; or, to put it otherwise, Allorhythmia and Bradycardia interchange. In one case which the writer watched for more than two years, there was constant bradycardia, the radial pulsations not being above 35 or 40 as a rule, and occasionally sinking to 30 or 28 in the minute. Here the impulse and sounds of the heart were equally infrequent, so that it was true bradycardia.

The condition appears to be less rare in later adult life, and in men than in women. Its antecedents vary, but it is probably connected with fibrous rather than with fatty degeneration of the myocardium, and with arterial atheroma rather than with valvular lesion of any kind. It has also been observed after recovery from Graves's disease, a fact which may be coupled with that of moderately slow pulse, and subnormal temperature accompanying convalescence from fever.

Dr Alexander Morison has described (in his monograph on 'Cardiac Failure,' 1897) cases of bradycardia in which, from time to time, there is sudden and complete cessation of radial pulsation and cardiac action, followed by very slow pulsation, which gradually increases up to 40 or 50. This period of "asphyxia" is not always accompanied by loss of consciousness; but when it is, there is an epileptiform seizure at the same time, and not infrequently the syncope is immediately fatal.

A case in which bradycardia came on very quickly after an attack of influenza was under the writer's observation at intervals for the two years of its duration. For several months the only other symptom was shortness of breath on exertion, but there gradually supervened more and more marked cyanosis without œdema, and occasional attacks described as faintness, suffocation, or oppression on the chest. There was never any pain or anginal symptoms, and no sign of valvular lesion; but the "fainting fits" became more frequent as time went on.

On the day before his death, while in his usual health, the patient was attacked by one of these seizures while sitting at dinner, and it was then ascertained that there was sudden loss of consciousness with flushing of the face, followed after a very short interval by recovery with pallor. The following morning the last attack was equally sudden, with complete loss of consciousness, clonic spasms of the eyeballs and limbs, cyanosis, laboured breathing, and death, in less than five minutes after the patient fell insensible on the floor in his dressing-room. No autopsy was obtainable, but the clinical features were those of eclampsia, not of syncope, and the case fully confirms Dr. Morison's description.

In any case chronic bradycardia is a grave symptom, and though it may last for several years, is probably never recovered from. In the two most marked cases which have come under the writer's notice, there were occasional attacks of dyspnoea not only on going upstairs, but while lying in bed. One of them ended in sudden death, the other promises to continue for still many years.

In our ignorance of the physiology of bradycardia, no rational treatment is possible; but it has been observed that digitalis and its allies are injurious.

(2) The diseases in which the frequency of the pulse is *increased* are numberless. They include all fevers and inflammations, and the great majority of valvular affections of the heart; also exophthalmic goitre and functional cardiac disorders in general.

In these conditions the blood-pressure is usually low, and the pulse is sometimes compressible, dicrotic, and often irregular.

There are, moreover, some cases in which a very frequent action of the heart appears to constitute a disease in itself, which may be distinguished as primary or idiopathic *Tachycardia*. Three such instances were recorded in the 'Brit. Med. Journ.' for 1867 by Dr Cotton, Sir Thomas Watson, and Dr Edmunds. The patients were all males of middle age. In two of the cases there were several attacks at varying intervals, each lasting from a few hours to two or three weeks. The rate of the pulse was from 200 to 230, yet it was perfectly regular. The termination of the attacks seems always to have been absolutely sudden; in Watson's patient, on one occasion, the beats of the heart, directly after having been counted at 216, fell to 72, exactly one third of the former number. Walshe states that, in

the cases of this kind, his patients, who were women, were by no means all of them hysterical or nervous, and some were distinguished by force of character. The causes which he recognised were: pedestrian excursions, the ascent of mountains, acute pain with effort to control its manifestation, and prolonged mental distress; but often no cause could be discovered.

Tachycardia is not always devoid of danger. A sensation of faintness and dyspnœa is sometimes present; and Watson's patient died during his fourth seizure, the heart, after death, being found large, as if it had been distended, while its muscular walls were very thin and soft.

The late Dr Bristowe had a remarkable case of rapid pulse, which is quoted by Sir Wm. Broadbent in his Croonian Lectures ('Brit. Med. Journ.," 1887, vol i, p. 659). A young man, under twenty, had a pulse of 200 or 240 beats in the minute; and the beats were ineffectual, for he suffered from dropsy and hæmoptysis. This condition of tachycardia had continued in occasional paroxysms since childhood. He improved with treatment, although the pulse was liable under excitement to run up to 300. He died suddenly while playing the piano. *Post mortem*, the heart was found dilated and the valves normal.

In 1870 Dr Wilks brought under the notice of the Clinical Society certain cases in which an extreme frequency of the pulse, associated with alarming dyspnœa and with palpitation, was due to nephritis, usually of scarlatinal origin. In almost every instance recovery took place within a few days, notwithstanding the alarming character of the symptoms.

In the *treatment* of acute albuminuric tachycardia, Wilks recommends purging, cupping, and salines with antimony, rather than the administration of stimulants. In his idiopathic cases of rapid pulse in women, Walshe found digitalis of no service, but nervine tonics decidedly useful; and Dr Morison speaks well of arsenic. The present writer has found little benefit from foxglove, strophanthus, or caffeine, and not much more from strychnia. The best treatment in severe cases has seemed to be lying down for weeks together, and the most useful drugs valerian, sumbul, and bismuth.

Irritable heart.—A valuable paper by Da Costa ('Am. Journ. of Med. Sci.,' 1871) on this subject, gave good evidence that long-continued, frequent, and irregular pulsation may at last cause a condition of cardiac hypertrophy. It was based upon no fewer than three hundred cases of soldiers in the army of the United States during the Civil War. These men, having been called from civil pursuits into active service without previous training, became liable to attacks of palpitation, to more or less severe pain in the chest, either of a sharp stabbing, or of a dull aching character, and to dyspnœa on exertion, so that they became unable to keep up with their comrades, and were distressed by the weight of their accoutrements. On examination the pulse was found to be greatly increased in frequency; it was unusually altered by position, so that there was sometimes a difference of thirty beats or more in its rate between standing and lying down; in some cases it was intermittent or irregular. The men themselves often looked healthy, though their hands were apt to be blue and mottled. The cause of the disorder seemed often to be hard service in the field, particularly forced marching; but in many cases it was directly brought on by an attack of diarrhœa, or of fever. The patients were generally young men under twenty-five.

After some months of treatment, recovery, often complete, ensued; but

in other cases there was still liability to cardiac symptoms on exertion. Hypertrophy of the heart was believed to have supervened in twenty-eight cases out of two hundred.

In the treatment of these cases, the most important prescription was found to be rest. Making the patient lie down for several hours daily often led to remarkably good results, and two men who were kept in bed—one by an attack of dysentery, and the other by a broken leg—improved rapidly.

Da Costa laid stress on the maintenance of great care during convalescence. Before allowing the men to return to their regiments he tested them by running and by other exercises, so as to see how the heart bore the strain. He gives reports of some patients who came under notice again after an interval of several years, and in whom no relapse had occurred.

Intermittence of the pulse.—In many persons the heart from time to time “intermits” or leaves out one of its beats, while its action in other respects remains perfectly regular. Sometimes the intermissions take place at intervals of only a few pulsations, sometimes not oftener than once in two or three minutes; sometimes the intermission is regular, sometimes quite irregular in its recurrence. The patient is often unconscious of the interruption of the heart’s action; often he notices it only when alone or in bed, or in rare cases he may feel an instantaneous and transitory faintness. More often, what draws his attention to the fact that his heart now and then misses a beat, is that the beat which follows each intermission is thumping, so that he will come to a physician complaining of palpitation. Apart from this sensation, it may sometimes be noticed that there is really an unusual force and fulness of the pulse in the radial artery after each intermission.

Intermittence of the heart’s action should never be made light of until the state of the organ and of the great vessels has been investigated. If there be any undue arterial tension in particular, it should be carefully noted; in one well-marked instance, intermittence of the pulse, observed by Dr Fagge from time to time during three or four years, ended in disease of the aorta and its valves. There is the more need of caution if the heart’s beats begin to intermit after slight exertion, such as walking faster than usual, or hurrying to be in time for a train.

Nevertheless occasional or even habitual intermittence of the pulse is in many persons compatible with a good state of health, and with a fair prospect of longevity, so that Heberden’s dictum still holds good: “Such trivial causes will occasion them (intermittent pulses) that they are not worth regarding in any illness, unless joined with other bad signs of more moment.” In numerous cases, when for many years the pulse has always intermitted, the heart is found after death to be perfectly sound and the coronary arteries normal. Walshe believed that some people feel more comfortable when the rhythm of the heart is intermittent than when it becomes (as it sometimes will for a time) perfectly regular.

In a patient eighty-six years old, a subject of gout, the writer noticed the pulse repeatedly to intermit for three, four, or even five beats; yet there was no discomfort felt, and no appearance of anæmia or of venous fulness.

It is said that intermittence of the pulse ceases during any illness attended with pyrexia. In one case Richardson (‘Trans. St And. Med. Grad. Assoc.’ 1869) found the pulse intermittent in an infant on the day of birth,

and this condition lasted for five years, after which it gradually disappeared; in another case he found it present in a boy five years old, who afterwards became entirely free from it. In adults it is sometimes due to flatulent dyspepsia. Dr Balfour, indeed, is disposed to think that it is rarely dependent upon any form of indigestion except associated with gout, but in this few will agree with him.*

In some persons intermittence of the pulse is produced by drinking tea; and in others tobacco-smoking has a like effect. In many persons intermittence of the heart's action appears to be traceable to the shock of sudden terror or grief. Richardson and Balfour mention cases in which it was set up by a railway accident or by a shipwreck; and the former relates the cases of two patients in whom intermittence of the pulse preceded an attack of mania, in one of them on several different occasions.

Irregularity.—A pulse may be irregular (apart from intermission) in two ways: the force of each beat may be unequal, or the interval between the beats may be unequal. Most often the two conditions are met with in combination. The most common form is a series of frequent, short and feeble beats following an ordinary one. The ventricle makes a series of ineffectual contractions in rapid succession, and the patient describes the result as a fluttering sensation within the chest.

Irregularity of the pulse and heart often accompany tachycardia and, like it, may occur in a perfectly healthy person. A youth who runs or rows in a race without suitable training will experience irregular as well as quickened pulse, and if he persists in the effort this may lead to palpitation, and at last to feebleness and syncope (asphyxia in the proper sense of the word).

The same irregularity may result from mental emotion, or from what appears to the examiner the trifling excitement of a *vivâ voce* examination for a diploma or for life insurance. In such cases one may find the pulse very irregular in rhythm and unequal in force, with perhaps a loud systolic murmur also; and yet a few minutes' repose will show that the whole disturbance is only temporary.

Even constant irregularity of the heart's action is sometimes less serious than might be expected, especially in persons no longer young, who are able to lead quiet lives, undisturbed by active exertion or strong emotion. Such persons may live for years, and pass their days happily and usefully. On the other hand, irregularity of the pulse may be a symptom of dangerous organic disease; for when no murmur can be detected, there is always the possibility of degenerative changes in the walls of the left ventricle.

As a rule, irregularity of the pulse should be regarded as of more importance than mere intermittence. A temporary acceleration of the pulse, for perhaps ten or twenty beats at a time, with subsequent slackening, while one has one's fingers on the wrist, is of no consequence. Irregularity which passes off when the patient sits down and no longer thinks about his heart, is of no grave moment, even though it returns on every fresh occa-

* A circumstance observed with regard to this kind of pulse seems to be very suggestive as to its mode of production. As is well known, intermittence of pulse, when the result of dyspepsia, is very apt to come on after the patient lies down in bed. Now, I have noticed in my own person—and patients have assured me that they have noticed the same thing—that when the pulse is intermittent overnight, it often is so on the following morning also, although it becomes regular after one has risen from the recumbent posture. The probable explanation seems to be that what causes the heart's action to intermit is the presence in the stomach of solid pieces of food imperfectly masticated.—C. H. F.

sion of excitement; but it becomes serious when the intervals grow shorter and the excitement necessary to affect the heart gradually less.

Irregularity with an infrequent pulse is much more dangerous than when it goes with tachycardia.

Irregularity of the pulse, as a constant symptom, is associated with mitral incompetence, and the later stages of all forms of cardiac disease. It is also an important sign of failure of the ventricle in cases of enterica and other fevers, in pneumonia, and in peritonitis. It is met with in some cases of flatulent dyspepsia, particularly when due to tea-drinking, and is a well-known result of the excessive use of tobacco. Lastly, it may be produced by an overdose of digitalis.

A curious irregularity, named *pulsus paradoxus* by Kussmaul, is that the radial pulse ceases to be felt during inspiration. It is an exaggeration of the physiological fact that in some grown persons and in many children the arterial blood-pressure rises in expiration and falls during inspiration, the rise producing a fuller and slower pulse, the fall a weaker and more frequent one.

Allorhythmia.—In some cases disturbance of the heart's rhythm shows itself, not in irregular irregularity—if the term is admissible—nor in regular intermission, but in the constant succession of beats alternately strong and weak, or in the constant coupling together of beats in pairs. The former condition was named by Traube the *Pulsus alternans*, the latter the *Pulsus bigeminus*. By Sommerbrodt ('Deutsches Arch.,' xix) they were included together under the common name of "allorhythmia." In English we might use the terms "doubled" or "twin" pulse, and "grouped" pulse to denote a succession of two or more similar beats, and "alternate" for the more or less constant succession of a strong and a weak beat. *P. trigeminus*, with the beats in groups of three, might be called "trebled, or triplet pulse."

A good example of the *pulsus alternans* was recorded by Dr Fagge in the 'Guy's Hospital Reports' for 1871, vol. xvi, p. 330.

The patient was a woman of thirty, who came into the hospital with a well-marked presystolic murmur. The usual rate of her heart's action was about 70. But sometimes it would rise to 92, and then only every other beat produced a pulse at the wrist, which accordingly was counted as 46; there was, however, reason to believe that the beats which failed to reach the radial arteries were attended with reflux into the systemic veins, inasmuch as a pulse could be felt at the root of the neck, apparently in the jugulars. It is worthy of notice that 70 is almost exactly the arithmetical mean between 46 and 92. At one time the allorhythmic state of the pulse could be stopped at will, by making her walk so as to quicken the heart's action; the rhythm was then normal, but afterwards, when the heart began to slow again, it fell into the peculiar alternate rhythm. Traube supposed the *pulsus bigeminus* to be a sign of the near approach of death, but this patient lived ten or eleven years after the publication of her case.

Allorhythmia, like arrhythmia and intermission, is not always a sign of cardiac disease; it has been noticed in cases of cerebral hæmorrhage or softening, and also during convalescence from pneumonia, peritonitis, or fever. It has also been frequently observed as a sequel of epileptic attacks, and Tripier, of Lyons, has collected a large number of these cases.* When the pulse is at times allorhythmic, it is apt at other times to be arrhythmic, and both may be felt in the same patient within an hour.

* Sommerbrodt compared allorhythmia with the modification of breathing associated with the names of Cheyne and Stokes; and the two conditions sometimes occur together.

Palpitation of the heart.—Under ordinary conditions a healthy person is unconscious of the heart's action—if one becomes aware of its pulsations, palpitation is said to be present. This does not usually denote permanent disease; it may occur to any person after great exertion, or under strong emotion. But, apart from such causes, it may be due to various kinds of disturbance of the heart; and it sometimes appears to be the chief symptom a patient complains of. In some cases in which the cardiac pulsations seem to the patient to be extremely violent, the physician may find, on placing his hand over the left side of the chest, that they are really quiet and natural; but more often they are really increased in force, so as to shake the chest, or the entire body, or even the bed the patient lies on. The attempt to lie on the left side often aggravates the palpitation, and causes throbbing of the carotids, a sensation as though “the heart were jumping into the throat,” or “the eyes bursting from the sockets,” flashes of light, dizziness, faintness, or an indescribable sense of discomfort in the region of the heart. In some severe cases there is extreme distress, with fear of impending death.

On examination, the apex-beat is seen to be in its natural position, but it occupies too extensive an area. Walshe described the impulse as feeling like a *blow* if the heart is well nourished, like a *slap* if it is feeble. The area of cardiac dulness is usually unchanged, but in prolonged paroxysms it may be increased to the right of the sternum. The sounds are loud and clear, with a metallic ringing character; and the first sound can occasionally be heard at a distance of some inches from the chest. A basic systolic murmur is often heard when there is no marked anæmia, and it has the characters of a pulmonary (dextro-sigmoid) bruit. Still more frequently transitory mitral regurgitation is present, causing a systolic murmur at the apex; but this is audible in many cases of tachycardia, with irregular pulse under excitement. In fact, although we are obliged for clearness to deal with each condition separately, palpitation with a functional apical bruit is most often met with under temporary excitement, combined with a frequent and irregular pulse.

Palpitation of the heart is present in various forms of organic disease, and may also accompany functional or neurotic disturbance of the organ. In the former case, and also in Exophthalmic Goitre, it is more or less persistent; while in the latter it is usually paroxysmal. An attack may last from a few moments to several hours, and may end with an abundant flow of watery urine.

Palpitation about puberty is often due to the heart not developing in proportion to the bodily growth.* During early adult life palpitation is frequently met with. Hysterical women are very liable to it; but perhaps the most severe cases are seen in young men. It may result from want of sleep or food, from sexual excesses or masturbation, from the abuse of alcohol, of tobacco, or of strong tea. When none of these causes can be made out, it may be due to flatulence after filling the stomach with imperfectly masticated food, especially before bedtime. It is when palpitation recurs night after night, while the patient is in bed, that this cause should be suspected. In other cases it comes on during exertion; but as Dr Balfour remarks, one characteristic of the nervous or functional nature of the palpitation is that it disappears if the patient quickens his pace. In such cases

* See Bowditch on “The Growth of Children,” and Beneké's observations on the volume of the heart at different ages, quoted by Dr Pitt (‘Brit. Med. Journ.’ Nov. 27th, 1886).

palpitation will be present when he is walking or dining alone, and absent when he is in a friend's company.

Cardialgia.—Palpitation is often accompanied by severe distress referred to the chest, but acute neuralgic pain is a somewhat rare symptom of organic disease of the heart. It most frequently accompanies lesions of the sigmoid valves or of the ascending aorta. It is a common symptom of aneurysm, and we shall see that in its most severe form it is part of the condition known as angina pectoris.

Treatment of palpitation, tachycardia, and irregular pulse.—Functional disorder of the heart is often due to overloading of the stomach, and regulation of the diet is essential. Moderation in the use of stimulants should also be insisted on, or entire abstinence; and the same applies to tobacco, tea, and all forms of excitement, including sexual indulgence.

When there is anæmia, iron or arsenic may be prescribed with advantage; but of drugs intended to affect the heart directly, digitalis is by far the most useful. This was Da Costa's experience, and it is in entire accordance with that of most English physicians. Occasionally, however, like every other drug, it fails either in diminishing the frequency of the pulse, or in correcting its irregularity. Other medicines which may then be used are strophanthus, bromide of potassium, hyoscyamus, cannabis indica, and ether. For the palpitation of hysterical women, assafoetida, Sp. Armoraciæ co., and valerian are indicated.

For palpitation accompanied by irregularity and intermittence of the pulse, there is no doubt that half an ounce of brandy is an efficient remedy; but the risk of dram-drinking becoming a habit makes it too dangerous. The writer would advise in preference the use of aromatic spirits of ammonia, Sp. Ætheris Co., Sp. Chlorof. and Tr. Card. Co. Nux vomica is often of great use in steadying the heart's action; and belladonna is particularly valuable when the pulse intermits.

Aconite seems to be of no service in cases of mere functional palpitation, but Da Costa obtained good results with it when the heart was beginning to undergo hypertrophy. It often exerted a marked influence upon the force of the cardiac beats without diminishing their frequency, whereas the opposite effect was produced by digitalis. Consequently, in suitable cases the two drugs may be given together with advantage. Veratrum viride seems to be intermediate in its action between them.

Tension of the pulse.—The methods of estimating resistance of the pulse to compression have been already considered. The older physicians used the names *pulsus durus* and *pulsus mollis* to denote the degrees of tension, and called a small incompressible pulse "wiry," a small and compressible one "thready."

The tension felt by the educated finger placed on the radial artery depends first on the force of the left ventricle behind, secondly on the resistance in the capillaries and contracted arterioles in front; and this feeling of pressure or tension will be modified by the softness and elasticity, or the hardness, rigidity, and thickness of the walls of the radial artery.

The highest tension is commonly found in the most chronic form of Bright's disease, when there is increased resistance in front and a hyper-

trophied ventricle behind. But high tension is also found in many cases of early and acute nephritis after scarlatina, and in a less degree in other acute inflammations, especially of the serous membranes; likewise in cases of gout and plumbism, and of epilepsy. It is also met with during pregnancy, in some cases of chlorosis, and in full-blooded, free-living patients, who find relief in blue pill, colchicum, and a purge. The hard pulse sometimes found in cases of apoplectic coma is in most cases due to concomitant Bright's disease.

A low tension with a relaxed artery is characteristic of pyrexia generally, particularly of the specific fevers, and of acute rheumatism.

In cases of pneumonia and meningitis and in some cases of acute Bright's disease, the pulse is large or "full," with high tension. This is the "bounding" pulse of the older physicians of which the mention always precedes the record of venesection. Another and a very characteristic condition is the strong, short, full pulse with low tension, which, when fully developed, is the collapsing or "water-hammer" pulse of Corrigan.

A small pulse with high tension, shown by the length of the systolic expansion and the slight degree of the diastolic fall, is the opposite of the last variety, and constitutes the "persistent" and hard pulse of chronic renal disease.

A small, weak, and compressible pulse, which is also irregular, is one of the characteristic symptoms of mitral regurgitation.

The same hardness which is felt by the finger in the contracted and thickened artery of Bright's disease may be produced by calcareous degeneration of the middle coat of the radial artery, or by the more common atheroma of larger vessels. The visible pulsation and emptiness during diastole, which in young subjects with elastic arteries is indicative of imperfection of the sigmoid valves, denotes in older persons a want of elasticity of the aorta and other arteries, due to degenerative changes.

A "fluttering" pulse is one very irregular, and perhaps intermittent; and a "running" pulse is one so weak and so frequent that it can no longer be counted.

The pulse of the radial artery may be always weak, and its lumen easily compressible, without there being any lesion of the heart or any deviation from health. Hence "a poor pulse" is often a deceptive guide in a patient seen for the first time, or in a candidate for life assurance. A short, "slapping" pulse—*pulsus celer*—associated with low tension, is often felt after depletion by hæmorrhage or purgation.

The pulse of high tension may often with advantage be treated by purging and moderate blood-letting; the soft pulse often calls for stimulants and steel. But as in other cases, such indications are only to be followed when the primary disease has been ascertained.

Absence of the pulse (etymologically denoted by the word asphyxia) is not only observed in syncope and collapse, when the heart's beats are too feeble to be felt at the wrist, but may be the result of peripheral causes affecting one or both radial arteries.

Weil has described the pulse disappearing in inspiration at one wrist only, and has noticed the same, less frequently, coincident with deep expiration. He thinks that this may depend upon adhesions of the subclavian artery with the pleura and the dome-shaped process of cervical fascia at the root of the neck.

Hamburger remarked a very singular fact with respect to the pulse. If a healthy person throws the shoulders well backward, and the arms backwards and downwards so as to cross the hands over the sacrum, on deep inspiration both radial pulses disappear for the time. Hyrtl explained this as due to compression of the subclavian artery by the first rib, and Hamburger believed that when this is not the case, it is due to perichondritis fixing the first cartilage, a frequent concomitant of phthisis with pleurisy of the apex. This experiment has been frequently repeated by the writer, who found that the pulse first becomes irregular, weak, and slow, and then disappears, to return at once on taking a fresh breath or moving the arms.

If the two radial pulses differ markedly in size or in force of beats, there is either some abnormal distribution of the arteries, or obstruction from a tumour pressing from without, or atheroma or some other local lesion. In many cases the tumour or internal lesion is an aneurysm, and this may either act by pressing on the artery or may diminish the flow of blood, and thus the size of the artery, by diverting it from its normal channel.

*Syncope.**—Fainting or sudden failure of the heart varies in degree. In the most severe cases the patient experiences distressing sensations of giddiness, nausea, and sinking at the epigastrium. He turns cold and pale, and breaks out in a clammy sweat; his sight becomes dim, his head swims, everything appears black, he hears rushing noises in the ears, and becomes insensible. His pulse is frequent and very weak, and it soon ceases to be felt at the wrist, though it may still be counted in the carotids. The heart's impulse grows more and more feeble, until it is no longer perceptible. Although one may still hear its sounds with the stethoscope, they are faint, and the second one is audible only at the base. The respiration, meantime, becomes infrequent, irregular, and shallow, and the pupils are dilated.

In slighter cases, after having suffered for some minutes from giddiness and nausea, the patient loses himself just for an instant, and then gradually recovers. It is not uncommon for recurrent "fainting fits," with an imperceptible pulse and very feeble cardiac action, to last for an hour or longer, and yet to end favourably. The subsidence of an attack is marked by gasping, or sighing respiration at intervals, and by gradual return of pulse, consciousness, and colour. These symptoms are often painful and distressing, more so than the passage to unconsciousness.

A case of supposed syncope in which the stoppage of the pulse and the interruption of the mental faculties are absolutely sudden, and in which the resumption of the heart's action and the recovery of consciousness are no less instantaneous, is probably not true syncope, but the minor form of epilepsy. A person feeling faint can usually sit or recline, she droops rather than drops; but an epileptic attack is more sudden, and hence the face and head are more often injured by a fall.

The heated air of a crowded room, the sight of blood (even from a cut finger), the strong odour of flowers, the introduction of a catheter, may

* *Synonyms.*—A fainting fit—A swoon—Lipothymia—Deliquium animi, Συγκοπή is the regular term for a swoon in Galen, Aretæus, and Plutarch; λιποθυμία is used in the same sense by Hippocrates, but has been appropriated by some later writers to collapse as distinct from fainting.

each cause fainting in certain persons. It is most apt to occur in young adults, and in women rather than in men; but it frequently befalls healthy and vigorous youths.

Standing up suddenly or for a long time, particularly when the hands are unsupported, as with soldiers on parade, or schoolboys in class, or women in a crowd, is apt to cause syncope. Even getting quickly out of bed gives a momentary sensation of faintness in many otherwise healthy persons, as they gain their feet. Walking or standing or attending a church or school before breakfast, and without taking a biscuit and a glass of milk as a precaution, are well-known causes of more or less developed syncope. The difficulty is no doubt the purely hydraulic one of adequate supply of blood to the brain. Again, certain kinds of pain, particularly crushing of a finger or a limb, and most of all compression of a testis, will produce faintness or deep and fatal syncope. And the mere apprehension of pain has sometimes the same effect upon one who will immediately afterwards bear its infliction with fortitude.

It is important not to allow those who are convalescent from fever, particularly influenza and enteric fever, to sit up in bed suddenly; and the same caution applies to those suffering from severe diarrhoea. Among the causes of the graver forms of syncope are hæmorrhage, organic disease of the heart and aorta, pulmonary embolism, and occasionally the too rapid withdrawal of ascitic or pleuritic fluid by tapping.

In syncope due to hæmorrhage there is a waxy pallor of the face and lips, of the hands and finger-nails, and of the whole surface. Consciousness is gradually lost, and occasionally epileptiform convulsions occur. Sometimes the patient lapses from time to time into a state of insensibility, regaining consciousness in the intervals of these "fainting fits." They recur if the patient attempts to sit up, and at the same time the pulse at the wrist becomes feebler. After death the heart is found contracted and empty. This form of death follows ruptured aneurysm, traumatic hæmorrhage, uterine flooding, and purpura, but seldom hæmoptysis, hæmatemesis, or intestinal hæmorrhage.

Syncope must be clinically distinguished from the condition known as *collapse*, although both are accompanied by failure of the heart, and their pathological difference is not clear. In collapse the pulse at the wrist becomes imperceptible, and the cardiac sounds are scarcely audible, as in syncope; but, in addition, the surface of the body, and especially of the hands and feet, is cold; the features are sunken, the eyes retracted in their sockets, and the orbits surrounded by dark rings. Yet the patient is often free from giddiness, nausea, or faintness, and may still be able to get out of bed. Collapse may result from various abdominal lesions, as peritonitis, strangulation of the bowel, and perforation of a hollow viscus, and is then probably due to reflex inhibition from the solar plexus.

The "cardiac" variety of *sunstroke* must also be mentioned. In this the sufferer gives no sign of illness until he falls, gasps, and perhaps at once expires before anything can be done to help him. Dr Maclean says that this is the form most often seen in soldiers exerting themselves in the heat of the sun when fully dressed and accoutred (cf. vol. i, p. 939).

Death by syncope from organic disease of the heart may be absolutely sudden; the patient falls to the ground or sinks back in his chair unconscious, and is dead before help can be given. The respiration may cease at the same instant as the beats of the heart, or a few deep gasps occur;

then a kind of shiver passes through the frame, pallor spreads over the surface, and all is over.*

The causes of sudden stoppage of the heart are various. In animals powerful stimulation of one of the *nervi vagi* arrests the cardiac beats; a similar effect follows irritation of the cardio-inhibitory centre in the bulb; frequently it is the result of direct or reflex inhibition through the vagi; and, lastly, it can be produced as a reflex effect by a violent impression upon peripheral nerves, as by suddenly crushing the foot or striking the exposed intestine with the handle of a scalpel in the frog (the *Klopfversuch*), or even by gentler stimulation when peritonitis has first been set up.

In human pathology all these varieties of inhibition seem to occur more or less frequently.* That of which in medical practice we know least is perhaps direct inhibition by stimulation of the vagus. Czermak, however, was able at will to stop the beating of his own heart by pressing the trunk of the pneumogastric nerve against a small osseous tumour in the neck. In cases of aneurysm of the aorta, it is not uncommon for sudden death to occur without the autopsy revealing a rupture of the sac or any other definite change; in such cases the fibres of the left vagus nerve are often spread over and inseparably blended with the walls of the aneurysm; and this may perhaps cause the arrest of the cardiac pulsations. Inhibition of the heart by stimulation of the centre in the bulb probably occurs when fainting is caused by emotion.

Death by reflex inhibition appears to be of frequent occurrence. There can be little doubt that the "fatal shock" which accompanies severe injuries of the limbs or of any part of the body is of this nature. But what is most striking is that abdominal diseases are specially apt to be attended with sudden death. In the collapse of acute peritonitis death is often sudden. Cases of sudden and fatal syncope from slight blows on the epigastrium, like the *Klopfversuch*, are not uncommon. The classical instance is that recorded by Sir Astley Cooper of a sailor, who, while "skylarking" with a shipmate, and lifted on his shoulders, received a tap on the pit of the stomach which caused immediate death.

Apart from these causes of sudden and fatal syncope which are nervous and inhibitory in origin, and from those which depend on hæmorrhage, are the still more numerous cases of syncope from mechanical arrest of the flow of blood from the heart to the brain. They occur most frequently as the result of aortic regurgitation or obstruction, next of mitral stenosis, and less commonly from pericarditis, rupture, and fatty or fibrous degeneration of the left ventricle.

In the *treatment* of a faint, the first thing is to give the patient air, and to lay him down with the head as low as possible; the clothes must

* It is often impossible, in such cases, to determine whether paralysis or spasm of the heart is the cause of death. If an autopsy is made, its chambers may be found either relaxed or contracted, either empty or more or less full of blood. But it is difficult to say how far its state may be modified by contraction of the ventricular walls after death. If they are flabby, while the muscles generally are in a state of rigor mortis, it seems fair to conclude at the time when life became extinct the heart stopped in a condition of diastole. On the other hand, stoppage in systole is not always due to spasm; it may be the result of rigor mortis.—C. H. F.

* If the analogy with the inhibitory phenomena observed in the physiological laboratory can be looked upon as tolerably complete, it is an interesting question whether in any circumstances the supervention of collapse or of fainting can be prevented by the injection of atropine, the effect of which in animals is to completely annul the normal inhibitory action of the vagus.—C. H. F.

be loosened about the throat and chest, and sympathising friends must be sent away. A bottle of ammonia may be held to the nostrils; or, if this be not at hand, a bunch of feathers may be burnt and the fumes inhaled. Cold water may be sprinkled on the face, and if the stomach be overloaded, an emetic of mustard should be administered; for this, as Anstie says, has a powerfully rousing influence upon the heart. Sal volatile, brandy, ether, or a tumbler of cold water may be given by the mouth; or, in severe cases when the patient continues unconscious, five drops of strychnia, or fifteen of either ether or brandy may be injected under the skin. Our house physicians frequently adopt this practice, and in many cases with marked success in stimulating the action of the heart, although the result is too often temporary.*

It is not yet quite clear whether galvanism is useful in cases of severe syncope. Ziemssen found ('Deutsch. Arch.,' xxx, 1881) in a patient whose chest wall was deficient so that the heart was covered only by the skin, that the heart's beats could be accelerated by powerful currents. Erb recommends that in galvanising the heart large electrodes should be used, one being applied over the surface of the organ, the other over the dorsal vertebræ; a current of high intensity should then be passed, its direction being reversed seventy or eighty times in the minute. Previous observers, as cited by Walshe, had found that with strong currents there was a risk of inhibiting the cardiac contractions instead of stimulating them. The battery is often applied when the heart ceases beating under chloroform, but it is doubtful whether it does good.

* Another method, advanced by Dr J. C. Reid ('Brit. Med. Journ.,' 1880, vol. ii, p. 1014), is that of pouring hot water over the præcordial region. He cites a case in an old man of seventy, who was thus restored from apparent death, and lived long afterwards.—C. H. F.

ANGINA PECTORIS

Ac velut in somnis oculos ubi languida pressit
Nocte quies, nequicquam avidos extendere cursus
Velle videmur, et in mediis conatibus ægri
Succidimus; non lingua valet, non corpora notæ
Sufficiunt vires, nec vox nec verba sequuntur.

VERGIL, *Æneid*, xii, 908—12.

Heberden's original account of the disorder—Onset and symptoms—Recurrence and event—Anatomy—Pathology—Angina vasomotoria—Pseudo-angina—Secondary angina—Cardialgia—Ætiology—Age and sex—Cases—Prognosis—Treatment.

THIS term, Angina pectoris,* may be, as we shall see, restricted to a very definite and striking set of symptoms associated with a definite anatomical lesion; or it may be applied to certain symptoms, however caused; or it may include almost any functional disorder attended with pain and referred to the chest. In its most marked and serious form, it may be defined as the most severe form of cardiac pain, occurring in sudden paroxysms, and accompanied by a sense of impending death; it returns again and again, and sooner or later is fatal.

It was first described by Heberden† as follows:—"There is a disorder of the breast, marked with strange and peculiar symptoms, considerable for the kind of danger belonging to it and not extremely rare, which deserves to be mentioned more at length. The seat of it and sense of strangling and anxiety with which it is attended may make it not improperly be called Angina Pectoris. They who are afflicted with it are seized while they are walking (more especially if it be uphill and soon after eating) with a painful and most disagreeable sensation in the breast, which seems as if it would extinguish life if it were to increase or to continue; but the moment they stand still all this uneasiness vanishes. In all other respects the patients are at the beginning of the disorder perfectly well, and in parti-

* *Synonyms.* — Pectoris dolor — Breast-pang — Syncope anginosa (Parry) — Asthma dolorificum (Erasmus Darwin) — Cardiodynia — Neuralgia cordis (Laennec) — Hyperæsthesia plexus cardiaci (Romberg) — Stenocardia (Eichhorst). — *Fr.* Angine de poitrine. — *Germ.* Brustbräune — Brustklemme.

† In the 'Medical Transactions' of the College of Physicians for 1768. Some French writers have set up a claim of priority for their countryman, Roushon, on account of a letter written by him to Lorry a few months earlier, in which is related the death of a cavalry officer, M. Charles, by what was probably the same disease. But so far as the account of a single case anticipated Heberden's observations, the priority belongs to Morgagni, who recorded one in a Venetian woman early in the eighteenth century (quoted by Gairdner 'Reynolds' System,' vol. iv, p. 537).

cular have no shortness of breath, from which it is totally different" ('Commentaries on the History and Cure of Diseases,' 1782, chap. lxx.

In its well-marked and primary form, angina pectoris is a rare malady. Cases are not infrequent which more or less resemble it in the character of the pain, but without the same danger to the patient's life; and these are called pseudo-angina. Again, anginal attacks are not infrequent in the course of organic disease of the heart and aorta, but these, although ending fatally, are not idiopathic. Whether or not they should be classed under the same heading depends on our definition of the term. The following description refers to the classical form of the disease, idiopathic and fatal.

Onset and exciting causes.—The first attack of Angina pectoris is almost always absolutely sudden. It usually occurs while the patient is walking, especially on rising ground, or with a strong wind against him, or shortly after a meal. But sometimes (as in the case of Arnold, of Rugby, recorded by the late Dr Peter Latham) one who has never suffered from angina before is awakened from his sleep by the first attack. Subsequent seizures are apt to be brought on by comparatively slight causes, until the patient gradually finds that one form of exertion after another is unsafe for him. Emotional excitement is a powerful exciting cause, as in the well-known case of John Hunter, who died within the walls of St George's Hospital in the midst of a dispute, of which he had foreboded the fatal result. In many patients the seizures, which at first took place only during the daytime, afterwards begin to recur in the night also. The act of stooping to pull on or lace the boots is a frequent cause of the attacks. In advanced cases they are brought on by such slight efforts as coughing, defæcation, or swallowing cold water.

Symptoms.—The pain in angina pectoris is usually referred to the lower part of the sternum, rather to the left than the right side; sometimes behind the middle or the upper part. Patients attempt to describe it, as tearing, stabbing, or lancinating in character. But along with this they feel something worse than pain,—the distress of suffocation and of impotence to move or to relieve the oppression. The agony is indescribable—so intense as to make the sufferer feel that unless it abates it must kill him. The pain often spreads round, generally through the left side of the chest, to the spine. It is sometimes accompanied by a sense of constriction, as though the sternum were forcibly drawn backwards: Gairdner cites the case of a medical man in whom there was a subjective sensation as though the front of the chest were "bulged out in a convex prominence, terminating suddenly at the lower end of the sternum in a sharp and deep depression." The pain radiates sometimes upwards into the neck or towards the occiput, more often, as Heberden said, down the left arm to the elbow or the fingers, occasionally down the right arm, and it is said in rare cases to shoot into the lower limbs or into the testes. In a case observed by Walshe, and another by Broadbent, it took a course the reverse of what is usual, beginning at the left wrist and extending upwards to the heart.

Attempts to define and classify pain must always be imperfect, since we have neither a gauge of its degree nor a criterion of its quality. But we may distinguish by comparison between the superficial tenderness of the skin or the peritoneum, the acute "thin" pain of an exposed dental pulp (neuralgia), the grinding pain of toothache (inflammation), the throbbing pain of a whitlow or gumboil (suppuration), the sickening and disabling pain of a bruised

testicle, the sharp stabbing pain of pleurisy, the dragging but acute pain of cramp and lumbago, and the discomfort, often exceeding pain in its distress, of hunger, thirst, and want of breath. To these we may add the sense of impotence felt in a nightmare, when the utmost effort of the will is powerless to move the muscles.

Judging from the description of patients, an attack of angina is marked by acute darting pain, like that of *tic douloureux*, behind the sternum and down one arm; by constriction and weight on the chest, which is not felt as want of breath; by a sense of impending death; and, lastly, by a depressing feeling of futile effort to overcome an obstacle like that of colic, of urethral stricture, or of fruitless labour pains.

A patient attacked by a fit of angina is instantly arrested in whatever he is doing; if walking, he stops motionless; if standing, he dare not sit down. It is, however, a curious fact that some persons, after having been pulled up by the pain three or four times at the beginning of a walk, will afterwards go on with ease. The feeling of constriction in the chest may cause the patient to speak of experiencing a "want of breath" or a "sense of suffocation;" but there is no dyspnoea in the proper sense of the term, and no lividity of the face. The breathing may be somewhat frequent, but this is because the patient instinctively keeps the thoracic movements as shallow as possible, for fear of increasing the pain. By an effort of the will he can, if he chooses, freely expand the chest, and there are cases in which a resolutely drawn deep breath gives momentary relief.

With regard to the state of the heart and pulse during a paroxysm of angina pectoris, accounts have differed; and probably all cases are not alike in this respect. It is said that the impulse and the sounds of the heart are unaltered throughout the seizure, that the pulse remains regular and neither frequent nor weak. Walshe observed that at least towards the close of a paroxysm, the pulse may be neither accelerated nor irregular. On the other hand, Parry long ago described the pulse as being more or less feeble, according to the violence of the attack; and Gairdner expresses the same opinion. In many cases it is expressly noted that the pulse has been small and irregular in rhythm, not increased in frequency, and sometimes very slow; but Heberden's remark remains true, that in most cases the pulse is not disturbed by the pain, although the face is deadly pale, and the skin covered with a cold sweat. In cases about to prove fatal, the pulse becomes imperceptible only just before death.

The mental faculties remain unimpaired throughout the seizure. The condition is not that of syncope. Slight convulsions or more severe tonic spasms have been described; and also flatulence with eructation or vomiting, followed by a copious flow of watery urine; the bladder may be evacuated during the attack, and a fit of angina never lasts more than a very few minutes, sometimes less than half a minute. But attacks may recur again and again for an hour. A patient of Dr Fagge's remained for several hours stooping over the end of a couch and refusing to move for fear of the pain returning; and when a seizure occurs during walking, it usually ceases as soon as the patient stands still. Trousseau tells how one patient lay motionless on his back, another backwards on his chair, a third leant forwards as far as possible, and a fourth may place himself on his knees and elbows.

Event.—Sometimes death appears to be absolutely instantaneous; Walshe relates an instance in which the patient had been reading quietly

in bed, and in which the thumb and the forefinger were found in the pamphlet on which he had been engaged, the bedclothes being also quite undisturbed. The same tranquility was observed in the case of Dr Chalmers, which Gairdner describes as an undoubted case of angina. In some of these cases, when former attacks were attended with severe pain, it seems probable that the fatal seizure was so brief as to be painless; while in persons who have never been known to suffer from angina, sudden death may be due to a first attack.

In a few cases death is more gradual, and preceded by increasing failure of the pulse, laboured breathing, and unconsciousness.

The paroxysms of angina pectoris return again and again, and there is generally an interval of some years between the first attack of the disease and its fatal termination. Whether a single paroxysm may be recovered from and never followed by others, seems to be doubtful.

Walshe once saw a patient who had been subject to angina for twenty-four years; and there is reason to believe that John Hunter had begun to have seizures twenty years before the fatal one. A still longer duration recorded by Dr Murrell, was that of a patient who had suffered for thirty years, the diagnosis of angina pectoris having been given by Sir Risdon Bennett twenty-six years before.

Too often the paroxysms return frequently. In a case recorded by Walshe, there were only three, one being a year, and the other half an hour, before the third and fatal one. Latham met with two cases, in one of which death occurred fourteen days, in the other ten days, after the first attack. The most rapid case on record is probably that of Dr Arnold, who, having never suffered from angina before, went to bed on the 11th of June, 1842, in apparent health, awoke in a paroxysm of pain at five in the morning, and died about a quarter past seven in a second attack.

Anatomy.—After death from angina, the heart is found to be relaxed and flabby, even though there is marked cadaveric rigidity of the muscles generally; but Walshe says that there is scarcely any blood in the cardiac cavities, which looks as though a ventricular systole had been the last act of life. In the severe and fatal form of angina, which includes the most striking and uniform cases, organic changes in the heart or large vessels are almost always present. The most conspicuous lesion is arteritis of the aorta at or near its origin, leading to atheroma, to calcification, and perhaps to aneurysm; but the most constant lesion is obstruction of the coronary arteries, from atheroma or an earlier stage of arteritis deformans. This condition was first suggested by Jenner, in a letter to Parry, and afterwards in one which he addressed to Heberden in 1778, but which he did not send, lest it should be seen by his friend Hunter, whom he rightly believed to be at that time a sufferer from angina. Sometimes the orifices of the coronary arteries are more or less completely obstructed by disease of the aorta, their coats in the rest of their course being healthy; but sometimes they are converted into thick calcified tubes in nearly their whole length.

In Hunter's case (1793) "the coronary arteries had their branches which ramify through the substance of the heart in the state of bony tubes, which were with difficulty divided by the knife, and their transverse sections did not collapse." There was atheroma and dilatation of the ascending aorta, and calcification of both aortic and mitral rings, but the valves were probably competent, and the cavities were not dilated. Sir

Everard Home notes with much emphasis two "milk-spots" on the surface of the heart, but he omitted to weigh it ('Life of Hunter,' by Adams, p. 203).

In Robert Hall's case (1831) Dr Pritchard reported "the muscular structure (of the heart) to be soft, like macerated cellular membrane; the left ventricle was judged to be one third larger than usual. The aorta contained in several places patches of bony matter." The valves are not mentioned, nor the coronary arteries (Memoir by Gregory, note D).

In Arnold's case (1842) Dr Bucknill and Mr Hodgson found the aorta and all the cardiac orifices healthy. The muscular structure of the heart in every part was remarkably thin, soft, and loose in its texture. The walls of the right ventricle were in some parts not much thicker than the aorta. There was but one coronary artery (?) which with some difficulty admitted a small director ('Latham's Lectures,' vol. ii, p. 377).

In the case of a young man, recorded by Dr Balfour (1877), the heart weighed thirteen ounces, the cavities were slightly dilated and hypertrophied, the mitral and tricuspid orifices enlarged, the aorta atheromatous, and the coronary arteries "both so extremely contracted as barely to admit the point of an ordinary surgical probe." The muscular structure was healthy to the naked eye, and under the microscope "presented no abnormality, except the presence of a considerable number of reddish-brown pigment granules in some of the fibres" ('Clinical Lectures,' p. 301). In a second case of angina, in a woman of eighty, Dr Balfour found disease of one coronary artery, with dilated ventricle and thin, pale, fatty muscle.

Apart from occlusion of the coronary arteries, angina (or symptoms clinically resembling it) most often appears in cases of atheroma of the aorta—whether leading to aneurysm or to valvular lesions with dilatation of the heart—and very rarely in cases of mitral disease.

Pathology.—The physiology of angina pectoris is still obscure. Following Laennec (who called it *névralgie du cœur*), Romberg, Friedreich, and Trousseau maintained that it should be regarded as a neurosis, or a "visceral neuralgia."* Anstie declares that those who suffer from the disease are always of nervous temperament, and that other neuroses are frequent in their families; and, like Trousseau, finds a relation of angina pectoris to asthma, while others see a resemblance to epilepsy and other paroxysmal neuroses.

Doubtless angina may from one point of view be regarded as neuralgia or cardialgia; but that it is more than this is clear, for neuralgia is never fatal; nor does neuralgia attack ten men to one woman.

Fifty years ago, Heine ('Müller's Archiv,' 1841) published a case, treated by Skoda, in which Rokitansky made the autopsy, and found the right phrenic nerve, the *N. cardiacus magnus*, and the descending branches of the left vagus, each involved in pigmented nodules, doubtless altered lymph-glands. In this case, however, the symptoms during life were not paroxysms of angina, but intermittency of the heart's action, continued during a period of from four to six beats, and attended with a feeling of inexpressible anxiety. In 1864 Lancereaux ('Gaz. Méd.,' 1864) had an opportunity of

* The same opinion is formally upheld by Eulenburg in 'Ziemssen's Handbuch,' but perhaps a more correct statement would be that he rejects it, since he would exclude from the disease, in its "purely nervous" form, the very cases involving danger to life, on which the foregoing description is based, and which from the days of Heberden to the present time have been regarded as the true and typical examples of it.

examining the body of a man who had long suffered from the classical form of angina pectoris, and at last died suddenly; he found a raised patch in the aorta between the orifices of the coronary arteries, and some fibres of the adjacent cardiac plexus were surrounded by nucleated fibrous tissue. Changes in the nerves (of whatever importance they may be) are recorded and figured by Peter ('*Traité des Maladies du Cœur*,' 1883) in two cases which came under his care, and he cites a fifth instance ('*Bull. de la Soc. Clin.*,' 1878). Such conditions, however, are neither constant nor, perhaps, pathological, and may be dismissed as beside the mark.

Angina vaso-motoria.—Clinical experience has taught that the immediate cause of the paroxysms of angina pectoris is a sudden rise in the tension of the systemic arteries. This opinion had been held by Traube; but what first proved its correctness was a series of observations made by Lauder Brunton on a patient in the Royal Infirmary of Edinburgh. The man was affected with aortic regurgitant disease, and was liable to frequent attacks of angina-like pain. Dr Brunton found that during these attacks the sphygmographic tracing of the pulse became rapidly altered, the wave being broader and lower, the diastolic disappearance, and both the ascent and the descent being more gradual—changes indicative of a marked increase of arterial tension. These observations led him to propose the inhalation of nitrite of amyl as being likely to relieve the pain by relaxing the peripheral arteries, and the experiment was attended with brilliant success.

The pallor and the coldness of the face and limbs that accompany angina do not prove the existence of arterial spasm, for failure of the heart's action might produce the same effect; but Trousseau noticed that the pallor is sometimes followed by a reddish or bluish hue, and the same thing was once observed by Anstie ('*Trans. Clin. Soc.*,' vol. iii), and attributed by him to paralytic dilatation succeeding spasm of the arterioles.

Under the name of "*Angina Pectoris vasomotoria*" Nothnagel recorded ('*Deutsches Archiv*,' iii) a series of cases, in which the earliest and most conspicuous symptoms of the paroxysms were coldness and pallor with numbness and stiffness of the limbs; the feeling of oppression at the chest, the giddiness, and the sense of impending death, were like those of classical angina, and were all apparently due to the efforts which the heart made to overcome the peripheral resistance. But the attacks were traceable to external cold, and were relieved by hot foot-baths and frictions; in fact, the state of the peripheral circulation seems to have been like that in paroxysmal hæmoglobinuria, and the milder forms of Raynaud's disease. Moreover only a dull pain is mentioned, seated chiefly in the cardiac region, but in one case extending over the whole of the left side of the chest, and sometimes down the left arm. This, therefore, though angini-form, is not the intensely painful, recurrent and painful disease described by Heberden.

It is remarkable that increased tension in the peripheral arteries did not cause slowing of the pulse: among Nothnagel's cases there was only one in which a fall from 80 to 64 or 60 beats in the minute was noted.*

We must therefore conclude that the hypothesis "*cardiac neuralgia*" is insufficient—it is, indeed, only a translation of "*breastpang*," and adds nothing to our knowledge of the pathology of angina—and that constrictive

* Landois's physiological explanation of angina, on which Nothnagel's theory is founded, will be found in Eulenburg's article in '*Ziemssen's Handbuch*' (Bd. xii, 2, S. 45—48), and also in Ross's '*Treatise on Nervous Diseases*' (vol. i, p. 731).

tion of the peripheral vessels is probably not constant, and certainly not the efficient cause of fatal paroxysmal angina.

Cardiac spasm.—Heberden rightly referred angina pectoris to the spasmodic, not the inflammatory, class of complaints; and several modern writers regard it as cramp of the left ventricle or of the whole heart—among them Dr Goodhart ('Guy's Hosp. Reports,' vol. xlv, p. 335). Of this, however, there is no evidence, and surely it would cause an immediate and constantly fatal issue of the first attack. Moreover, how could the radial pulse be felt during the paroxysm? Nor is the theory mended by supposing cramp of a portion of the ventricle; for if this occurs in animals, it is a fibrillary contraction which is never recovered from: and why should persistent contraction of a few fibres of the heart produce such severe effects?

An hypothesis advanced by Traube is that the heart, during a paroxysm of angina, is in a state of sudden and extreme *dilatation*; but this is still more devoid of evidence in its favour, and, if possible, would at once cause the arterial pulse to cease and the patient to become unconscious.

Any hypothesis which refers the attack of angina to weakness of the ventricular contractions, as from fatty degeneration or anæmia, is insufficient; because the symptoms of angina are more than those of syncope, and also less, for there is no loss of consciousness.

Pseudo-angina.—Some pathologists regard very mild attacks of more or less distinctive anginal symptoms which have been called "pseudo-angina," as essentially of the same nature as the "true," *i. e.* the severe and fatal disease. Some of the cases described as vaso-motor angina may belong to this group; and those anginal attacks which are complicated with hysteria or flatulent dyspepsia. Within a single year Dr Fagge saw two young clerks in the same London bank, each of whom described attacks that appeared indistinguishable from those of angina pectoris, although their age rendered it very unlikely that the affection would prove dangerous to life. But that the age of the patient does not always form a safe criterion is shown by a case of Dr Balfour's—that of a man, aged only twenty-four, who died after four months' illness with paroxysmal pain in the epigastrium, and in whom (as had been correctly diagnosed during life) the base of the aorta presented a ring of atheromatous thickening, by which the mouths of the two coronary arteries were greatly narrowed. Such a case, however, is altogether exceptional.

In most cases of pseudo-angina which the writer has seen, the paroxysms have lasted much longer than those of "true" angina pectoris, and the pain, though severe, præcordial, and running into the left arm, was not so terrible and paralysing as in the dangerous cases. The vaso-motor symptoms were often present, but the attacks were more frequent, and recurred over a longer duration of time. They are more frequent in women than in men, they are met with at a comparatively early age, and (by definition) they never end fatally.

Local anæmia.—The most certain immediate condition to bring on a paroxysm of angina appears to be very high blood-pressure in the left ventricle; and the most constant anatomical lesion is atheroma of the first part of the aorta and narrowing of the coronary arteries. This may be found after death when there has been no angina during life; but we must suppose that the supply of blood to the myocardium has been just enough. It seems arbitrary to exclude cases of anginal paroxysms occurring in the

course of sigmoid disease or aneurysm, when the coronary arteries are unaffected; and it is very difficult to draw the line between such secondary attacks and the typical "idiopathic" angina. Nor is it less arbitrary to deny the title to many cases, which are called pseudo-angina, only because they are not fatal.

On the whole, it seems at present the safest course to regard angina pectoris as a group of symptoms rather than a disease; symptoms sometimes without any organic lesion, and then free from danger; symptoms sometimes complicating chronic obstruction and incompetence of the sigmoid valves, atheroma of the root of the aorta and of the coronary arteries, and aneurysm of the first part of the aorta. When there are no signs of physical lesions, we have clinically to deal with the symptoms alone, and make of them a disease, as we do of epilepsy or megrim. When, however, they prove fatal, we expect, if there have been no signs of aneurysm or valvular disease, to find atheroma of the sinuses of Valsalva, and of the coronary arteries, and, as a rule, they are present; just as when Jacksonian eclampsia proves fatal we expect to find a tumour or other gross lesion of the brain.

Ætiology.—Concerning the natural history of angina pectoris there is little to add to what has already been mentioned. If it is related to gout, the connection is indirect; arteritis deformans of the base of the aorta and its coronary branches is of frequent occurrence in gouty patients, but also in those who are not gouty.

It is a remarkable circumstance, first noticed by Sir Gilbert Blane, that angina is much more frequent in the upper classes than among the poor. Gairdner questions the facts given, but there is no doubt of the general belief. It must, however, be remembered that while many physicians have large hospital practice and see few rich patients, with others the reverse is the case; that the fatal diseases of public men excite interest and are fully recorded; and that the attacks of angina are so sudden and pass off so quickly that a patient subject to them would scarcely apply for admission to a hospital. Moreover, the first seizure is sometimes fatal. Hence almost the only cases likely to be recorded among the poor would be among out-patients happening to be attacked on their weekly visit, or in-patients admitted to hospital with organic disease of the heart or aorta.

However it may be accounted for, it is probably true that, as Sydenham remarked of gout, and as we found true of megrim, an unusual number of the cases of fatal angina pectoris—always a rare disease—which have been recorded since Heberden wrote, have occurred in men of education and ability. Gairdner believes that the philosopher Seneca was subject to angina, and Sir John Forbes held the same opinion; but the absence of severe pain, and the fact that Seneca died a voluntary and easy death by venæsection at about the age of seventy, surely preclude the inference; rather does the "history of the case" and the patient's own account of his attacks point to chronic bronchitis, ending in emphysema with spasmodic asthma; and this was the judgment of Parry and of Stokes. John Hunter undoubtedly died of angina pectoris, and so did Dr Arnold. Dr Chalmers's death during sleep was unlike ordinary angina; but angina was doubtless the cause of the last illness and death of a still more eloquent preacher, Robert Hall. A patient who described the distress of an attack as "a sense of dissolution, not the fear of it," was "one of the most gifted men" his

physician, the late Sir J. Russell Reynolds, "ever knew, and one most competent to analyse sensations."

Cases.—Excluding, on the one hand, secondary anginal attacks in cases of aneurysm or disease of the aortic valves, and on the other the exaggerated description of neurotic and hysterical patients, angina pectoris is very seldom seen in hospital practice.

A typical case occurred in a patient of the writer, between fifty and sixty, short and stout, a man endowed with great energy and ability. He had no discoverable disease of the heart or aorta, the lungs, or the kidneys; and after being less than a year subject to attacks of increasing severity, all characterised by cardiac pain shooting into the arms, by a sense of imminent death, and by complete immunity in the intervals, he died in one more severe than any that preceded it (1887).

A second case, seen with Mr Edward Acton, in 1890, was in a stout man of seventy-three, with very feeble cardiac action, occasionally irregular or intermittent pulse, and probably extensive atheroma, but with no cardiac murmur.

A third, seen with Dr Few, in 1884, occurred in a healthy farmer between forty-five and fifty, with marked symptoms of angina and no signs of organic disease of the heart or aorta.

In a fourth case, a patient of Dr Hetley, of Norwood, a stout, healthy-looking man, about fifty-five, active, temperate, and intelligent, came complaining of severe breast pain, shooting into the arms, and accompanied by a sense of danger and distress, which compelled him to stop walking and take hold of any support. What showed this to be a case of pseudo-angina was that the attacks were sometimes repeated several times in a day, that they never occurred if he was walking with a friend and engaged in conversation, and that they had lasted some time without involution.

The following are 32 cases of angina of which the writer has notes, excluding those in which anginal symptoms accompanied valvular disease or aneurysm. Twenty-eight of these cases were seen in private, and only four in hospital patients.

There were twenty-eight men and four women. All the women and six of the men suffered from "pseudo-angina," *i. e.* from attacks which, from their duration or other characters, were judged not to be of the same kind as the rest; and all of these were given a good prognosis.

The ages were as follows:—Three patients were under thirty (25, 27, 28); only one of them, a young man of twenty-five, showed the typical disease; 4 between thirty and forty, all of them pseudo-angina; 7 between forty and fifty, 2 pseudo-angina; 10 between fifty and sixty, 2 pseudo-angina; 3 between sixty and seventy, 1 at sixty pseudo-angina; and 5 between seventy-two and seventy-five, all of them typical cases.

"Males are most liable to this disease, especially such as have passed their fiftieth year" (Heberden). Walshe quotes Sir John Forbes's collection of 88 cases: of these 80 occurred in men and only 8 in women, while of 84 patients 72 were more than fifty years old.

In Arnold's case a tendency to angina pectoris (or rather, perhaps, to atheroma) appeared to be inherited. His father died of angina, and his second son of sudden syncope from disease of the aortic valves, preceded by anginal attacks.

Beau described eight cases of angina due to tobacco-smoking; similar ones have been recorded by other French physicians, and one by Eulenburg. Mostly they are not fatal, and belong to the group of pseudo-angina; but in a case recorded in the 'Brit. Med. Journ.,' August, 1878, the patient died. The cases of "tobacco-heart" seen by the writer have been marked by rapid and irregular action of the organ, with a weak compressible pulse, and much nervous depression, and often by attacks of vertigo, but never by severe pain or any other anginal symptoms.

Prognosis.—The prognosis of “true” or primary angina pectoris is very grave: the attacks return, and sooner or later one proves fatal. That of secondary angina is the prognosis of atheroma of the ascending aorta and the sigmoid valves. That of pseudo-angina is favourable.

Walshe in every one of twenty-four cases examined by him during life, was able to make out some morbid change either in the heart or in the aorta, or in both. Dr Balfour says that he has never met with an instance of angina in which signs of dilatation of the heart were not present; and the late Dr Latham’s experience was that, among thirteen cases, there were only three in which neither increased dulness on percussion nor any murmur on auscultation was present; and even in those three cases the cardiac impulse was extremely feeble, while the sounds, “though natural in kind,” were “raised to their highest intonation and diffused over the entire front of the chest.”

Not only is every case of typical angina pectoris most grave in its ultimate forecast, but also most uncertain in its duration. A patient’s first seizure may have been mild, yet after a longer or shorter interval it may be followed by a fatal one.

Treatment.—In the treatment of angina pectoris more can be done than formerly. The older physicians could only recommend large doses of laudanum and brandy; but now, knowing how slowly absorption from the stomach takes place, we prefer to give morphia subcutaneously. Dr Balfour has shown that it is safe to use chloroform freely, so as to narcotise the patient; and when the remedy is at hand this appears to be the most effectual method of relief. In protracted paroxysms he follows it at once by subcutaneous injection of morphia, so that the chloroform sleep may pass into the morphia sleep, from which the patient awakes after some hours, exhausted but free from pain. It is worth remembering that patients under the influence of morphia take chloroform easily, so that one may first inject the former, and then procure chloroform for inhalation.

But we have also a specific treatment of angina pectoris by nitrite of amyl or nitro-glycerine. The advantage of the former agent is the rapidity of its action. The best way is to employ the glass capsules, each of which contains from three to five minims of it. One of them is broken within the folds of a handkerchief, and the vapour is inhaled as freely as possible. In from fifteen to twenty seconds the face flushes, a sense of fulness in the head is experienced, the pulse at the wrist loses its tension, and the pain ceases. The introduction of this valuable remedy we owe to Brunton, whose discovery was the outcome of the work of the physiological laboratory (1867). The effect of the drug is to relieve spasm of the arterioles, and thus bring down the systemic pressure.*

The allied compound *nitro-glycerine* was first tried by Dr Murrell in 1877. Its physiological action is less rapid than that of nitrite of amyl, but it may be used at the first warning of the attack, either in an alcoholic solution of 1 per cent. or in the form of chocolate tablets. In beginning the treatment it is generally best to prescribe at first half a minim or one minim of the solution. If the dose is pushed to fifteen or twenty minims, patients are liable to experience alarming effects—headache, a rushing noise in the ears, a sensation of fulness in the neck, and sometimes nausea.

* Dr Balfour found in two cases that nitrite of amyl kept in a stoppered bottle loses its efficacy in cutting short the paroxysms of angina, though it still flushes the face.

The chief use of nitro-glycerine is in preventing the recurrence of seizures. In many cases, if taken in one-minim doses from three or four to six or seven times in the day, the patient is freed from his attacks, and may be able after a time to leave off taking it, and remain apparently well, able to walk long distances and even uphill without discomfort. Dr Fagge, however, recorded two cases in which patients thus freed from their symptoms ultimately died suddenly after one of them had returned to active business on the Stock Exchange; and Dr Balfour says of nitro-glycerine that he has "used it without any benefit in the treatment of angina."

As an instance of the successful administration of nitro-glycerine in what was probably "pseudo-angina," the following case was recorded by Dr Fagge in 1882. A bank clerk, about twenty-one years of age, had for some weeks been suffering from a "sudden tightness of the chest," which would stop him in walking, so that he could not walk more than thirty or forty yards. He experienced a pain at the lower end and a little to the right of the sternum as well as near the spine at about the same level. He also noticed that he could not lie on the right side in bed without discomfort. His own impression was that the seat of the affection was in the lungs rather than in the heart. He had a pulse of 120, but this was in part due to nervousness, for it soon afterwards fell to 104. No sign of any organic cardiac affection could be detected. One minim of the solution of nitro-glycerine three times a day was prescribed, and in about a week he lost his complaint entirely.

Before the introduction of nitro-glycerine, arsenic was the medicine which proved most efficacious in warding off the seizures, and it may still be prescribed with benefit. Another drug undoubtedly useful in some cases of angina pectoris is potassium iodide.

Whatever physic is prescribed, it is essential that the patient should live by rule, avoiding whatever leads to flatulence and indigestion, never lifting weights, or walking uphill or against the wind, and shunning mental excitement, violent emotions, and passions of all kinds.

AFFECTIONS OF THE MYOCARDIUM

*“ἐν δὲ τῇ οἱ κραδίη μεγάλα στέρνοισι πετάσσει.”—Iliad, xiii, 282.**

HYPERTROPHY AND DILATATION.—*History—Physiology of the process—its estimation—its anatomy—diagnosis—physical signs—Ætiology—valvular lesions and pericarditis—renal disease and arterial sclerosis—emphysema—exercise and strain—tobacco and beer—Dilatation from specific fevers, from anæmia, and directly from rheumatism—Symptoms—Treatment*

FIBROUS DEGENERATION.—*Chronic myocarditis—Anatomy—Origin and pathology—Thrombosis—Syphilis—Cardiac aneurysm—Symptoms—Course and event.*

Acute myocarditis—Its origin, anatomy, and results.

FATTY DEGENERATION—*Fatty overgrowth and infiltration—Intra-fibrous fatty metamorphosis: its histology, causes, and symptoms.*

Granular or pigmentary, and calcareous degenerations—Rupture of the heart.

BEFORE the invention of the stethoscope, the little that was known of organic disease of the heart referred almost entirely to its bulk. It was known to be wasted in certain cases, and to be frequently enlarged, the cavities more capacious, and the walls much thickened. Valvular lesions were either not noticed or not understood. More attention was paid to the size, form, and colour of the “polypes” or masses of blood-clot which are found in the cardiac cavities and pulmonary artery after death.

Laennec, in the fourth part of his great work, which refers to the heart and vessels, puts hypertrophy and dilatation of the heart first. These subjects occupy six chapters, pp. 258—284; then follow five on atrophy, softening, induration, and fatty degeneration of the heart, pp. 285—301. “Valvular Disease, its Anatomy and its Physical signs” occupies a single chapter of only thirteen pages, while pericarditis takes 35 and aortic aneurysm 41 pages. At that time enlargement of the heart was regarded as a primary disease; but the progress of knowledge has limited idiopathic maladies in this as in other departments of medicine, and we now know that in nine cases out of ten hypertrophy or dilatation is secondary.

Hypertrophy, generally attended with dilatation, of the heart is an almost inevitable result of all the more grave affections of the cardiac valves; another frequent cause of the same changes in the right chambers is emphysema and pulmonary obstruction generally; and a third, affecting

* “Against his bosom beats his quivering heart.”—POPE.

chiefly the left ventricle, is chronic Bright's disease. There remain cases in which an adherent pericardium is associated with dilatation or hypertrophy, and those of primary hypertrophy with chronic myocarditis and fibrous overgrowth.

HYPERTROPHY AND DILATATION.—Enlargement of the heart had been described by the pathologists of the eighteenth century, and was more accurately discriminated by Corvisart, who named dilatation without hypertrophy *anévrisme passif*, and the two combined *anévrisme actif*. Laennec adopted this nomenclature. Hypertrophy with dilatation of the left ventricle, the most striking and frequent condition, was diagnosed by a strong pulse and apex-beat, diminished resonance on percussion over the cardiac area, and a red colour of the face; but Laennec admits that these symptoms may be all absent, and that *le pouls surtout est très trompeur*. He described the first sound (*la contraction du ventricule gauche*) as dull and prolonged, and what we now call the second sound (*la contraction de l'oreillette*) as very short, faint, and sometimes inaudible.

Dilatation of the auricles was also described, and Corvisart recorded a case of "partial dilatation of the heart," or as we now call it cardiac aneurysm. Induration as well as softening of the muscular substance of the heart, atrophy, and fatty degeneration were all recognised by Laennec.

Theory of hypertrophy.—Increased activity of an organ produces, as a rule, hypertrophy, though the process is probably not always the same. The necessary condition for overgrowth is no doubt hyperæmia, and this probably begins as the result of increased waste of tissue from exertion. The passive tissues hypertrophy either in direct consequence of hyperæmia, as the clubbed fingers of chronic cardiac disease and of long-continued venous congestion generally; or from some unknown stimulus, as in the overgrowth of the bones seen in osteitis deformans, or of the spleen and lympharia in leuchæmia and Hodgkin's disease. Of the active organs, those of the nervous system, whether belonging to the central, the conducting, or the peripheral and receptive apparatus, appear to be incapable of true hypertrophy, however much exercised: the so-called cerebral hypertrophy (vol. i, p. 850) is probably always a form of interstitial sclerosis. Glands hypertrophy from the stimulus of over-use, as the liver in beer-drinkers, the kidneys in diabetes, or one kidney when the other has been destroyed by some local disease.

The voluntary muscles hypertrophy remarkably—by increase of the fibres both in number and in size—in response to exercise; but the effect of training in removing interstitial fat is often sufficient to mask the true muscular development and make the hard and powerful muscles of an athlete less bulky than those of an indolent free-liver. Moreover, in this, as in other physiological developments, the degree of change is limited by the law of each individual organism. However well nourished and well worked, the muscles will not hypertrophy beyond a certain point for each man, just as the possible degree of speed or flesh-forming is limited for each horse or ox, and the possible attainments of the intellect for each human being.

Involuntary muscular fibre undergoes hypertrophy from over-use, at least as readily as that which is set in action by the will: the ciliary muscle grows larger as the lens becomes less elastic; the bladder hypertrophies behind a stricture; the intestine, the stomach, or the œsophagus above a

chronic obstruction. In like manner the ventricle or the auricle hypertrophies as the result of narrowing of its orifice of egress, by which the discharge of its contents is obstructed. In the left auricle and left ventricle this is frequently seen; on the right side of the heart, stenosis of the pulmonary or tricuspid orifice is rare; but when present, the same effects are produced, except that for some unknown reason hypertrophy of the right ventricle is marked by more hardening and closeness of texture, with less increase of bulk, than that of the left.

The obstruction need not be from narrowing of the ostia through which each cavity discharges its contents; it may depend on mechanical hindrance to the flow of blood further on. Mere dilatation of the arch of the aorta can have no effect of this kind, but this dilatation is always associated with loss of elasticity; and rigid inelastic arteries lead indirectly to hypertrophy of the left ventricle. Increased friction from roughness of the tunica arteriosa intima, contraction of the arterioles, obstruction in the capillaries from external pressure, retardation of the flow in the veins from deficient muscular exercise or deficient respiratory movements—all these conditions increase intra-ventricular blood-pressure, and thus stimulate the muscular walls to hypertrophy. In the same way obstruction in the pulmonary capillaries produces hypertrophy of the right ventricle.

There is no reason to regard hypertrophy of the heart as an effort of nature to provide sufficient blood for the system any more than the "explanation" of hypertrophy of the bladder is that retention of urine is harmful. This process, like that of inanition, is in some cases advantageous to the organism, in other cases injurious; the termination of life, as much as its commencement, is a physiological, natural, pre-ordained event; and the business of pathology is to discover the physiological laws under which the organs act when the conditions of "health" are changed for those of "disease."

Now the law for all muscular tissue is that, under suitable conditions of nutrition and blood-supply, it responds to the stimulus of opposition by increase first of energy, and then of bulk. The increased vigour of contraction of a frog's gastrocnemius when weighted answers to the increased vigour of the skeletal muscles when the limbs are put to hard work, and the increased force of contraction of a muscular cavity when distended by its contents. Only, in each case the nutrition of the muscle must be good, and the task put on it of moderate magnitude.

Dilatation.—Increased stimulus, however, does not always call forth adequate hypertrophy. Impaired nutrition of the muscular fibres, from want of food, from imperfect digestion and absorption, or from loss of blood, may prevent them responding as they should; just as labour and gymnastic exercises, if undertaken by the ill-fed, the anæmic, or the invalid, or if disproportioned to the powers of childhood or old age, will not cause hypertrophy of voluntary muscles, but rather atrophy and increased weakness. The same result is seen if the labour is too hard and the tasks too heavy.

Moreover, increased fluid pressure will always tend to dilate the walls of the containing cavity, even when its elastic as well as its contractile force is increased by hypertrophy of its muscular coats. Accordingly, some degree of dilatation is rarely absent, even when compensatory hypertrophy is well developed. This is seen in the dilated stomach which follows pyloric stenosis, the dilated œsophagus which forms a pouch above a stric-

ture, the dilated intestine above a chronic obstruction, and the dilated renal pelvis, ureter, and bladder, which result from stricture of the urethra.

Hypertrophy and dilatation.—In the heart, dilatation may occur with but little hypertrophy, particularly in the auricles, from mitral or tricuspid regurgitation; and hypertrophy may occur with little or no dilatation, as in uncomplicated cases of chronic Bright's disease; but as a rule the two conditions are met with together, both at the bedside and in the deadhouse. Nevertheless we can, in the majority of cases, recognise as the leading process (better perhaps by studying the physiology of the circulation during life than the cardiac anatomy after death) either the compensatory and favourable one of hypertrophy, or the ingravescent and unfavourable one of dilatation.

The hypertrophy of the cavity of the heart which lies next behind a narrowed orifice is, we have seen, a physiological and vital result of the increased pressure within it which has to be overcome. The dilatation which follows the same condition is a passive and physical result, the mere stretching of the muscular walls impairing their elasticity, as we see in the dilatation of an old glove.

The increased blood-pressure due to narrowing of an orifice of exit from a cardiac cavity is obvious, that due to regurgitation into it will be explained when we come to consider the effects of valvular lesions on the cavities of the heart in detail. That from atheroma of the arteries we have already spoken of, and that from chronic renal disease depends on raised blood-pressure in the whole systemic system of arteries, the origin of which will be considered in the chapter on Bright's disease.

There is one form of obstruction which is less obvious because it is scarcely pathological. It is that caused by over-exertion in what often appears to be healthful labour or athletic sports. Each contraction of a muscle, if extreme, and particularly if long sustained, causes pressure on the arterial trunks which pass through or beside it.* This leads to increased blood-pressure in the aorta and left ventricle, and becomes a stimulus to cardiac hypertrophy and a cause of dilatation of the heart and aorta, as well as of irritative arteritis of the latter.

The healthy heart naturally possesses a considerable amount of reserve force beyond that which is required to carry on the circulation under ordinary conditions. Hence, although the strain upon it is augmented by muscular efforts of all kinds, it is generally able to meet the call without suffering damage. Even if its walls yield to the increased pressure which they have to bear, the dilatation is, in most cases, only temporary; they generally recover themselves when the body returns to a state of rest, and the blood-current resumes its usual tranquil course.

But such is not always the case. It sometimes happens that violent or frequently repeated exertion leads to permanent enlargement of the heart. As in other cases, we must suppose either that the strain is too severe, or too prolonged, or too frequent, or else that there is deficient power in the muscular fibres, and this may be sometimes traced to previous disease, or to an insufficient supply of food.

Physiological dilatation from increased blood-pressure and rapid action of the heart affects the right ventricle, and is relieved by the "safety-valve action" of the tricuspid valve, as explained by the late Wilkinson King in

* The smaller arterioles which supply the muscle itself are probably protected by this physiological dilatation at the moment of stimulus of the motor nerve.

the 'Guy's Hos. Rep' for 1837 (1st series, vol. ii, p. 104). A more serious and less frequent condition is for the left ventricle to dilate, but this also admits of recovery. If it persists it may produce a permanent tachycardia and palpitation with irregular pulse, and finally, the condition of arterial anæmia and venous congestion, which is the natural physical result of all diseases which impair the efficiency of the heart as a forcing-pump.

Physiological hypertrophy from "overstrain," without rheumatism or ill-nourishment or diphtheria having previously damaged the muscle, is less liable to become permanent. It probably ensues naturally in every young man who undergoes training for athletic exercises. Just as the chest expands, and the skeletal muscles grow and harden, and the nervous system improves in alertness, stability, and co-ordination, so the left ventricle becomes thicker and the heart less liable to be disordered by sudden calls upon its powers. In such men, who keep within the limits of what, for them, are moderate efforts, we may often notice that the impulse is strong and somewhat heaving, the apex a little further to the left, the pulse full, strong, and not readily quickened by excitement or emotion.

Here, again, however, what begins as a physiological process may become pathological. The hypertrophy oversteps the limits of health, and along with the same physical signs we have a rapid and sometimes irregular action of the heart, an easily excited pulse, and liability to dyspnoea or faintness on even moderate exertion. That this is sometimes the effect of too severe, or too long-continued, or too frequent efforts, appears certain, although it is difficult to bring direct proof of the fact.

In the cases now discussed, the hypertrophy of one ventricle or auricle, or of all four cavities of the heart, is true overgrowth of the muscular tissue; and the dilatation is passive enlargement of the cavity with only comparative thinning of its walls. Hypertrophy with interstitial fibrous overgrowth, and dilatation with fatty degeneration, or with adipose overgrowth and local or general attenuation of the parietes, are conditions that will be referred to separately (pp. 210, 216).

Definitions.—As regards the application of terms, which has sometimes been confused, it seems reasonable that "simple hypertrophy" should mean an increase in the thickness of the heart's muscle, its cavities remaining unaltered in capacity; "simple dilatation," an increase in their capacity, the amount of muscle remaining stationary. The former is frequently observed as a result of Bright's disease; and although the latter is seldom if ever seen in the deadhouse, it must be supposed to occur at the beginning of many cases of what afterwards appears as dilatation with some hypertrophy. In almost all cases consecutive to valvular lesions, or pulmonary stricture, and in many consecutive to chronic renal disease, the condition is one of dilatation with hypertrophy, or hypertrophy with dilatation.

"Concentric hypertrophy," or thickening which encroaches on the cavity of the left ventricle, probably does not occur. It is said that the description of concentric hypertrophy given by Corvisart and followed by other French pathologists, including Laennec, was influenced by the number of hearts examined, during the reign of terror, of persons executed by the guillotine: for after rapid death from hæmorrhage in healthy subjects the left ventricle contracts closely, and looks smaller and much thicker than in ordinary cases.

A dilated heart is said to be hypertrophied if its weight as a whole is above the normal standard, even though every part of its walls may be

below the natural thickness. When hypertrophy and dilatation coexist, it is to the latter rather than to the former that the case approximates clinically.

Measurements.—In order to ascertain whether a heart is hypertrophied, one has only to weigh it after it has been emptied of blood, and separated from the great vessels and pericardium. According to Peacock ('Reynolds' System,' vol. iv), the ordinary range of the weight of the organ is, in men who have died from acute diseases, from nine to eleven ounces; with chronic diseases, from eight to ten ounces; in women affected with acute disease, from eight to ten ounces, with chronic disease, from seven to nine ounces. But in large and powerful men, who have been killed by accident or have died after a short illness, the weight may sometimes be as much as twelve ounces, or even more.

The most frequent weight, a more important fact than the average weight, of the heart in men is 10 oz. or a little under (Peacock) or over (Reid) that weight. The most frequent weight in women is 9 oz. After adult life is reached, the heart still continues to increase in size and weight up to 30, and even later, though with rapidly diminishing increments.

It is much less easy after death to determine the presence of dilatation, at least in its slighter degrees; for when well marked it is obvious enough.

When in a hypertrophied heart the chambers seem to be of normal capacity, the question remains, whether, in a fully relaxed state of their walls, they might not be larger; in other words, the anatomical proof of hypertrophy without dilatation is far from simple. Peacock gave the following as the normal measurements. The figures in the second and fourth columns are those given by Bizot.

Thickness of the—	<i>Men.</i>				<i>Women.</i>			
	Millimetres. P.		Mm. B.		Millimetres. P.		Mm. B.	
Walls of the right ventricle: base	4.16	...	4	...	4.16	...	3	...
" " " midpoint.	4.35	...	3	...	4.5	...	2	...
" " " apex	3.19	...	2	...	2.92	...	2	...
" left " base	11.58	...	10	...	11.02	...	9	...
" " " midpoint.	13.15	...	11	...	12.6	...	10	...
" " " apex	5.4	...	8	...	5.62	...	7	...
Septum between the ventricles	12.89	...	11	...	10.57	...	9	...

The weight of a hypertrophied heart, without either valvular lesions or Bright's disease to account for hypertrophy, rarely exceeds fifteen ounces; but in one case observed by Dr Fagge it reached thirty-three ounces without any obvious cause. In cases of valvular or renal disease, the heart sometimes reaches a weight far above the figures just quoted.

In most cases we have no means of determining the rate at which the heart enlarges; probably the process is often slow and gradual. But when the starting-point of secondary hypertrophy can be fixed, it has sometimes appeared to be extremely rapid. Dr Stone ('Lancet,' 1879) related two examples of heart disease following injury, in each of which, if the heart was healthy at the time of the accident, it must have gained weight at the rate of nearly an ounce a week during the five months that elapsed before the patient's death—a rate more rapid than the increase in size of the pregnant uterus. Dr Goodhart ('Path. Trans.,' vol. xxx) published a case, the history of which would imply that within three or four weeks the heart grew to a weight of nineteen ounces, as the result of pericarditis.

There has been much discussion as to whether cardiac hypertrophy depends upon an overgrowth of existing fibres or upon a formation of new

ones. Schroetter (in von Ziemssen's *Handbuch*) states that the fibres measure 0·03 mm. in a hypertrophied heart, as compared with a normal thickness of 0·007 mm. Friedreich also is said to have arrived at 0·025 mm. as the mean of ten measurements of the fibres of a hypertrophied left ventricle. But Rindfleisch says that he failed to discover any such difference, and his conclusion is that the fibres (which, it must be remembered, branch as skeletal fibres never do), undergo a further splitting up, which leaves them apparently of the same size as before.

The order in which dilatation and hypertrophy are developed is not always the same. In weak subjects, or in subjects with "weak" hearts, or when the strain is peculiarly severe and sudden, it is probable that dilatation occurs first as the result of over-distension of the cardiac muscle, and that hypertrophy follows later when rest allows of natural recuperation in young and more or less healthy patients. In other, and probably more numerous, cases, hypertrophy appears to be the primary change. In either case, what brings about a failure of the heart's function is progressive dilatation, to which the hypertrophy is no longer adequate.

It has been supposed that breakdown of a hypertrophied ventricle depends on the supervention of fatty degeneration. But Cohnheim ('*Vorlesungen*,' vol. i, p. 72) thought the frequency of fatty degeneration in hypertrophied hearts exaggerated, and believed that in most cases there is mere "fatigue" or "exhaustion" of the fibres. Dr Allbutt ('*St Geo. Hosp. Rep.*,' vol. v) has drawn attention to the interesting fact, that in the file-cutters of Sheffield, who are constantly using the arm in rapid flexions, the biceps muscle undergoes great enlargement, but that after a few years it again wastes, and falls below its normal size.

Diagnosis.—The clinical recognition of cardiac hypertrophy and dilatation rests partly upon percussion, partly upon inspection and palpation, and in a smaller degree upon auscultation. It is one of the more difficult conditions to determine by physical examination, and it is but little aided by general symptoms; so that more mistakes are made on this point than on any other in the diagnosis of diseases of the chest.

By *percussion* we seek to determine to what extent the heart comes in contact with the anterior wall of the chest; but the results depend not only on the state of the heart, but also on that of the lungs. If the lungs are emphysematous, the area of dulness due to the heart may be normal or diminished, notwithstanding that the organ is enlarged; if the lower and anterior part of the chest on the left side is flattened, or if the corresponding part of the lung is collapsed, the cardiac area may be increased, though the heart is no larger than natural. There is even a difference in the extent of cardiac dulness, according as the breath is drawn deeply in or forced out, so that for accurate percussion the patient should breathe gently. To arrive at a satisfactory result one should first use light, but careful and deliberate, percussion, and then make a series of marks with an aniline pencil upon the patient's chest, indicating the different points at which dulness begins to pass into resonance round the circumference of the organ; and after drawing the figure, the outline should be tested and corrected by fresh percussion, pausing after each stroke of the fingers to listen, and repeating the stroke until the resonance or dulness of the note elicited is certain. The process demands both time and care.

In health a more or less triangular figure, representing the extent of an

absolutely dull note, is thus obtained. Above it forms an angle at the upper border of the fourth left costal cartilage, close to the sternum. From this point two diverging lines are traced downwards. One of them corresponds with the left border of the sternum, for although the right ventricle, uncovered by lung, lies beyond this line as far as the median line, the tone yielded by the bony tissue when percussed masks the cardiac dulness enough to prevent accurate definition of the space occupied by the heart on this side.* The other line extends downwards and outwards, passing to the inner side of the nipple until it reaches a point at which the apex of the heart can be felt beating. Along this line the transition from dulness to resonance takes place gradually, so that above it one can draw another line running more or less parallel and about half an inch distant, which indicates the upper limit of partial dulness, as the lower one indicates the upper limit of absolute dulness; this upper line begins above at the level of the third rib. To complete the triangle, a base-line is drawn from the lower end of the sternum to the point at which the heart's apex beats. It can scarcely be traced by percussion, because the cardiac dulness passes insensibly into that caused by the left lobe of the liver.†

It must be added that when the stomach is distended with gas, the tympanitic percussion sound is often transmitted beyond the region of the stomach, for dulness spreads. To prevent this the stroke should be light and made from the wrist.

The statements above made as to the natural extent of the cardiac dulness are applicable only when the patient is standing upright or lying on his back. When he lies over to the left, that side of the triangular area shifts more or less in different persons to the left; and this is more marked when the heart is hypertrophied.

When the heart is enlarged, the upper angle of the area of cardiac dulness remains at the fourth rib or the third space. If it reaches as high as the second, there is probably some morbid condition beyond hypertrophy and dilatation—perhaps aneurysm, or pericardial effusion, or mediastinal tumour. The position of the two sides of the triangle deviates much more from the normal. The dulness may reach to the right border of the sternum, or half an inch or still further to the right; this is an indication of increase in size of the right ventricle, or of displacement of the entire heart. Or the dull area may reach as far as the nipple, or still further to the left, and may also extend much lower than usual, until the apex-beat lies in the sixth or even in the seventh interspace; this is a sign that the left ventricle is enlarged, or that the heart is displaced to the left as by effusion in the right pleura, or downwards as by an aneurysm of the aorta.

If the right ventricle is enlarged, the apex-beat is pushed outwards, and pulsation may also be felt in the epigastrium. When the right auricle also is enlarged, as it usually is in cases of chronic bronchitis and emphysema with tricuspid dilatation, it causes a considerably increased dulness on percussion, to the right of the sternum in the fourth space. In consequence of this displacement of the two lines marking the sides of the dull cardiac area, this acquires, when the heart is enlarged, a more quadrilateral and

* The sternum (as stated by Guttman) may be rendered less vibratile if the hand be laid over the upper part of it; or if an assistant press firmly with the hands placed upon the costal cartilages on each side a dull sound may be obtained over the cardiac area behind the sternum.

† Sometimes, however, as Dr Gee says, a distinct heightening of pitch and increase of resistance can be made out in passing from the cardiac to the hepatic dulness.

less triangular form. If the left ventricle alone is enlarged, it will grow downwards and to the left, so that its impulse will be felt in the sixth or seventh space, and much outside the nipple-line.*

For practical purposes enlargement of the left ventricle may be determined by tracing one diagonal line from the inner end of the fourth left costal cartilage downwards and outwards to the apex-beat, and another line at right angles from the base of the ensiform process outwards and somewhat upwards to the point of impulse again. Normally the first line should not exceed two and a half inches in length; when the heart is much enlarged it may be four or five inches. The second line should measure about one and a half inches; it may be increased by disease to over two inches.

Even when the lungs are neither emphysematous nor retracted, percussion does not give an infallible indication of enlargement of the heart; for great hypertrophy of the left ventricle may be present in cases of Bright's disease without there being any increase in the area of dulness. The heart seems to bury itself in the hollow of the left lung, so that it is not more widely in contact with the chest wall than in health. Thus in cases of cerebral hæmorrhage, one often finds at the autopsy an enormously hypertrophied heart, which the day before it was impossible to detect, though expected and sought for.

Enlargement of the heart may sometimes be discovered by inspection or palpation; and displacement of the apex-beat to the epigastrium, downwards, or to the left, is appreciable by the eye as well as the hand.

When we have determined that the heart is enlarged, the question still remains how far the enlargement is due to dilatation or to hypertrophy; and the solution of this question, so far as it is soluble, is sought by careful examination with the eye and with the hand and finger. In both cases the heart's impulse is seen and felt more widely than that of the natural "apex-beat." When there is great hypertrophy, the impulse is often diffused, laboured, and heaving, so that even a stethoscope, with the observer's head resting upon it, is lifted as though by an irresistible power. In some cases, while the fifth and sixth ribs are pushed forwards, other parts of the chest-wall are sucked in.

The deliberate heaving impulse of hypertrophy and the short slapping impulse of dilatation are both characteristic to the *tactus eruditus*.

In cases of nearly pure dilatation the impulse becomes (to use Walshe's words) "either a short feeble slap, followed by a sudden fall back of the organ, or a more prolonged faint tremulous motion." In the latter case it often has an undulatory character to the eye. Another peculiarity of the heart's action which indicates dilatation rather than hypertrophy is irregularity in the force of successive beats, or in their rhythm.

It is disputed whether enlargement of the heart ever causes increased prominence or bulging of the præcordial region and widening of the intercostal spaces, as compared with the opposite side of the chest. Schroetter, following Skoda, maintains that it occurs only when there is also pericarditis, by which the textures forming the chest wall are softened.

Little direct information as to the existence of hypertrophy or of dilatation is yielded by *auscultation*. But it is to be particularly noted that in

* Walshe taught that distension of the right auricle contributes to the increase of dulness to the right of the sternum, that distension of the left auricle may cause deficient resonance in the third and second left intercostal spaces; and that an impulse of presystolic rhythm in one of these positions may be due to enlargement of one or the other of the auricles.

cases of simple hypertrophy, with little or no dilatation, the first sound is not louder than usual, but fainter.* It is often dull and muffled. In cases of simple dilatation the first sound is often loud, though if the muscle is soft and flabby it may be weak; but, whether faint or loud, it is shorter, and has more tone than in health, that is to say, it is more like a second sound. When dilatation and hypertrophy occur together, the first sound is often widely audible over the surface of the chest.

Any alterations in the second sound depend upon the state of the arterial tension, and afford only indirect indications of the condition of the heart itself.

One or both of the cuspid valves may be rendered incompetent as a result of the widening of orifices which necessarily takes place as either of the ventricles dilate, and a systolic regurgitant murmur may result.

Seitz ('Deutsch. Arch.,' xi, xii) drew attention to the fact that the movements of an enlarged heart within the pericardial sac are sometimes attended with grazing sounds, like those produced by pericarditis. In one case observed by him these continued up to the time of death, and no doubt was entertained as to the existence of pericardial inflammation; yet at the autopsy there was no thickening or opacity of its surface. Walshe, too, speaks of "rubbing additions to the first sound at the apex" as not very uncommon. One such instance occurred several years ago at Guy's Hospital, and led to a diagnosis of pericarditis; but on *post-mortem* examination none was found; and Dr Sturges once met with a similar surprise. We occasionally have the same experience in the case of pleurisy (vol. i, p. 1096).

Ætiology.—(1) Among causes of secondary hypertrophy stand first the lesions of the cardiac valves. All obstructions to the flow of blood lead to over-pressure behind and under-pressure in front of the obstruction by the common laws of hydraulics. If the obstruction is in the systemic capillaries the stress will fall on the aorta and left ventricle in systole, if it is in the aorta or in the sigmoid orifice it will fall on the left ventricle. If the obstruction is at the mitral orifice the stress will fall first on the left auricle during systole, and next upon the pulmonary veins and capillaries. If it is in the lungs or in the pulmonic (dextro-sigmoid orifice) the stress will fall on the right ventricle; if, lastly, as we occasionally find, there is stenosis of the tricuspid orifice, the increased pressure falls on the right ventricle and the great systemic veins. The result is, in most cases, firstly, hypertrophy as a physiological, and, secondly, dilatation as a physical effect.

Regurgitation through the aortic or mitral orifices by reason of the incompetent valves also causes over-pressure in the cavity behind, and under-pressure in the cavity in front of the leaking valve, but the over-pressure is most during diastole, and there is no direct impediment to the auricle or the ventricle emptying itself in systole. So that here there will in most cases be dilatation from the first, and only later and imperfect hypertrophy.

The compensatory hypertrophy of aortic stenosis is in the walls of the left ventricle, of mitral stenosis in those of the left auricle, of that of mitral regurgitation is not in the left auricle, but in the right ventricle, which hypertrophies to overcome the obstruction in the pulmonary capillaries.

(2) Of late years it has been recognised that an important cause of

* So Laennec, and so Walshe, who describes it as "prolonged and weakened, sometimes almost to actual extinction, the sensation reaching the observer's ear being rather one of impulse than of sound." But every beginner is surprised that a large muscle does not produce a loud sound.

cardiac hypertrophy or dilatation, apart from valvular obstruction or incompetence, is extensive pericardial adhesions occurring in childhood, while the active growth of the heart is going on. This subject will be again considered among the effects of pericarditis.

(3) The most chronic form of Bright's disease, that associated with contracted kidneys, is the one which leads to cardiac hypertrophy. There is high blood-pressure in cases of acute nephritis, but they do not last long enough to cause hypertrophy of the left ventricle; and in chronic cases of tubal nephritis the pressure almost always relaxes. What is the cause of the high pressure is still a difficult question, and will be considered in the chapter on Bright's disease. The effect of the arterial tension is to call forth hypertrophy of the systemic ventricle exactly as it is called forth by aortic (sigmoid) stenosis. Here dilatation is always secondary to hypertrophy and marks the later stages of the disease, a fact which supports the doctrine stated above, that the immediate effect of obstruction (whether valvular or capillary) is, as a rule, hypertrophy, not dilatation of the cavity next behind it.

At the bedside it is often difficult and sometimes impossible to determine whether the state of the kidneys accounts for the cardiac symptoms, or whether there is a valvular lesion to which they may be due, or whether there is primary hypertrophy and dilatation of the heart. Even in the deadhouse the very same points may remain doubtful.*

(4) Another common and well-recognised cause of cardiac hypertrophy and dilatation, when it affects the right side of the organ, is obstruction to the pulmonary capillaries, either due to emphysema, to chronic bronchitis, to bronchiectasis, cirrhosis of the lung, neglected empyema, or very chronic fibrous tuberculosis.

(5) Another possible cause has been found in extensive pleural adhesions. But it seems doubtful whether the cases supposed to be of this kind which have been recorded by Bäumlér and by Brüdi ('Deutsches Arch.,' xix) really warrant the conclusion. It must be remembered that the right side of the heart is subject to hypertrophy and dilatation from mere abolition of large capillary tracts, as in carnification of the lung by pleural effusion.

(6) It seems to be pretty clearly shown that aneurysm of the aorta, not implicating the sigmoid valves, has no effect in causing hypertrophy of the left ventricle.

We now come to the direct causes of enlargement of the heart, independent of valvular lesions or disease of the kidneys or of the lungs.

(1) One cause of what may properly be termed relative hypertrophy of the heart is derangement of the natural growth of the organ about puberty. The heart, like the brain, does not develop at a uniform annual rate; but grows rapidly up to the sixth or seventh year, and again between fifteen and twenty. *La mégalo-cardie de croissance* has been described by Sée and other French physicians. It is often seen in children under puberty who are put to work above their strength, or in schoolboys who tax their powers in athletic contests beyond their strength. There is often a quick pulse and palpitation, sometimes dyspnoea, and frequently headache. The physical signs of hypertrophy are present, and occasionally a systolic

* The kidneys may be of good size and not unhealthy in look, and yet may be shown by the microscope to have undergone changes so extensive as fully to account for any enlargement of the heart that may be present.

apical bruit. When the pulse is rapid and irregular, they come under the description given above (p. 198), and are successfully treated by more or less prolonged rest, varied by strictly regulated exercise. The most useful drugs are digitalis or strophanthus or the two combined, and also either steel or, if that fails, arsenic.

(2) A clear and unquestioned cause of hypertrophy of the heart in adults is *over-exertion*. Da Costa, in his observations on 'Irritable Heart' among soldiers in the American war (p. 5), states that in twenty-eight out of a hundred cases there was evidence of hypertrophy. In one of them death occurred from strangulated hernia eleven months after cardiac symptoms began; the left ventricle, though not apparently larger than natural, had its walls seven eighths of an inch thick at the thickest part.

Fränzel ('Virchow's Archiv,' 1873) recorded a like affection in nineteen soldiers engaged in the Franco-German war, especially in those who took part in the arduous march to Orleans, or in the attack upon Belfort.

In 1870 Dr Myers published an essay in which he showed that cardiac affections in general were more common in soldiers than in sailors, and in the Foot Guards (who are chiefly stationed in London) than in the men of the Metropolitan police. In a large proportion of cases he found neither valvular nor aortic disease, but tachycardia and palpitation of the heart, leading after a time to its enlargement (cf. *supra*, p. 172). The general opinion formerly was that the cause lay in the crossbelts, heavy accoutrements, and tight clothing which the men used to wear, and by the urgent advice of Maclean and Parkes the old form of knapsack was abolished, and a "valise equipment" was adopted in its stead. Myers laid stress upon the effect of the tightness of the tunic collar, and this inconvenience was also removed. Dr Veale ('Army Med. Dep. Report,' xxii) stated that cardiac hypertrophy with tachycardia and palpitation, which is its most conspicuous symptom, are still very frequent; and probably the true solution of the difficulty was found by Surgeon F. A. Davy, who referred it mainly to the "setting-up drill," during which recruits are compelled to "swell the chest" so as artificially to expand it ('Army Med. Dep. Report,' xviii), and prevent free expiration. It appeared that in soldiers standing under drill, the frequency of the respirations increased to about 40, and the pulse to 110 in the minute; that the heart's rhythm was often disturbed; and that the impulse was more forcible, and was felt over a wider area than natural.

Among civilians the ill effects of over-exertion on the heart were fully recognised by Dr Peacock in reporting, in 1864, upon the health of the miners in Cornwall, who, besides heavy hammer-work in the day, have to climb ladders of immense height in order to get out of the pit every evening. He found that many of them suffered from cardiac dilatation.*

In a series of articles in the 'Deutsches Archiv' for 1873 and 1874, Seitz showed that at Zürich cases are not infrequent of cardiac affection, which after death are found to be due to a primary enlargement of the heart. The patients were almost all men, and engaged in heavy labour of one kind or another. Again, Münzinger, in the 'Deutsches Arch.' for 1877, described as "the Tübingen heart" a similar affection without any valvular lesion. It occurs both in men and in women who work as labourers in vineyards on the slopes of hills, up which heavy burdens of manure have to be carried. A

* The cases described by Dr Allbutt in the 'St George's Hospital Reports,' vol. v, p. 23, hardly fall into the present category, for he assigned an important share in the sequence of events to chronic changes in the aorta and its valves.

point on which he laid stress was that these poor people are ill-fed, living on potatoes and puddings, and scarcely ever tasting meat. Dr Allbutt, too, regards insufficiency of food as important in the ætiology of cardiac affections due to overwork and strain, especially taking long and active exercise while fasting; and he justly remarks that one reason why young men of the upper and middle classes do not more often suffer ill effects from athletic sports is their good food. The same remark applies, we may add, to navvies and other men engaged in severe labour, who always live well.

In the young and vigorous there is a reserve force which is equal to any reasonable demands; but in weakly, ill-fed persons, in convalescents from acute illness, and in the old and decrepit, the heart may fail under slight efforts.

(3) Beside long-continued severe exertion, there is little doubt that more transitory muscular efforts may produce effects which chiefly show themselves in a condition of more or less marked hypertrophy of the left ventricle.

Prof. Laache, of Christiania, has drawn attention to such cases in his monograph, '*Recherches cliniques sur quelques affections cardiaques non-valvulaires*' (1895), and has recorded some remarkable instances of hypertrophy with dilatation following a single muscular effort, as in walking (case No. 10), in some cases complicated by the patient being of intemperate habits. But in the striking case given on pp. 12-13, one may surely believe that the mitral stenosis discovered in a fisherman's wife a year after great exertion in rowing her boat to escape a threatening storm, must have been present before the effort was made; and that the hæmoptysis which immediately followed on her arrival in port was due to this valvular lesion, up to that time compensated, breaking down under an unusual strain.

Similar cases of cardiac hypertrophy without valvular disease that the writer has met with have been all of a transitory nature. The symptoms—rapid and irregular pulsation, with palpitation—following a sudden effort, as in running a race, rowing, or lifting a weight, have yielded to time and treatment.

In other cases he has observed an impulse displaced to the left, with a heaving character and increased cardiac dulness, in athletic young men who had no sign of disease of the valves or of the kidneys, and no palpitation, dyspnoea, tachycardia, or other symptom of cardiac disorder. These seem to be cases of physiological hypertrophy, and the conditions appear gradually to pass away afterwards, as the large muscles of an athlete in training resume their ordinary dimensions as soon as they are no longer kept working under unusual pressure. This physiological cardiac hypertrophy is also met with in women during pregnancy, and it subsides after delivery.

(4) The effect of *tobacco-smoking* on the heart has long been recognised, and Traube brought evidence to show that it not only caused tachycardia and irregular pulse, but also hypertrophy. In all cases of irregular pulse and short breath, we should inquire as to this possible toxic cause of the disorder, for when the habit is given up the most remarkable improvement follows.

(5) Of late years we have recognised as an undoubted cause of cardiac hypertrophy excessive *beer-drinking*. This was, not unnaturally, first observed in Munich by Bollinger; but Aufrecht and other physicians in Germany have recorded cases, as have Laache in Norway, and several physicians in this country (see an article by Dr Theodore Fisher in the '*Guy's Hosp.*

Rep.,' for 1894, vol. li, p. 97; one by Dr Beddard in the 'Clin. Journal,' 1897, p. 130; and another by Dr G. A. Gibson in the 'Journal of Pathology,' November, 1894, p. 37). It seems clear that ardent spirits do not produce the same effect, and in eight fatal cases at Guy's Hospital, where the heart weighed from fourteen to twenty-two ounces, there was no cirrhosis of the liver. Whether excessive water-drinking would have any effect of the kind has not been ascertained; but to the beer may probably be ascribed the fatty or more frequently fibrous degeneration of the heart which is usually present.

The late Prof. Roy, in conjunction with Prof. Adami (now in Canada), published an interesting paper in the 'Transactions' of the Royal Society for 1892, in which they showed that apart from direct action of alcohol on the cardiac muscle, alcohol may act by producing peripheral neuritis of the vagus; and this has been shown to occur, apart from the experimental evidence on animals by Dr Sharkey ('Path. Trans.,' vol. xxxix, p. 27), in a case where the affection of the vagus led to persistent tachycardia and death.

Among causes of dilatation without hypertrophy, may be first mentioned *anæmia*, which often produces a fatty change in the muscular fibres of the heart, and may thus lead to subsequent yielding of its walls. The question was discussed by Dr Goodhart ('Lancet,' vol. i, 1880); and he showed that in women who are suffering from chlorosis the heart's impulse is diffused and displaced outwards, and that when anæmia proves fatal the left ventricle is found to be dilated.

After exanthemata and other *febrile diseases*, hypertrophy is rare, but dilatation is very frequent. Pyrexia is known to damage the muscular tissues, and Dr Goodhart recorded ('Guy's Hosp. Rep.,' xxiv) four or five instances in which sudden or nearly sudden death occurred during scarlatinal dropsy, and in which the heart was found dilated or fatty. Dr Lees has published numerous observations to the same effect, showing that during diphtheria, influenza, and enteric and other fevers, the heart may become for the time dilated, and that it does not always recover after convalescence ('Brit. Med. Journal,' January 5th, 1901). The cardiac sounds rapidly become weak, and the first sound comes to resemble the second until a *tic-tac* only can be heard, while the pulse is small, compressible, and feeble. These threatening symptoms pass off under alcohol and strychnia, and the patient may recover; but in other cases they may end in death, and at the autopsy the left ventricle alone, or the whole heart, has been found notably dilated. The writer met with a striking case in a boy about seven years old, who died during convalescence from diphtheria with the above symptoms, and in whom the heart was found much dilated without hypertrophy, with no valvular disease or pericarditis, and with no disease of the kidneys or the lungs.

Apart from the effect of febrile toxins in general on the heart, there is a special liability to affection of the muscle as well as the endocardium and pericardium as the result of acute *rheumatism*, and this may lead to cardiac dilatation.

A girl aged eleven, in Guy's Hospital, died of cardiac dropsy six months after a rheumatic attack. The most conspicuous lesion was dilatation of the left ventricle, which had reached such an extent that, although its walls measured only from one eighth to a quarter of an inch in thickness, the organ weighed ten ounces. There were no signs of

pericarditis. There had probably been regurgitation through the mitral orifice, for the papillary muscles were much wasted, but the valve itself was healthy or only slightly thickened.

Dr Goodhart and Dr S. West have brought similar cases before the Pathological Society, and Dr Lees and Dr Poynton read a joint paper on the subject before the Royal Medical and Chirurgical Society (June 28th, 1898). They found notable dilatation in 92 of 150 cases in children who died from rheumatism, and indication of the same dilatation during life. The writer has lately met with an unusually complete case, in which a boy of twelve, under the care of Dr Curwen, of Brighton, after a fresh attack of rheumatism, developed rapid dilatation of the left ventricle, which before was certainly not enlarged.

Symptoms.—The symptoms of primary enlargement of the heart vary widely in different cases. In the earliest stages they consist in palpitation and subjective sensations of pain or discomfort in the cardiac region, partly in an increased frequency of pulse, which is often irregular in rhythm.

Another symptom, which is absent or slight in the merely functional diseases of the heart, is dyspnoea. This at first comes on during exertion only; the patient finds that he cannot walk so quickly as before with comfort, and that going uphill or ascending a flight of stairs makes him feel short of breath. From this condition there are all gradations up to a point at which even the slightest movement becomes difficult.

In health muscular exertion makes the beats of the heart more frequent and calls for greater vigour of systole; and in cases of enlargement of the heart, after slight exertion or excitement the pulse becomes accelerated.

The fact that there is a sensation of dyspnoea, even when the left ventricle alone is hypertrophied, supports the view which has lately prevailed among physiologists, that dyspnoea generally is more a cardiac than a pulmonary condition.

An early effect of cardiac dyspnoea is that the patient is unable to sleep with the head low. Instead of one pillow he has to use two or three. In extreme cases he cannot lie down at all, and is obliged to sit up in bed or to lean forwards. This condition is termed *orthopnoea* (vol. i, p. 1042). The upright position facilitates the descent of the diaphragm, which in the recumbent posture is hampered by the pressure of the abdominal viscera, and especially of the liver. It also allows of free movements of the ribs unimpeded by the weight of the body.

We are familiar with the fact that dilatation of the left ventricle leads to a secondary dilatation of the right, owing to the pulmonary obstruction produced, and it is difficult to understand how the order of events can be reversed. Pathologists, however, are agreed that if the most frequent cases of enlargement of the right side of the heart (from pulmonary emphysema or other obstruction in the lungs) be excluded, dilatation of the right side is almost always accompanied by a like condition of the left. The following case is a rare example of primary hypertrophy with dilatation of the right ventricle, without primary or subsequent affection of the left.

A man, aged forty-one, was admitted into Guy's Hospital in 1880 on account of dropsy of the abdomen and legs. On examination there was a loud systolic murmur at the ensiform cartilage, musical in quality at that spot, and propagated towards the right nipple as much as towards the left. On account of the rarity of primary disease of the right chambers this diagnosis was given doubtfully, but the autopsy left no question of the fact. The heart, which weighed $16\frac{1}{2}$ oz., was extremely broad and rounded in shape; the right

ventricle was large and massive, with large fleshy columns, but the left was small and flaccid; the right auricle formed the greater part of the base of the organ; the tricuspid orifice admitted more than five fingers, and its edge was thick and opaque.

It had been a striking feature of this case that there was no orthopnoea; the patient, though very dropsical, lay quite low in his bed. At the time the easy state of the breathing was regarded as a further argument against the view that a cardiac affection of whatever kind was the cause of the ascites and of the anasarca; for it has hitherto been the universal opinion that dyspnoea must necessarily be produced by any lesion of the right side of the heart interfering with the blood-supply to the pulmonary capillaries. But it admits of doubt whether this opinion is well founded, and the point is one well worthy of consideration in future cases.—C. H. F.

Some writers describe the pulse, in cases of cardiac hypertrophy, as full, tense, and resisting; and the patient as florid in face, and liable to rushing of blood to the head, and to throbbing cephalalgia. But although these statements apply to cases of cardiac hypertrophy with high arterial tension, as in chronic Bright's disease, they do not apply to those in which the hypertrophy of the left ventricle is primary. Careful observations all point to the conclusion that whether hypertrophy or dilatation be the result of overstrain of the heart, the effect on the pulse is to weaken its force and lower its tension.

Prognosis and treatment.—The slighter degrees of enlargement of the heart subside when the cause is removed; but when hypertrophy has developed fully, it is only as the result of long and patient treatment that a cure can be looked for. Physiological rest—either confinement to bed, or in less severe cases lying on the sofa during the day—is essential.

Among drugs, *aconite* is most useful. Da Costa testifies decidedly to its value when the cardiac impulse is unduly forcible; he generally gave one or two minims of a tincture three times as strong as the British tincture, three times a day; or gr. $\frac{1}{60}$ to $\frac{1}{30}$ of *aconitia*. Walshe speaks highly of the same remedy; the dose which he recommends is gr. $\frac{1}{8}$ of the alcoholic extract of *aconite*. In some cases *veratrum viride* may be employed with advantage; Da Costa administered drop doses of the fluid extract, or five to ten minims of the tincture, three times daily. *Bromide of potassium* is another useful remedy; but the iodide does not seem to be of any service. The writer has seen apparent benefit from the exhibition of *belladonna*. Giving up smoking has often succeeded in curing the cardiac affection, even when of long standing; but he has not seen such good results from giving up alcohol.

If the early symptoms of cardiac hypertrophy are neglected and no adequate rest is taken, the probable result is that sooner or later the hypertrophy gives place to dilatation. In such circumstances *digitalis* is often of the greatest possible service.

Treatment of cardiac hypertrophy and dilatation by gymnastic movements of the arms, as well as by walking, usually combined with the use of warm baths, plain or saline, has of late years been much before the public as well as the profession. The effects described are probably exaggerated in degree and dependent on more than one cause; but in many cases they appear to be good.

If unchecked, primary cardiac hypertrophy and dilatation pass at length into a condition of "asystole," leading to a series of changes in the lungs and in the liver, and to dropsy of the dependent parts of the body, precisely like the effects of the same conditions when the result of disease of the valves.

FIBROUS DISEASE OF THE HEART.—In many cases of cardiac overgrowth we find that the thick ventricular walls are not all composed of muscular tissue, but that there is more or less addition of fibrous tissue also; so far, therefore, such enlargement is not purely hypertrophic, and cannot be reckoned on to compensate obstructions in the circulation. As a rule, we find this fibrous (or, as it is oddly called, "fibroid") degeneration in patches, but occasionally it appears as a diffuse change.

The late Sir Richard Quain described in his Lumleian Lectures ('Lancet,' for 1872) what he termed "connective-tissue hypertrophy," in which the muscular fibres throughout the organ are surrounded by connective tissue in all stages of development—round cells, spindle cells, and bundles of fibres. Dr Charlewood Turner, in a paper read before the International Medical Congress for 1881 ('Trans.,' vol. i, p. 427), described, with microscopical drawings, the histology of this condition as one of chronic interstitial myocarditis. He observed not infrequently coincident deposition of calcareous salts; but fatty degeneration was rare as a complication. The thickness of the heart's wall is in such cases increased, but the most striking peculiarity is its firm, tough, leathery feel. As a proof that this change may be overlooked, Quain refers to a heart, weighing more than forty ounces, which had been for thirty years preserved as a specimen of cardiac hypertrophy in the museum of St George's Hospital. On careful examination the increased size was found to depend only in part upon muscular overgrowth, and in part upon excess of the connective tissue.

This diffused hypertrophic sclerosis is very rare as a primary lesion, although it frequently accompanies the consecutive muscular hypertrophy of the left ventricle, due to Bright's disease or to valvular lesions. But no fewer than eleven cases of circumscribed fibrous degeneration came under observation in Guy's Hospital in one period of less than twelve months (1873-4). Altogether twenty-seven cases were recorded by Dr Fagge, who described it as follows. In its slighter degrees it consists in the presence of streaks and patches of a milky white colour in the substance of the muscular tissue. The wall of the heart is thereby rendered more hard and resisting to the knife, but it may also acquire a succulent and spongy appearance, and when incised its cut surface looks uneven. Microscopically there is seen a perfectly developed connective tissue, forming wavy bands, running in the same direction as the muscular fibres, some of which, or the remains of them, are still embedded in it. Or there may be a dense fibrous plate, looking like a piece of tendon, and consisting of a glassy substance with regularly arranged fissures or spaces, indicating the planes of fibrillation. It creaks when cut, and sometimes it contains calcareous salts in such quantity as to make it crackle under pressure, like an egg-shell.

The seat of this lesion is sometimes the apex of the heart, sometimes a part of the anterior or posterior wall of the left ventricle, sometimes the septum. It seldom or never begins in the right ventricle, but it may invade it by extension from the left. The fibrous patch is occasionally surrounded on all sides by muscular tissue, so that it touches neither the endocardium nor the pericardium; and it is then very likely to be overlooked, unless all parts of the organ are completely sliced up. In other cases it reaches one or both surfaces; and the endocardium then shows a marked local opacity, while the visceral pericardium is found thickened or adherent to the parietal layer.

Some pathologists have thought that fibrous disease of the heart is

generally secondary to pericarditis or to endocarditis, and that one or the other spreads to the myocardium. But adhesion is often present only where the fibrous patch reaches the outer surface of the heart; and even when the whole serous sac is obliterated, the two layers are often found to be firmly united at that spot, but elsewhere so loosely that it is easy to separate them. Moreover, if extension from a general pericarditis occurred, one would expect to find connective tissue dipping into the muscle at many different points, which is not the case. Lastly, in one of Dr Fagge's cases, pericarditis was recognised by physical signs about two months before death, when the fibrous degeneration must have been of long standing.

When the part of the left ventricle that is affected by fibrous disease includes the base of either of the fleshy columns of the mitral valve, the process often spreads into that column, which becomes shrunken. This condition is distinct from the far commoner one, in which the summits of the mitral columns undergo conversion into fibrous tissue by extension from the tendinous cords at their apex.

In all cases of fibrous degeneration there is considerable atrophy of muscular tissue at the seat of the lesion. Not infrequently the whole substance of the wall is involved from one surface to the other, with not a trace of the normal structure between. The thickness of the wall is, as a rule, much diminished, but occasionally there is obvious increase of bulk.

Pathology.—The origin of this fibrous degeneration is perhaps not the same in all cases. In some it is no doubt irritative, a true *myocarditis*. The first stage is an infiltration of leucocytes; these develop into connective tissue, and the atrophy and disappearance of the muscular fibres at the seat of the disease is a secondary effect of the compression of the muscle itself, and also of its nutrient arteries, by the contraction of the new-formed tissue, as in cases of cirrhosis of the liver or kidneys.

In four of twenty-seven cases taken from the records of autopsies at Guy's Hospital there was history of a former attack of acute rheumatism.

In other cases *syphilis* gives rise to fibrous patches, in a later stage probably not distinguishable from those due to other causes, but when seen earlier identified by the presence of gummata, as in eight cases cited by Lancereaux. The development of gummata in the heart is by no means limited to the left ventricle; in two cases the right ventricle was alone affected, and in one the right auricle. Among Dr Fagge's twenty-seven cases of fibrous disease of the heart there were four in which, from the presence of specific lesions elsewhere, the existence of syphilis could be safely asserted, although in only a single instance were gummata detected; one as large as a bean in the right ventricle, and small, hard, yellow nodules embedded in a reddish gelatinous substance at the growing edge of a fibrous patch in the left ventricle. In one instance the septum was changed through nearly its whole thickness into a tough fibrous material, with puckering and depression of the endocardium. The disappearance of gummata in the most advanced stage of the disease corresponds with what is observed in the liver, the testis, and other organs.

The first case of this remarkable condition was brought before the Pathological Society by Wilks, in 1856, and was the subject of a paper of historical value in the 'Guy's Hosp. Reports' for 1863 (vol. ix, 3rd series). Many similar observations have since been recorded at home and abroad; among later authors, by Dr Loomis in the 'American Journal of Medical Science' for October, 1895, by Dr Theodore Fisher in the 'Bristol Med.-

'Chir. Journal' for December in the same year, and by Dr S. Phillips in the 'Lancet' for January 23rd, 1897, where he records several cases of syphilitic fibrosis of the heart, some due to gummata and some to endarteritis of the coronary vessels.

Adherent *thrombi in the cardiac cavities* may give rise to an inflammatory change, which spreads through the wall of the part to which they adhere. A well-marked instance of this once occurred in the auricular appendix. It is not improbable that the result might be the formation of a patch of fibrous degeneration; and such an origin might explain its frequency at the apex of the left ventricle, for this point is very apt to become the seat of local coagulation during the course of enteric and other fevers.

But by far the most frequent cause of this degeneration of the heart is *obstruction* of a branch of the coronary arteries by thrombi, emboli, or atheroma. In a series of eleven cases of fibrous disease of the heart brought by Dr Fagge before the Pathological Society in 1874, there was one in which the cardiac muscle was converted into a greenish-brown substance, of the texture of wash-leather. Under the microscope it was found to be merely muscular tissue, which retained its striation, and showed remarkably well the branching and reuniting of the fibres. Its characters contrasted strongly with those of the muscular fibres which lay within the area of the fibrous change; they exhibited but slight striation, or had undergone fatty degeneration. In the case of another patient, aged sixty-two, with regard to whom the probable existence of fibrous disease of the heart was repeatedly discussed at the bedside, it was found after death that the posterior wall of the left ventricle was much thinned, and was to a large extent converted into a lustreless, yellowish-green substance, almost exactly like that just described; no fibrous change, however, had taken place. From these two cases it appeared clear that the peculiar change in the muscular tissue must be the primary affection, and that the fibrous degeneration must be secondary.*

Weigert showed ('Virchow's Archiv,' vol. lxxix, 1880) that fibrous degeneration of the heart is often the result of blocking of a blood-vessel (*infarctus*). This was confirmed by the observations of Hüter (*ibid.*, vol. lxxxii, 1882), who found precisely the same dry greenish or yellowish-brown patches as those above described, and traced them to obstruction of branches of the coronary arteries, sometimes thrombosis, sometimes embolism. He recorded eighteen cases of fibrous disease of the heart, in which the affection was associated with sclerotic changes in the coronary arteries corresponding more or less closely in distribution with that of the fibroid patches. Dr Fagge had drawn attention in the above quoted paper ('Path. Trans.,' 1874) to the relation between fibrous disease of the heart and *arteritis deformans* of the systemic arteries generally. In the second of his two cases above mentioned, the coronary arteries were extremely diseased, and some of their branches were completely obliterated.†

It might have been expected that the loss of so much contractile power as must result from the transformation of muscular into fibrous tissue would seriously weaken the heart. But Cohnheim found experimentally that in the rabbit a large part of either ventricle, or even the entire lower

* Dr Ormerod, ten years before, had suggested this relation ('Brit. Med. Journ.,' 1863).

† It seems impossible to reconcile Hyrtl's and Cohnheim's statement that the coronary arteries and their branches possess no anastomoses, with the results of the injections made by Legg and by Samuel West, who found that they could readily fill one artery from the other, the two communicating over the surface and at the apex of the heart.

third of the heart, may be held fast in a clamp without the arterial pressure becoming lower in consequence. In harmony with this fact, clinical experience shows that cardiac fibrosis may give rise to no symptoms whatever. In three of the above-quoted twenty-seven cases, in which it was discovered at the autopsy, the patient had died from some other cause. On the other hand, the frequency with which dilatation and hypertrophy are associated with fibrous degeneration proves that in many patients the systole of the ventricle is thereby more or less interfered with; in ten of our twenty-seven cases the heart was considerably enlarged, weighing from twenty-one to thirty-five ounces.

A peculiar alteration in the shape of the ventricular cavity is produced by the excess of fibrous tissue in its wall; it becomes deepened from before backwards, so that the mitral valve lies much further from the anterior surface of the heart than usual. Occasionally the valve is also separated from the posterior surface by a considerable interval.

Hyaline changes in the cardiac muscle and cloudy swelling have been studied by Virchow, Leyden, and other pathologists, and are probably due to a diffuse parenchymatous myocarditis. The condition is the histological counterpart of the acute dilatation described above as supervening on febrile states, as diphtheria, influenza, enterica, scarlatina, and rheumatism.

Cardiac aneurysm.—During the systole any portion of the wall of the heart that has undergone local degeneration must be exposed to great pressure; one can imagine it forming a protrusion like those that are observed in the frog's heart when the action of digitalis is beginning to manifest itself.* Indeed, when the whole thickness of the cardiac muscle is destroyed, a permanent yielding of the affected part almost always results. Sometimes it forms a shallow pouch, sometimes a sac of greater or less size, communicating with the ventricle by a comparatively narrow opening. This condition is now generally known as "cardiac aneurysm."† It depends almost always upon a previous fibrous change in the muscle. Dr Legg, in his Bradshawe Lecture (1883), cited three cases which appear to show that it may be produced by fatty degeneration; but there is no doubt that these cases are quite exceptional; and the reason doubtless is, that in fatty hearts the degeneration is too widely diffused, and the ventricular systole too feeble, for great pressure to be thrown upon any one part of the chamber.

Aneurysm of the heart, like the fibrous degeneration which leads to it, is met with at all periods of adult life up to an advanced age. Among Hüter's eighteen cases of fibrous disease (only four of which occurred in patients under sixty) there were four in which aneurysms were present; and the ages were fifty-six, sixty-two, seventy-three, and eighty. Both fibrous degeneration and cardiac aneurysm are far more common in men than in women.

Aneurysms of the heart seldom attain a great size, but may occasionally become as large as the fist. They then, of course, project from the surface of the heart; but the smaller ones, especially if there are several of them, are sometimes excavated within its substance. In a remarkable instance ('Path. Trans.' 1874) Dr Fagge found the wall of the left ventricle tunnelled out in all directions by aneurysms, of which the largest was the size of a walnut. Fibrin is often deposited in the sac of a cardiac aneurysm;

* See a paper by Fagge and Stevenson, in the 'Proc. Roy. Soc.' for 1866.

† The same term was used by Corvisart and earlier pathologists to denote general dilatation of the cavities of the heart, and it has also been applied to destruction by septic ulceration of part of the wall of the heart in connection with a like affection of the valves.

in the specimen just referred to, many of the cavities were filled with an adherent greenish, gelatinous substance, with curd-like degenerating flakes. Wilks observed (*ibid.*, vol. iii) a case in which there was found attached to the apex of the heart a cured aneurysm the size of a pigeon's egg, of which the walls were calcareous and the interior completely consolidated ('*Path. Trans.*,' 1857).*

Symptoms.—Neither fibrous disease nor aneurysm of the heart can be certainly detected during life. The presence of the former may perhaps be suspected when cardiac symptoms, without evidence of valvular lesions, are present in a patient showing signs of arterial disease. A syphilitic history, too, may lead one to infer that the heart has been the seat of gummata, out of which fibrous tissue has developed; and in such cases good results may be hoped from a prolonged course of mercury and iodide of potassium. One striking instance of this came under Dr Fagge's notice, and Dr Balfour has known cases of "excited action of the heart with hypertrophy," which yielded to antisyphilitic treatment, and were perhaps due to diffuse fibrosis.

The physical signs of fibrous degeneration with hypertrophy are indistinguishable from those of enlargement due to overgrowth of the muscle. In either case a systolic apex-murmur may or may not be audible; at one time it may be present and not at another. In a few instances the pulse has been unusually slow, varying from 28 to 48 in the minute.

When there is a large aneurysm projecting from the heart's apex, a careful mapping out of the area of cardiac dulness might possibly point to the real nature of the case; but the disease would more likely be regarded as an aortic aneurysm pushing downwards into the cardiac region. In one instance Skoda is said to have observed bulging of an intercostal space overlying the seat of a cardiac aneurysm.

The symptoms of fibrous disease of the heart are those of cardiac incompetence from whatever cause:—dyspnœa on exertion, or following flatulent dyspepsia, or coming on at night in the form of asthmatic paroxysms; then more or less marked cyanosis and coldness of the extremities; and, lastly, congestion of the systemic capillaries with dropsy. The period that elapses from the beginning of the patient's illness up to the time of his death is occasionally short,—in one of Dr Fagge's twenty-seven cases only seven weeks. Other cases run a protracted course, and are benefited by treatment with arsenic or strychnia.

In many cases, however, the heart goes on steadily, and apparently discharging its functions as in health, until the patient suddenly falls down dead.

For example, Dr Whipham ('*Path. Trans.*,' xxi) has recorded a case of fibroid disease of the heart in a man, aged twenty-nine, who fell dead from his horse while riding in Hyde Park, having started in good spirits and apparently perfectly well, and having never before exhibited any symptoms of cardiac disease. The heart was of normal size and weight, and the valves perfect. The fibrous growth was very extensive, in discrete patches connected with the endocardium. There was no sign of syphilis.

Such abrupt and fatal syncope is difficult to explain. But there is an exact parallel for it in the results of the experimental ligature of one coronary artery, or even of a large branch of one coronary artery, in the dog, as practised by Cohnheim ('*Virch. Arch.*,' vol. lxxxv). After this operation the heart for a little while goes on beating with perfect regularity,

* I well remember the autopsy, for it was the first at which I was present when I entered as a student of Guy's Hospital in October, 1856.—C. H. F.

and maintains the arterial pressure at its normal level. But at the end of about ninety seconds its pulsations become somewhat less frequent, and their rhythm is slightly disturbed; and about half a minute later both ventricles suddenly stop at the same instant, after which no stimulus whatever succeeds in restoring their contractions.

ACUTE MYOCARDITIS.—Acute or subacute inflammation of the muscular tissue of the heart may take place as an independent change under special conditions, particularly as a result of acute fevers, as above noted (vol. i, pp. 41, 137); but it more often forms part of a rheumatic endocarditis or pericarditis. In the latter case it is not at all unusual to see the layer of muscle immediately beneath the serous membrane involved; it is pale and soft, and the microscope shows it to have undergone a change in texture of a yellow fatty kind. In some exceptional cases the whole thickness of the walls is seen to be affected; then dilatation is apt to occur, and may lead to speedy death or become a permanent morbid condition. Sometimes, long subsequent to the original attack, its effect may be seen in a fibrous patch, showing on section white fibrous streaks between the muscular fasciculi.

A more acute inflammation of the muscle, or rather one of a different kind, is met with in pyæmia, when numerous small abscesses may be found scattered through the cardiac tissue. A single circumscribed abscess is sometimes met with in connection with septic endocarditis. Wilks long ago observed that secondary abscesses of the heart are apt to be found along with pyæmic deposits in the kidneys, when the primary source of pyogenic micrococci is in one of the bones.

FATTY DISEASE OF THE HEART.—(1) *Adipose overgrowth and infiltration.*—The layer of adipose connective-tissue beneath the pericardium is often found increased in elderly persons, and in others disposed to make fat, and within limits this is a physiological condition which gives rise to no symptoms during life. It chiefly affects the right side of the heart, especially at the base of the ventricle.

Sometimes, however, the fat grows in upon the muscular fibres so as to thin the cardiac wall, and this becomes a cause of atrophy of one or both ventricles. Occasionally the adipose growth may penetrate right through the wall until it meets the endocardium, and this is most often seen near the apex of the right or the left ventricle.

Moreover the adipose tissue increases between the fibres. A large amount of interstitial fat in the muscles of the limbs, as in cattle fatted for the market, and in men who drink largely of beer, is probably indicative of over-feeding and under-work. When the change affects the diaphragm it may become serious; but this form of fatty heart was at one time regarded by pathologists as of little importance, because it did not affect the muscular fibres themselves. It certainly, however, encroaches upon them, and diminishes the active force of the circulation. In beer drinkers this condition goes with fatty liver, fatty deposit under the pleura and peritoneum, and fatty overgrowth in the skeletal muscles. Such persons are notoriously ill-fitted to withstand the effects of severe injury or of operations; and in some cases of sudden death, the cause appears to be a soft and flabby state of the heart, with adipose hypertrophy.

(2) *Fatty degeneration.*—This is a different pathological condition,

and was recognised as such by Laennec. The heart is free from any excess of adipose tissue, or may have less than ordinary; but its tissue is pale, soft, and easily torn. On the inner surface, particularly of the musculi papillares of the left ventricle, pale, yellow, zigzag markings are seen, described in the 'Med.-Chir. Trans.' for 1850 (vol. xxxiii) by Quain as "tabby degeneration." Under the microscope the fibres are found to have lost their striæ, and black granules appear instead, at first in transverse lines, as if the change had affected disc after disc. Next the dark granules become larger and acquire a bright glistening centre; at last the striæ become obscured, and all trace of structure disappears.

It has been stated* that the amount of ethereal extractives from such hearts is not greater than normal; if so the frequent wasting of the normal adipose tissue and the usually small extent of the degenerative process, often limited to the left ventricle, may explain the result. Stevenson, however, found the fatty matter nearly doubled; Böttcher and Valentiner support the same conclusion; and Krylow has apparently settled that there is a decided relative increase, though much less than might have been anticipated.

The most frequent causes of this fatty degeneration of the heart (which is quite independent of obesity either of the heart or other parts) are, first, anæmia; and secondly, certain mineral poisons. It is most constant and well marked in those remarkable cases of idiopathic anæmia first described by Addison, and since known under the names of "Anémie grave" and "perniciöse Anämie." It is also often found in cases of leuchæmia and Hodgkin's disease, and occasionally in phthisis, cancer, and other wasting and anæmic disorders.

It is a constant appearance in fatal cases of poisoning by phosphorus, when the liver is also the seat of remarkable fatty degeneration; and the same thing has been observed in poisoning by arsenic, and occasionally as the result of mercury or of lead. It has been sometimes, but not constantly, observed when the coronary arteries have been much diseased, particularly when their calibre is narrowed by atheroma. Hence this form of degeneration has to some extent a common origin with fibrous disease of the cardiac tissue. But it does not appear to be caused by embolism or thrombosis; nor is it a frequent or important complication of cardiac hypertrophy and dilatation, whether due to valvular, renal, or pulmonary lesions.

Lastly, acute fatty degeneration is often seen as a superficial change immediately beneath an inflamed pericardium.

The *symptoms* of either form of fatty disease of the heart are very obscure and doubtful. It is usually surmised from our knowledge of pathology rather than diagnosed by physical signs.

When a patient is pale, and the blood is decidedly deficient in corpuscles or in hæmoglobin, or in both; when the cardiac impulse is weak but irritable, or sometimes "slapping," *i. e.* strong but short; when the first sound is accentuated and has lost its proper character, so as to resemble the second, we may surmise that the heart is in fatty degeneration. The radial pulse may be quite unaffected; and general symptoms of lividity, dyspnœa, and anasarca probably only appear when there is concomitant dilatation.

Adipose overgrowth may be suspected when a person past fifty, florid

* Hermann Weber, "Zur Lehre von der fettigen Entartung des Herzens," 'Virchow's Archiv,' xii, 326 (1857). Sir H. Weber has informed the writer that subsequent extension of his inquiries confirmed his previous conclusion.

and over-stout, with a white, soft, "satiny" skin and early arcus senilis, suffers from dyspnoea, and has a weak cardiac impulse.

The *result* of fatty degeneration is undoubtedly in certain cases sudden and fatal syncope. Often, however, it is found after death has occurred in other ways. It does not appear in itself to lead to dilatation, to which it is rather secondary; but it certainly may end in rupture of the heart.

Experience in the deadhouse does not bear out the assertion that fatty degeneration is a frequent complication of valvular disease. In fact, this lesion usually comes unexpectedly before the pathologist, though it is often diagnosed at the bedside, or in the returns to the Registrar-General.

On the whole, ingrowth of adipose tissue is probably a more frequent and perhaps a more dangerous lesion than interstitial fatty degeneration.

A granular or brown degeneration of the cardiac muscles has been frequently observed, but its relations to fatty degeneration and its pathological significance are still obscure. In its most marked condition, it is identical with what has been described as pigmentary degeneration, present, according to Wilks and Moxon, in most persons dying at an advanced age, and also as the result of some chronic wasting maladies. The muscle is dark to the naked eye, and under the microscope each fibre is filled with reddish yellow granules, arranged round the nucleus of the muscle-cell. They appear to be derivatives of hæmoglobin. This form of degeneration generally occurs along with atrophy of the heart, and does not appear to lead to any symptoms during life.

Spongy or cavernous degeneration has been observed in the foetal myocardium by Virchow.

The rarest form of cardiac degeneration is that which has been described by Köster as calcareous infiltration. A good account of it, with two original specimens figured, was given by the late Dr Coats in his 'Manual of Pathology.' It sometimes complicates cases of fibrous sclerosis and cardiac aneurysm.

RUPTURE OF THE HEART.—This rare and interesting pathological condition owes its practical interest to the importance of discriminating it as a primary condition from rupture as the result of injury. In the latter case the lesion is almost always in the right ventricle or in one of the auricular appendages; while idiopathic rupture almost always occurs in the left ventricle. Of fifty-five cases from the records of Guy's Hospital, forty-three affected the left ventricle and only seven the right; while the auricles still more rarely rupture spontaneously. Usually the muscle is already weakened by fibrous, fatty, or granular degeneration; but several instances are on record in which no such changes have been detected.

Morgagni (Epist. xxvii, 10) had already remarked that rupture of the left ventricle is more common than that of the right; and that this latter is more frequent than rupture of the auricles.

Auguste Ollivier states that, out of 49 instances, the rupture was seated in the left ventricle in 34, in the right ventricle in 8, in the left auricle in 2, and in the right auricle in 3, and that in two cases both ventricles presented several ruptures. The results were very different in respect of ruptures occasioned by external violence. In 11 instances of this description, the right cavities were torn in 8 and the left in 3. In these 11 cases the auricles were torn in 6.

The age of the patient is generally over sixty. George the Second died of rupture of the heart at the age of seventy-six: it is remarkable that he was the first English sovereign who exceeded the age of seventy.

Panum, the eminent Danish physiologist, died of rupture of the heart in 1885, at the age of sixty-five.

The rent does not always go through the entire thickness of the wall, and may possibly in such cases be recovered from; but usually hæmorrhage into the pericardium ensues from the torn vessels, and causes death as certainly, though not so rapidly, as when a larger rent opens directly into the ventricular cavity.

PERICARDITIS

Ὅταν τὸ ὕδωρ πνίγγῃ, τὶ δεῖ ἐπιπίνειν.

Proverb quoted by ARISTOTLE ('Eth. Nic.,' lib. vii, cap. 2).*

Rarity of idiopathic pericarditis—its usual antecedents—Anatomy—fibrinous, serous, hæmorrhagic, and purulent effusion—Physical signs—Symptoms—Diagnosis—Prognosis and events—Adherent pericardium: its effects: its recognition—Hypertrophic pericarditis and pulsus paradoxus—Milkspots on the heart—Hydropericardium—Hæmopericardium—Treatment of pericarditis and of pericardial effusion—Paracentesis.

INFLAMMATION of the pericardium has been recognised as an anatomical condition ever since autopsies were systematically made; and attempts were made even before the discovery of auscultation to diagnose it during life—for the most part unsuccessfully. Laennec recognised the characteristic pericardial rub and its likeness to that of pleurisy. Inflammation of the pericardium is much less closely connected with disease of the viscus it covers than inflammation of the pleura, pia mater, or peritoneum; but otherwise it closely resembles them in its pathology.

Ætiology.—Pericarditis is very rarely idiopathic, *i. e.* it is not set up as a primary disorder by exposure to cold, like pleurisy; indeed, the pericardium is too deeply situated to run such a risk. Moreover, the heart is not, like the lung, liable to acute inflammation, which might spread to the membrane covering it. If we exclude the rare cases in which pericarditis is set up by direct mechanical injury, it can, in most cases, be traced to a general malady, or to local disease of a neighbouring part. The great majority of cases of pericarditis are secondary to either rheumatism or Bright's disease.

Professor Bäumlér, now of Freiburg, published three cases of idiopathic pericarditis in adults, in the fifth volume of the Clinical Society's 'Reports,' and the writer once saw an uncomplicated idiopathic case with Dr Dalton, of Norwood, in a healthy man about forty. Acute pericarditis is said to have followed prolonged bodily exertion—for example, a long march after exposure to cold; but surely the sequence was only accidental.

It is often secondary to pleuro-pneumonia, and scarcely any acute disease is more rapidly fatal than double pleuro-pneumonia with pericarditis; but in these cases the pulmonary symptoms override those of the pericardial inflammation. Both organs may be simultaneously attacked, *i. e.* the

* A proverb: When water chokes a man what can he drink?

pneumococci affect lung and pleura and pericardium either successively or at once.

In patients under forty years of age rheumatism is by far the most frequent cause of pericarditis, and in children under puberty almost the only one. After middle life it is rarely due to this cause, but the writer once saw an old gentleman of seventy-three who died of rheumatic pericarditis. It occurs as a rule a day or two after the pyrexia and synovitis have appeared; and its advent is almost a guarantee of the accuracy of the primary diagnosis, for pericarditis does not occur in cases of gout, of osteoarthritis, or of gonorrhœal synovitis, except that severe form of the last, in which there is suppuration of one or more joints—in which, in fact, the pericarditis is pyæmic and due to a streptococcus, not to a gonococcus.

Pericarditis is sometimes the first manifestation of acute rheumatism, pain and swelling of the joints coming on only when it has existed for two or three days. Hence, when a patient dies of pericarditis after a very short illness, one cannot exclude a rheumatic origin—especially in the case of children, since in them rheumatic pericarditis preceding the synovitis is much more common than in adults.

Next to acute rheumatism, Bright's disease is the most common cause of pericarditis. It may occur with the more acute tubal form, or with chronic interstitial nephritis; and in persons above fifty the latter is by far the most frequent cause of pericarditis.

Tuberculous pericarditis is not uncommon in the deadhouse, particularly in children, but is not often observed at the bedside, and occurs chiefly when several serous membranes together are attacked by tubercle.* Formerly pyæmia was said frequently to give rise to this and to other serous inflammations, but it is now believed that pericarditis occurs in pyæmic cases only when suppuration has first attacked the myocardium.

In Russia pericarditis has been often observed in the course of scurvy. Gout has also been mentioned as a cause, but this has probably only acted indirectly, through the renal disease which so often complicates it. Pericardial effusion with some inflammatory lymph in addition to the serum of dropsy is often discovered after death in cases of cardiac dropsy, but usually when the kidneys as well as the heart are diseased.

Mediastinal suppuration, caries of the ribs, and a mammary abscess are other occasional causes of pericarditis; also malignant growths of the mediastinum, or secondary deposits from cancer of the breast, liver, or stomach. Lastly, syphilitic gummata may cause pericarditis, as in a case published by the writer ('Path. Trans.,' 1869, p. 94).

Anatomy.—The characters of pericarditis are those of inflammation affecting a serous sac with an endothelial surface. At an early stage the membrane loses its lustre. Then coagulable lymph appears on its surface, often first around the roots of the great vessels, and gradually forms thick concentric layers of fibrin. The parietal pericardium likewise becomes lined with a similar false membrane, and between the two surfaces there is more or less serous or sero-purulent liquid. In consequence of the incessant movements of the heart the surface now becomes remarkably roughened. Sometimes it bristles with villous processes, like the paunch of a

* Dr Musser, of Philadelphia, has recorded a remarkable case of acute tuberculous pericarditis, with effusion of sixty-four fluid ounces of bloody serum ('Trans. Coll. Med. Phil.,' November, 1888).

ruminant, or becomes honeycombed, like a sheep's stomach. Or, to use Laennec's comparison, the surface looks as if butter had been squeezed between two flat pieces of wood, which then were suddenly separated. So shaggy does the heart sometimes look in these cases that it was formerly known as the *cor hirsutum*. The progress made by pathology between 1750 and 1850 is shown by the fact that Haller described this "hairy" heart as occurring especially in bold and adventurous men. The thickness of the false membranes in acute pericarditis is greater than is at all frequent in peritonitis or even in pleurisy.

In other cases pericarditis leads to the effusion of serum with only flakes of fibrin. When the disease is acute, the amount of the exudation cannot much exceed the twelve to eighteen ounces that it is possible to inject into the sac after death; when this limit is reached the diastole of the heart is interfered with and great distress is produced, terminating in the rapid death of the patient. But in chronic cases a much larger quantity of fluid may accumulate in the pericardium; more than three pints have been found.

The serous effusion is sometimes tinged with blood, particularly in cancerous and, it is said, in scorbutic cases.

Suppuration is much less frequent than in other serous sacs, but this probably depends chiefly on the preponderance of rheumatism as a cause, for rheumatic inflammation, whether of the joints, the pleura, or the pericardium, is seldom or never purulent. Purulent or sero-purulent effusion is not uncommon in cases of Bright's disease, and is constant in those of septic pericarditis.

Empyema following pneumonia is occasionally complicated by suppurative pericarditis; and much more rarely purulent pericarditis follows Scarlatina, Enteric fever, or Influenza.

In very rare cases the fluid in pericardial effusion has been known to undergo decomposition, with the evolution of foetid gas. This condition, *hydro-pneumopericardium*, has also been observed when suppuration or malignant disease has perforated the pericardium from the mucous surface of the oesophagus or stomach.

Inflammation often spreads from the pericardium to adjacent parts. Thus pleurisy may be set up, especially on the left side. In other cases the mediastinal tissues become affected, so that the parietal pericardium is fixed to the sternum by dense adhesions (cf. p. 151). The areolar tissue above the heart may participate in this change, and the left innominate vein may be plugged with coagulum—a condition which leads to œdema of the left arm without general dropsy.

Another and a very important extension of pericarditis is to the myocardium. The outermost layers of muscle are soft, and of a pale yellow or dull greyish-red colour. On microscopic examination it is found that superficial fatty degeneration of the fibres has taken place.

Signs.—The recognition of pericarditis at the bedside turns upon the discovery of its characteristic physical signs, for the general symptoms, taken by themselves, are uncertain guides. The earliest sign is generally the friction-sound or *rub*. It is true that in some cases, even before this can be heard, the onset of pericarditis may be suspected from the heart's action becoming disturbed and "tumbling," and the first sound being prolonged; but only the detection of a rub converts this suspicion into a cer-

tainty. The murmur always accompanies the first cardiac sound, or both; it is never only diastolic. A double, "to-and-fro sound" is very characteristic, and scarcely less so a continuous churning noise going on without any complete intermission. Its quality varies like that of a pleuritic rub, from a harsh, dry, tearing sound, to a soft murmur like a râle, and in certain cases it is decidedly musical.

The rhythm of the heart is often changed to a very characteristic three-beat or cantering cadence—the *bruit de galop* of Laennec. The three sounds heard appear to be the first sound, the systolic rub, and the diastolic rub. The seat of a pericardial rub is usually the base of the heart, and it is never heard beyond the cardiac limits.

It sometimes lasts for weeks; in other cases it rapidly disappears, after being audible for a few days, or only for hours. Sometimes this disappearance of the sound is not due to absorption of the effused lymph and recovery, but to the two serous surfaces becoming separated by fluid, so as no longer to rub together as the heart moves. In this case the rub may again be heard after the serum has been absorbed.

Occasionally a pericardial, like a pleuritic rub, can be felt as well as heard, and the thrill is often very distinct.

The presence of fluid in the pericardium is indicated by special signs, the most important of which is an increase in the area of the cardiac dullness; and in practice it is generally found that the augmented dullness is first discoverable at the base of the heart. Instead of the percussion-note in the left third interspace being but little less resonant than at the corresponding point of the opposite side, it becomes completely dull, and this dullness often reaches as high as the second rib, and sometimes even higher. The area of pericardial dullness, when there is considerable effusion, is extended in all directions; and the triangular space of normal cardiac dullness is not only enlarged, but altered in shape by the upper angle being broadened, so as to make it almost quadrangular.

When the quantity of fluid is considerable, the left lung is pushed to one side and partly compressed; indeed, dullness on percussion may exist over so large a part of the left side as to cause the case to be mistaken for one of pleuritic effusion. This was the case with a patient of the writer's, a woman, in whom the gradual but at last enormous distension of the pericardial sac was ascribed to a much smaller concomitant pleural effusion on the left side.

Another sign of pericardial effusion, if extensive, is bulging of the præcordial region; and occasionally the diaphragm may become so depressed that the epigastrium is pushed forwards, and the left lobe of the liver downwards.

The apex-beat is first raised, the heart being drawn up towards the great vessels as the pericardium fills. The impulse becomes gradually fainter until it disappears, as the serum effused gradually separates the apex from the chest-wall. The base of the heart is much less moveable, and hence a friction-sound over the right auricle may be heard after considerable effusion has taken place. Indeed, Dr George Balfour holds that a rub once heard in that position is not lost by the largest effusion.

Dr Ewart has found that the right clavicle is raised by a large pericardial effusion, so that the upper border of the first rib can be felt up to the sterno-clavicular joint ('Brit. Med. Journ.' 1896). He also lays stress on a sign of extensive pericardial effusion which has been recognised by several

independent observers (Dr Sansom, Dr Frederick Roberts, and Dr Allbutt among others), namely, a small patch of dulness, usually situated at the angle of the left scapula or at the left base close to the spine. This is probably produced by a portion of lung being carnified by pressure of the distended pericardium, and it is sometimes accompanied by bronchial breathing and bronchophony or ægophony. The writer has not met with this physical sign.

Comparatively small quantities of fluid may suffice to separate the heart from the chest-wall, and its impulse may consequently be diminished or imperceptible. This sign, however, though valuable when we see it appear, is far from constant, for it is remarkable how large a collection of fluid may fill the back of the pericardial sac, while the heart seems to float up in contact with the chest-wall. Even with extensive effusion, an impulse can often be felt in the fourth interspace.

The physical signs above enumerated are, positively, of the greatest value: a friction-sound is conclusive as to the presence of lymph; and increased dulness upwards, with weakening of the cardiac sounds, points decidedly to effusion, although we must not forget the possible presence of mediastinal abscess or tumour. Negatively the physical signs are less certain, for one may fail to discover pericarditis a few hours before death in cases of double pleuro-pneumonia, and yet the heart may be found covered with recent lymph.

Symptoms.—These vary remarkably; and in Bright's disease pericarditis is often latent. Good authorities state that pain in the cardiac region and in the epigastrium may be severe, and that pressure over the heart or on the pit of the stomach may cause distress; but in most cases the patient feels no pain, nor is there any tenderness. It was maintained by Bouillaud and by Addison that pericarditis is painful only when it is associated with pleurisy, the pericardium itself being insensitive both in health and disease. Severe anginal pains are probably due when present, which is very seldom, to some complication.

When pericarditis is acute, and particularly when there is large and rapid effusion of fluid, there is much dyspnoea. The patient can hardly speak for want of breath, his features are anxious and drawn, his nostrils dilate with each inspiration, and he sits with his head raised; but it is remarkable that, when there is copious effusion, he sometimes lies by choice flat in his bed, with scarcely a pillow, because the least elevation of the head produces faintness. In most cases, however, there is marked orthopnoea.

The heart's action may be regular or irregular, quiet or greatly disturbed with distressing palpitation.

The pulse in acute pericarditis may be quite unaffected. Usually, however, it is more frequent than normal, and sometimes rises to 150. In cases with effusion it often becomes irregular or intermittent.

The earlier writers mention the occasional occurrence of violent cerebral disturbance in acute pericarditis. Maniacal delirium, rapidly fatal, is sometimes present; and the case has been regarded as one of cerebral inflammation, until the autopsy showed that the pericardium was the seat of disease. But similar cerebral symptoms occur in acute rheumatism, independently of pericarditis, when the temperature is greatly raised; and this renders it doubtful whether, when pericarditis is present, it is really concerned in the production of the delirium. The same may be said of the

choreic movements occasionally observed in pericarditis, for there is a very close relation between rheumatism and chorea. Apoplectiform stupor, hemiplegia, or convulsions may probably be traced to embolism of the cerebral vessels from endocarditis. All the symptoms mentioned in this paragraph appear to be confined to cases of rheumatic pericarditis, and not to belong properly to the pericardial inflammation.

Dysphagia has been observed in cases of pericardial effusion, and has been referred to pressure on the œsophagus by the sac distended with fluid.

Diagnosis.—This is not always easy. A to-and-fro rub is usually unmistakable, for it has never the blowing character of an endocardial murmur. But a soft systolic rub may resemble some kinds of valvular or functional bruit; and a pleural friction-sound may acquire a cardiac rhythm if its seat is the anterior margin of the left lung.

The chief points of distinction from endocardial murmurs are the systolic or double rhythm of a pericardial rub, its loudness (*i. e.* its apparent nearness), its occasional increase on pressing the stethoscope, its “shuffling” or scraping character, and its not being conducted upwards towards either clavicle, nor carried round to the axilla. In most cases the pericardial friction-sound would be recognised by an uninstructed listener as due to two surfaces rubbing together, whether the character of the noise reminded him of a piece of folded cloth, or a momentary grazing of two hard surfaces, or the harsher friction of sandpaper, or the creaking of new leather. It is never blowing in character, nor musical, nor does it increase in loudness up to its end.

When effusion has occurred before the case is seen, diagnosis is often difficult. The best rule is to map out the limits of cardiac dulness on the surface of the chest, and watch its shape and daily progress.

The first difficulty is to distinguish pericardial from pleuritic effusion on the left side, pushing the heart over, and continuous with the cardiac dulness. The displaced impulse and seat of the first sound ought to clear up such a case. A circumscribed empyema might more closely simulate pericardial effusion, but the impulse would still be felt and the sounds be plainly heard. When the patient has not been previously seen, it may be necessary to explore the seat of dulness with an aspirating syringe.

The next question is whether the dulness is due to distended pericardium or to mediastinal suppuration or new growth (*cf.* pp. 150, 156).

Lastly, a pericardial effusion has been mistaken for an enlarged heart, and a large heart with adherent pericardium has been punctured in the belief that there was pericardial effusion. The way to avoid such error is to remember its possibility and expend the greatest care in palpation and auscultation.

Event.—It is rare for acute pericarditis to be the direct cause of death. In the pericarditis of acute rheumatism the immediate danger is very small. In Bright’s disease, death often follows quickly upon the occurrence of pericarditis as a complication, and sometimes effusion accumulates so rapidly as directly to hamper the heart, apparently by interfering with its diastole.

In favourable cases the symptoms gradually subside, and generally disappear in from twelve to twenty days; the fluid effusion diminishes by absorption, and a *redur* friction-sound may be heard. After a time the lymph is also absorbed; but before this occurs the pericardial surfaces are often more or less extensively glued together, and become permanently

adherent. There is no reason to suppose that adhesions occur in every case of pericarditis in which fibrinous coagulation has occurred; but it is probably a more common result than complete return to a normal condition.

In most cases pericardial adhesions follow ordinary rheumatic pericarditis, or occasionally that of Bright's disease; but in 153 cases recorded after death, Dr Perry found no less than twenty-three in which the inflammation was tuberculous in origin.

Adherent pericardium.—The anatomical characters of pericardial adhesions vary greatly in different cases. Sometimes, especially after the lapse of a long time, they are reduced to a mere film of connective tissue, which the fingers can tear through with little difficulty. In other cases they are exceedingly tough, so that the only way to denude the heart is to strip off pericardium and adhesions together from the muscular fibres. Again, they may be uniformly of great thickness; or they may include masses of altered lymph, accumulated in certain parts of the pericardial sac, and especially round the great vessels; or they may be formed of successive layers of fibrin more or less easily separated. Lastly, the lymph may in course of time undergo calcification, and thus the heart be enclosed in a rigid case.

Obliteration of the pericardial sac by adhesions did not escape the notice of the older pathologists, but they supposed it to be a congenital defect.* Although its real nature has been long understood, its clinical import has been disputed. Probably this depends on the nature of the adhesions: if thin and areolar they appear not to hamper the heart's movements in any way, but a thick mass of hard fibrous tissue closely surrounding the organ may undoubtedly give rise to serious symptoms. Another distinction is between pericardial adhesions which occur in adult life, and are often innocuous, and those which form in childhood, and hamper the growth of the heart.

By Hope and other early auscultators it was maintained that an adherent pericardium always tended to cause hypertrophy of the heart. But the result is not constant. Indeed, the presence of thick pericardial adhesions is often associated with atrophy and dilatation of the ventricular walls; in part, perhaps, the result of the myocarditis which often accompanies pericarditis rather than of the adhesions themselves.† Dr Perry found in ten consecutive cases of adherent pericardium in children dying between seven and thirteen, that there was hypertrophy as well as dilatation of the heart, on the right side as well as on the left.

Dr Sequeira has lately drawn attention to the bearing on the pathology of pericardial adhesions of the observations made on animals by Hill and Barnard ('Journal of Physiology,' 1898, vol. xxii, p. xliii). In health the inelastic resistance of the fibrous pericardium prevents undue distension of the right side of the heart. But when softened by inflammation the pericardial sac yields either to acute effusion in the serous cavity, or to gradual dilation and hypertrophy of the right side of the heart. Dr Sequeira believes that this is a more important cause of symptoms than

* The pericardium really may, as a rare anomaly, be congenitally absent. Dr Matthew Baillie described this condition in the first volume of the 'Med.-Chir. Trans.' See also his 'Morbidity Anatomy,' p. 13.

† See the late Dr G. H. Barlow's remarks in the 'Guy's Hosp. Reports' for 1847, Sir William Gairdner's in the 'Edin. Med. Journ.,' 1851, Sir Samuel Wilks's article in the 'Guy's Hosp. Reports' for 1871, vol. xiv, p. 196. Also the late Dr Sturges's article in 1892 ('Lancet,' vol. i, p. 622), and Dr Sequeira's in 1899 with 130 cases ('Med.-Chir. Trans.,' vol. lxxxii, p. 401).

myocarditis, whether superficial and acute, and accompanied with fatty degeneration, or chronic with fibrous degeneration and overgrowth.

As Wilks long ago showed, most of the fatal cases due to adherent pericardium occur in the earlier period of life, in childhood or youth. If a patient with adherent pericardium lives to be over twenty, he is not likely to die from its effects. And when this condition is found above twenty-five or thirty, or even in old age, the patient has died of some other complaint. Dr Sequeira has shown that the effects of pericarditis are most apt to show themselves clinically about 13—15 in the case of girls, and about 17 and 16 in the case of boys.

The early auscultators attempted to diagnose adherent pericardium by various signs which experience has shown to be fallacious. One of these was the occurrence of systolic depression at the site of the cardiac impulse. Slight retraction of the spaces close to the sternum during the systole is by no means uncommon even when the pericardium is healthy; but it appears probable that obliteration of its cavity may generally be inferred when a considerable area of the chest wall is drawn in. Still, however, we may doubt whether this can occur without the pleura over the heart being adherent and the left lung being withdrawn from its natural position; and if so, it might perhaps be met with as a result of old pleurisy apart from any pericardial adhesion.

This systolic drawing-in instead of impulse was first carefully investigated by Skoda. He showed that it depends upon the natural movement of the heart in systole (downwards, forwards, and to the left) being hindered. The atmospheric pressure is then unsupported, and slight depression in the fifth space ensues. Probably for this result there must also be some adhesion or atrophy preventing the ear-shaped process of the left lung from expanding and filling the space. Another cause of the same phenomenon is dense adherence between the visceral and parietal pericardium, and between the parietal pericardium and the chest wall. This may be the result of rheumatic pericarditis, but is more likely when there has been extensive thickening of the anterior mediastinum in addition.

Basal adhesions are much more effectual in producing this result than apical, as was proved by Weiss and Friedreich.

Dr John Broadbent has observed systolic retraction of the two or three lowest intercostal spaces and ribs on the left side in cases of adherent pericardium.* See Dr Ord's remarks on the symptoms of adherent pericardium in the seventeenth volume of the 'St Thomas's Hospital Reports.'

Some observers have endeavoured to diagnose pericardial adhesion from the fact of the heart's impulse not being displaced during inspiration, or from the position of the organ remaining unaltered when the patient lies on different sides; but these signs are very uncertain.

Dr Osler has described as a sign of adhesion of the pericardium, in the phrenic region, systolic retraction upwards, causing a depression between the seventh and eighth costal cartilages in front and between the eleventh and twelfth ribs behind.

In a paper published in the 'Bristol Med.-Chir. Journal' for June, 1894 (vol. i, p. 906), Dr Theodore Fisher drew attention to the occurrence of a diasystolic apical murmur as a sign of pericardial adhesions in children. He had observed this in the wards of Guy's Hospital, and the fact was confirmed by the writer, by Dr Hale White, and by Dr. Perry. In thirteen

* In his monograph on "Adherent Pericardium," 1895.

cases of adherent pericardium with no mitral stenosis examined after death, a presystolic bruit had been heard in five.

Since the effects of tightly adherent pericardium are most marked while the heart is still undergoing growth, this condition is of much more serious importance in children than in adults. The immediate effect of tight adhesions seems to be prevention of free diastole, but afterwards systolic contraction is probably also hampered; the ultimate result is most frequently, at least in children, dilatation with hypertrophy. Apart from the physical signs just discussed, the want of development of the heart leads to symptoms of a weak circulation, and to the arterial anæmia and venous congestion which we saw to be the ultimate results of all organic cardiac lesions if not otherwise cut short. The type of disease is mitral rather than aortic, and there is often considerable dropsy. The right as well as the left ventricle is found enlarged.

Accordingly, if we find a child with symptoms of chronic cardiac disease, but with no audible murmur, and particularly if he has suffered from rheumatism, we may with great likelihood diagnose an adherent pericardium. Frequently in such cases there is valvular disease as well, and then pericardial adhesions make the result far more serious.

Mediastinal implication.—A remarkable case was recorded by Griesinger in 1854, where not only the pericardium was found adherent and enormously thickened, but the adjacent anterior mediastinum was filled with a continuous mass of indurated fibrous tissue.

He observed in this case the *pulsus paradoxus* or *pulsus inspiratione intermittens*, i. e. an irregular and frequently an intermittent pulse which becomes imperceptible with each inspiration, the cardiac beats being unaffected, whether regular or irregular before. Moreover the distended veins, instead of collapsing as usual with the free entry of blood to the right auricle which follows the expansion of the thorax, became fuller with each inspiration, apparently from the dense fibrous adhesions of the mediastinum dragging upon the innominate veins and superior cava. Kussmaul described three similar cases in 1873, and explained the diminution and cessation of the radial pulse as due to stretching and narrowing of the aortic arch by adhesions. Similar cases of chronic hypertrophic pericarditis, with the same intermission of the radial pulse and fulness of the veins during inspiration, have been described by Traube, Bäumlér, and other observers, both in Germany and in England (cf. *supra*, p. 151). In some of these cases there were concomitant hypertrophic adhesions of both pleuræ and of the peritoneum (as in the case referred to, vol. i, p. 1084).

Milkspots.—We frequently find the visceral pericardium thickened and opaque in patches, on the prominent parts of the right ventricle in front or of the left behind, or on the auricles. These “milkspots” or “corns,” as they have been termed, are not adherent to the parietal layer, and are not produced by acute pericarditis. They are the result of friction, and are seen most often when the heart is hypertrophied, and where there is more than usual friction, as from a soldier’s old-fashioned cross-belt. They are probably of no clinical significance.

Hydropericardium.—The pericardium may be filled with fluid under two conditions—excessive inflammatory exudation, and passive effusion as a part of general dropsy. In the latter case it is seldom recognised during life and seldom gives rise to serious symptoms. Moreover, in cases of renal dropsy, it will commonly be found that (as in hydrothorax) the effu-

sion is not pure serum, but shows by the presence of fibrin that the process is active as well as passive. Accordingly, we may regard hydropericardium as usually the result of inflammation, and not of mere dropsy. An ounce or two of clear serum is often found in the pericardium after death from anæmia or wasting diseases, and is productive of no symptoms during life.

The healthy pericardium of an adult man holds from twelve to fifteen ounces of water, but under pressure will expand so as to hold rather more. The largest amount of effusion observed by the writer in acute cases was fifteen ounces of serum in a case of rheumatic pericarditis in a man of twenty-two; and in chronic cases, forty-two ounces in a woman who died of Bright's disease.

When, however, the fibrous sac has been softened by inflammation and gradually distended by long-continued exudation, it may hold one, two, or even three pints.

With the pericardium thus distended, the normal cardiac dulness is increased, as in dilatation with hypertrophy of the heart, but it is increased upwards and to the left, not downwards, except in the last stage of enormous accumulation.

The lung may be compressed at the back of the chest. See an interesting paper by Dr Ewart in the 'Brit. Med. Journ.,' for March 21st, 1896 (p. 717). It has been observed by Professor Bäumlér that when the sac is thus full of liquid effusion, the normal respiratory waves of blood-pressure become so much increased as to be perceptible in the sphygmographic tracing of the pulse. The most marked symptoms are usually orthopnoea, stridor, irregular pulse, and præcordial oppression.

Such cases appear for the most part as sequelæ of rheumatism or Bright's disease, when the acute symptoms have passed off, and then their recognition is not difficult, particularly if the case has been watched from the beginning. If seen first when the effusion is already extensive, diagnosis is more difficult, and most of all when there is also effusion in the left pleura.

In a case figured many years ago by Sibson, the liver and stomach were pushed downwards, the lungs upwards and backwards, and a large part of both chest and abdomen was filled by the monstrous pericardial sac, which contained fifty-two fluid ounces.

Hæmopericardium.—The serum of a pericardial effusion is sometimes tinged with blood. This may be caused by injury, but more often it denotes malignant disease, as in similar cases of pleurisy and ascites. Dr Allbutt met with a remarkable case where effusion of pure blood into the pericardium followed rupture of a coronary artery in an elderly lady, who lived about sixteen hours after the onset of symptoms (quoted in Dr F. Roberts's article in 'Allbutt's System,' p. 767). Rupture of the heart or of an aneurysm at the root of the aorta leads to effusion of blood into the pericardium; and death—which is not always sudden—ensues owing to pressure on the heart preventing its diastole. Several cases of hæmopericardium from scurvy, however, have occurred in Russia, in which paracentesis was performed with success.

Pyopericardium or purulent pericarditis may occur in Bright's disease or from extension of empyema; but it is most common as the result of rupture of a pyæmic abscess of the heart, or as a complication of pneumonia. Dr Dickinson recorded a case in which nearly a pint of pus was removed from the pericardium ('Clin. Trans.' 1889, p. 48).

Pneumopericardium.—Gas in the pericardium is an exceedingly rare condition, resulting either from accidental communication with the pleural cavity or from decomposition of pus.

Prognosis.—Primary rheumatic pericarditis is seldom dangerous except in children, and even then the danger is rather from serous effusion or subsequent adhesions than directly. When secondary to pneumonia, to Bright's disease, or to specific fevers, pericarditis is a very grave complication, and so it is when tuberculous or septic in origin.

Treatment.—The earlier auscultators sought to recognise pericarditis at the earliest possible moment, in order to combat its progress by antiphlogistic measures. It is most instructive to peruse the graphic and confident description, by a writer of literary skill like Latham ('Lectures on Diseases of the Heart,' xii—xv), of the signs of acute pericarditis, of the vigorous treatment of the disease by bleeding and by mercury, and of its rapid subsidence under these measures. At the present time few would regard the danger as imminent, and probably none would believe that the classical treatment would avert it. Venesection is now seldom employed in pericarditis, and if leeches are used it is only with the object of relieving distress and dyspnoea. The treatment now generally adopted is as follows:—The patient is kept in bed; the præcordial region is covered with a poultice, or a thick layer of cotton wool; and light fluid nourishment is given, with a saline mixture. To relieve pain and quiet the heart's action, Dr Bäumlér, from experience in his own case, advises the application of a bladder of ice.

Nevertheless, not only may a few leeches, applied over the sternum in the early stage of pericarditis, relieve distress and perhaps limit the inflammation, but, when there are symptoms of embarrassed circulation with orthopnoea, an irregular pulse, arterial anæmia, and venous congestion, the abstraction of five or six ounces of blood from the arm is found in some cases to give remarkable relief, and probably is never injurious. The remedy, however, is used to meet a special complication, and not with the intention of curing the disease. The writer once obtained striking relief in a case of adherent pericardium with great dyspnoea in a boy of nine, by venesection to four ounces.

Some physicians apply a blister as soon as a pericardial rub is audible. But beside the practical inconvenience of this treatment in preventing auscultation, its benefit is more than doubtful, and in cases of Bright's disease blisters should, if possible, be avoided.

When, however, large effusion has taken place, a blister, quickly followed by a poultice, so as to promote free effusion of serum, is undoubtedly efficient in hastening absorption; at any rate, it has more than once been followed by a rapid diminution of the dulness. Iodide of potassium is frequently given, with the hope of favouring absorption, and its efficacy as a diuretic makes this reasonable.

The value of a blister was well shown in two cases under the writer's care. One was a boy of seven, with such urgent symptoms of distress from distension of the pericardium after rheumatism that it was intended to tap him. By the prudent advice of a senior colleague a large blister was first tried, and (when that did not rise satisfactorily) blistering fluid with a poultice afterwards, and complete success followed. The other was a girl of fourteen who had a (probably rheumatic) pericardial rub with no sym-

ptoms whatever. She was therefore left without treatment for a week without the least change in the physical signs. A blister was then applied, and the rub disappeared in twenty-four hours.

Paracentesis.—When dyspnoea is urgent, and suffocation threatens, the operation of tapping the pericardium should be considered; its performance is justified by the imminence of the danger or by the failure of a large blister to relieve. "For when a patient is suffocated by pericardial effusion no medicine avails." This operation was suggested more than two centuries ago by Riolanus, but it appears to have been first practised by Romero, of Barcelona, in 1819, and with success (quoted as the first of fifty cases, in a dissertation on "*Paracentesis Pericardii*," by Hindenlang, 1879). Aran once injected iodine into the pericardial sac after removing about two pints of fluid, and the patient recovered.

The operation of paracentesis was first performed in England by Mr Wheelhouse, of Leeds, on a patient of Dr Allbutt's, with good result ('*Brit. Med. Journ.*,' 1868 and 1870; '*Lancet*,' 1869. Eighty cases were collected and discussed by Dr Samuel West in the '*Med.-Chir. Trans.*,' for 1883 (vol. lxi, p. 235, table p. 268 *seqq.*).

A hypodermic syringe may first be inserted to remove all doubt as to the diagnosis. A slight incision is then made, and the trocar is passed gently into the fourth or fifth left intercostal space, about an inch away from the sternum, so as not to wound the internal mammary artery.

The operation has now been performed in a considerable number of cases, and when the pericardium is found after death distended with pus or with serum, we regret that paracentesis was not carried out. The writer has several times had the pericardium tapped, once only with marked benefit, but never with any untoward results.

One of the most successful cases of paracentesis pericardii recorded was Dr S. West's. The patient was a lad of sixteen, who had been suffering from increasing dyspnoea for three weeks before he applied for admission to the Victoria Park Hospital. The physical signs led to the belief that a large pericardial effusion existed, and he was accordingly tapped. Fourteen ounces of pus were withdrawn. In a few days the same amount was again taken away; but the fluid still re-forming, an incision was made into the sac through the fifth interspace and a drainage-tube inserted. Two quarts of purulent fluid were thus removed, and the patient gradually but completely recovered.

ENDOCARDITIS AND VALVULAR DISEASE OF THE HEART

“Oft have I seen a timely-parted ghost
Of ashy semblance, meagre, pale, and bloodless,
Being all descended to the labouring heart,
Who in the conflict that it holds with death
Attracts the same for aidance 'gainst the enemy,
Which with the heart there cools and ne'er returneth
To blush and beautify the cheek again.”

2 K. Henry VI, iii, 2.

ENDOCARDITIS—*The Subacute, Simple, or Benign form—Its relation to rheumatism and its etiology generally—Its anatomy—Its signs.*

The Chronic or Sclerotic form—Its origin and anatomy—Its effects.

The Ulcerative, Septic, or Malign form—Its origin, anatomy, and results—Its relation to pyæmia and other infective processes—Prognosis—Treatment.

VALVULAR LESIONS—*Their origin and anatomy—Effects on the heart itself and on other organs—cardiac dropsy—dyspnœa and orthopnœa—hæmoptysis and other symptoms—Physical signs: cardiac murmurs—their rhythm—their locality and conduction—their physical cause.*

Aortic or sigmoid stenosis—Aortic regurgitation—Mitral stenosis—Mitral regurgitation—The anatomy, physical signs, and symptoms of each.

Lesions of the pulmonary or dextro-sigmoid valve and of the tricuspid—Functional murmurs—Combined lesions.

Relative frequency of valvular lesions—Their general prognosis—Special prognosis as to duration and suddenly fatal termination.

Treatment of organic diseases of the heart, in general and in particular.

Congenital cardiac lesions—Disease of the heart in childhood and in old age.

CARDITIS, or inflammation of the substance of the heart, was formerly described, but rather in accordance with the belief that every tissue was liable to every disease than from observation. There is no true carditis, as there is no true inflammation of the brain. The degenerative processes of the muscular tissue of the heart have been described in the last chapter. The inflammations of the heart do not directly concern the muscle, but the covering and lining membranes. The former, pericarditis, was the subject of the last chapter. The latter, endocarditis, with its effects, is still more important, and will now be considered in its three distinct forms.

SIMPLE ACUTE ENDOCARDITIS.*—The first is a moderately acute endo-thelial inflammation resembling pericarditis, pleurisy, and peritonitis, more closely perhaps than arteritis or phlebitis. The structure of the lining membrane of the heart is almost identical with that of the great serous cavities, the chief differences being the absence of close relation with the lymphatic system; this and the fact of the former membrane being continually bathed with the circulating blood are the most important peculiarities of the endocardium. It is remarkable that although the endocardium is continuous with the intima of the arteries and veins, no acute inflammation of the latter is met with, like the endocarditis of rheumatism.

Simple endocarditis is clinically most allied to pericarditis and pleurisy, next to peritonitis, and more distantly to meningitis and synovitis. It is never suppurative, and does not lead to ulceration.

The process is subacute in its course, mild in its effects on the condition of the body in general, serous and fibrinous (or, as the German pathologists call it, "croupous") in the character of its exudation, adhesive and contractile in its effects. Adhesions, however, can only occur where the parts affected come into contact, and hence are limited to the neighbourhood of the valves; the formation of delicate bead-like nodules of fibrin, "puckering" and sclerosis, are the characteristic effects of valvular endocarditis, which have led to the epithets "warty" (*endocarditis verrucosa*) and "vegetative" being applied to this form of inflammation. The features, however, which have earned the last epithet, are chiefly due not to inflammatory exudation, but to coagulation of fibrin from the circulating blood. Away from the valves, endocarditis produces opacity and thickening, like the milkspots of the pericardium.

Endocarditis is usually limited to the left chambers of the heart; it is extremely rare to find the right auricle, ventricle, or valves affected, except along with those of the left side, and in the great majority of instances they do not share in the inflammation. In the foetus the right side is *more* frequently affected by endocarditis, and this appears to show that the more active function of the systemic half of the heart in extra-uterine life is the cause of its being more obnoxious to disease after birth.

Ætiology.—By far the most frequent cause of simple endocarditis is Rheumatism, *i. e.* the acute febrile disease, with multiple synovitis, which is often called rheumatic fever. The so-called "chronic rheumatism," "muscular rheumatism," "rheumatic gout," "gonorrhœal rheumatism," and the other disorders which are still often vaguely styled rheumatic, show that they have no claim to the title by never producing endocarditis.†

The only other diseases which produce endocarditis as an occasional complication are Chorea, Scarletina, and Pyæmia (see three cases published by Dr Fagge in the 'Path. Trans.' for 1866). Some authors mention smallpox, measles, and diphtheria, and others acute or chronic Bright's disease, but the evidence on which these statements rest is far from conclusive. Even scarlatina is probably only operative by its liability to be followed by multiple synovitis; and a choreic murmur has been most often preceded by genuine rheumatism. Pyæmic endocarditis is very rare and of no clinical importance; and if we consider chorea to be itself directly and closely allied to rheumatism (*cf.* vol. i, pp. 882, 892), the murmur of chorea

* *Synonyms.*—Subacute endocarditis—Endocarditis simplex v. benigna—E. verrucosa et vegetans (in part)—Rheumatic endocarditis.

† The only apparent exception is gonorrhœal arthritis; but that is only when the articular affection is local, unusually severe, and suppurative; *i. e.* when it is not due to gonorrhœal but to streptococcal infection.

may be ascribed to rheumatic endocarditis even when there is no history of preceding rheumatic fever. Pathologically, then, as well as clinically, acute non-infective endocarditis may be regarded as "rheumatic" in the strict sense of the word.

It usually begins early in the course of the fever—occasionally before the synovitis has appeared, or altogether without synovitis, as seems proved by the testimony of such authorities as Graves, Stokes, Trousseau, and Latham.

Acute endocarditis is more frequent in the mild rheumatism of children than in the more severe disease as seen in adults, and far more common between puberty and thirty years of age than later.

Anatomy.—Inflammation is recognised after death by the presence of an exudation which makes the smooth, shining endocardium opaque and rough, and thus leads to deposition of fresh fibrin from the circulating blood. It is this lodgment of fibrin on the inflamed surface which constitutes the peculiarity of endocarditis, and makes it differ from inflammation of other parts. The living blood is always ready to deposit fibrin on a roughened surface, especially when the current is in any way checked.

The first indication of endocarditis is probably redness and vascularity, afterwards the appearance of a row of fibrinous granulations. These bead-like nodules are formed along the curved line on each side of the corpus Arantii where the sigmoid valves touch, and along the corresponding edges of the mitral or tricuspid valve. If examined microscopically they are found to consist of ordinary inflammatory elements, viz. leucocytes, and fibrin. The whole of the valve may be infiltrated with the inflammatory products, and thus become swollen and softened. In recent endocarditis these minute granulations along the valves are the only indications of the inflammation; at a later stage they become larger, and projecting into the stream of blood, attract coagulating fibrin. Thus vegetations on the valves have a double origin, being originally derived from the valve itself, and having therefore a true inflammatory source, but subsequently increased by deposition of fibrin from the blood. It is not only the valvular endocardium that is affected; the left auricle is often found lined with a thick opaque membrane, and all the cavities may show the same evidence of past inflammation.

Symptoms.—The presence of endocarditis is indicated by a murmur or bruit (Fr. *bruit anormal*) heard over the region of the heart. The disappearance of the murmur by no means warrants denial of previous endocarditis, since there is good reason to believe that inflammation of the valves may be completely recovered from. In such case we suppose that the temporary incompetence of the valve has been removed. The local signs which point severally to the different valvular lesions will be considered at length below (pp. 243, 249).

The general symptoms of endocarditis are very slight. In itself it probably produces little pyrexia, and only moderate acceleration of the pulse; nor is pain by any means a symptom to depend on.

¹ Most cases even of the more severe form of endocarditis run their course and give no pain or other warning of the presence of a process of grave import for the future.

The prognosis is favourable as far as the endocarditis is concerned. The future evils arise from the deformities of the valves which are likely to be produced.

CHRONIC FIBROUS ENDOCARDITIS.*—Subacute inflammation of the valves often leads to their puckering and incompetence, with consequent regurgitation of the blood-stream through the leak, when the valve is closed. Often there is so much cicatricial thickening that obstruction is the result, from narrowing (contraction, stenosis) of the opening, with or without concomitant leaking from incomplete closure of the valve. Chronic endocarditis produces similar effects; but while in most cases it is merely the last stage of acute endocarditis, it is sometimes a slow and insidious process from the first. The valves become stiff and thicken, and the ostia gradually contract. This process, chronic from the beginning, is probably never the result of rheumatism; it is found associated with atheroma, intemperance, gout, and cirrhosis of the kidneys. It may be observed in youth, but becomes more common in the later periods of adult life. It is often complicated by deposition of calcic and magnesian phosphates, with carbonates of the same metals, in the indurated fibroid tissue. It thus becomes anatomically identical with atheroma of the arteries, and with this chronic arteritis it is associated clinically as well as in pathology.

The relation of chronic valvular deformities to rheumatism is through the acute form of endocarditis; probably more than half of the cases are thus rheumatic in origin. See the writer's paper in the 'Guy's Reports' for 1871, and Sir Dyce Duckworth's in the 'St Bartholomew's Reports' for 1877.

The relation of valvular lesions to age, strain, lead poisoning, gout, and renal disease is through atheroma.

From an analysis of the records in Guy's Hospital, Dr Pitt has found that sclerotic endocarditis, as it affects the mitral valves, and leads to stenosis of the left auriculo-ventricular ostium, is frequently associated in women with uterine disorders which lead to ascending nephritis or to consecutive renal cirrhosis.

The late Dr Peacock and some other writers, including Douglas Powell ('Lumleian Lectures,' 1899, p. 68), have believed that many cases of obstructive sclerosis of the mitral orifice are congenital.

The origin of this sclerotic form of endocarditis is therefore to be sought in (1) the acute or subacute rheumatic form; (2) gout and chronic Bright's disease, or rather the sclerotic "diathesis," if we may use such a phrase, which forms part of arterio-capillary fibrosis; (3) strain on the aorta and its valves from over-exertion; (4) some cases appear to be due to syphilis, and may be compared with the aneurysms which follow this disease; (5) a certain number of cases remain, and some of these perhaps are congenital.

The symptoms, the prognosis, and the treatment of Chronic endocarditis are identical with those of the structural valvular lesions which it produces.

ACUTE ULCERATIVE ENDOCARDITIS.†—There is a marked distinction between the comparatively benign form of cardiac inflammation above described and a more acute, dangerous, and septic form. This may be compared with the virulent forms of peritonitis which occur in the puerperal state. Beside the usual fibrinous products of endocarditis, which are

* *Synonyms.*—Sclerotic endocarditis—Valvular atheroma—Chronic fibroid contracting endocarditis—Endocarditis verrucosa—Endurcissement cartilagineux (*i. e.* fibrous) et osseux *i. e.* calcareous) des valvules du cœur (Laennec).

† *Synonyms.*—Arterial pyæmia (Wilks).—Acute diphtheritic endocarditis (Eberth)—Malignant endocarditis (Osler)—Infective endocarditis—Acute septic endocarditis—Endocarditis vegetans (in part).

present in exuberant amount, there is ulceration of the endocardium, often leading to rupture of the chordæ tendineæ or perforation of a valvular cusp. Moreover micrococci or septic bacteria are found in the masses of fibrin, and the morbid process is accompanied by symptoms of septicæmia. Lastly, detached fragments of fibrin, which in the case of ordinary endocarditis produce only mechanical results, by blocking the arteries into which they are carried, become now the means of transport of infective microphytes, which excite similar suppurative "pyæmic" inflammation wherever they lodge as emboli. Hence this variety of endocarditis; and the whole process was described by Wilks as one of internal or arterial pyæmia ('Guy's Hosp. Rep.,' 1870).

Ulcerative endocarditis is, as a rule, a secondary process, attacking a valve already injured by chronic sclerotic inflammation, most frequently of rheumatic origin. It very rarely begins as a direct result of chorea, scarlatina, or rheumatism, and is unknown as a consequence of Bright's disease, of gout, or of syphilis.

That pneumonia is an important antecedent of ulcerative endocarditis is certain, but there is no doubt that the frequency of this association in the cases reported from Montreal by Dr Osler was exceptional (16 cases of septic endocarditis in 100 of pneumonia). In 352 cases of pneumonia at Guy's Hospital, collected by the writer, and 73 seen in consultation, there were only seven of endocarditis.

Other acute diseases, particularly a fresh attack of rheumatism, enteric fever, and erysipelas, may light up this septic endocarditis.

As a primary disease it is rare, and seldom occurs in septicæmic conditions, in which it might be looked for, as pyæmia, puerperal fever, diphtheria, and scarlatina.

Ulcerative endocarditis, like the benign form, is, as a rule, confined to the left side of the heart, but affects the pulmonary or tricuspid valves in addition more often than is the case in non-infective cases. It is frequently found on the endocardium of the ventricular or auricular wall, and sometimes direct infection can be traced from a mass of fibrin, which sways backwards and forwards with the blood-stream, and touches part of the wall of the heart. It only produces a similar affection of the arteries when an embolus lodges in them.

Sometimes it begins with a rigor, followed by high pyrexia with sweating, and this may be repeated as in other cases of suppurative fever; but often the onset is insidious, and the symptoms those formerly known as "typhoid." In some rare cases, there are no symptoms but those of cardiac disease with embolism. In all cases anæmia is a very marked symptom.

The *diagnosis* of ulcerative endocarditis depends, first, on the same physical signs as those of the benign form, for the mechanical effect of both on the valvular mechanism is the same; and secondly, upon the raised temperature, and the signs of infective embolism of distant parts. When we find a patient in a state of fever, and discover a cardiac bruit; when to this are added hemiplegia, aphasia, or other symptoms of cerebral embolism, albuminuria, or hæmaturia, pointing to embolism of the kidneys, or increased splenic dulness, with a palpable tumour and tenderness in that region; or when an acute aneurysm forms in the arteries of the limbs—we may then safely diagnose ulcerative endocarditis. It is no doubt often present when none of these symptoms can be detected, and should always be suspected when a case of valvular disease is unusually prolonged and

rebellious to treatment, particularly if the murmurs heard vary from time to time.

The diseases for which Septic endocarditis is most often mistaken are Enteric fever, Malarial fever, and acute Rheumatism with synovitis.

Anatomy.—The first stages are like those of the benign form, but the fibrinous masses are found more abundantly, and are more easily detached. Hence embolism is particularly common.

When these vegetations are long and mobile, they come into contact with the surface of the ventricle, and there set up a fresh inflammatory process, so that the part touched is also soon covered with a patch of fibrin. In this manner a vegetation on one valve may bore a hole through another with which it comes in contact. The mitral seems more likely to be thus affected than the aortic valve.

Subsequently the texture of the valves becomes involved, the tissue is loosened, and the process goes on to *ulceration*. A rent or perforation takes place in the aortic or mitral cusps, or an aortic valve may be partially detached from its base, or the chordæ tendineæ of the mitral ulcerated and broken, their loose ends floating about and covered with fibrin. As a consequence of these changes, adhesion or coalescence of the valves may take place, with obstruction at the orifices, or retroversion of the valves, leading to the worst kind of regurgitation.

An ulcer in the endocardium may burrow deeper and invade the muscle until an abscess is formed. This finally discharges its contents into the heart and constitutes an acute aneurysm. It usually occurs at the root of the valve, and it may sometimes reach the surface of the heart, when, if it bursts, it sets up fatal pericarditis.

The most constant secondary lesion is a swollen, soft spleen, with hæmorrhagic or fibrinous blocks from embolism of a branch of the splenic artery; next to that, corresponding pyramidal wedges in the kidneys, with a border of effused blood. Occasionally, petechiæ of the skin or mucous membranes or retina are seen and intestinal hæmorrhage from mesenteric embolism.

Microbes.—In most cases of ulcerative endocarditis fibrinous clots become detached, and infecting the blood, set up a fatal blood-poisoning; micrococci are frequently met with in them, as was first shown by Heiberg, of Copenhagen. The result is infectious embolism, causing "pyæmia," abscesses of the spleen, kidneys, and other viscera, including the heart itself, and aneurysms of distant arteries.

The most frequent microbes found in the ulcerations on the valves or the mural endocardium are streptococci or staphylococci. Sometimes gonococci or rod-shaped bacteria are present, and in cases of pneumonia the diplococcus (or pneumococcus) described above (p. 14). Acute ulcerative endocarditis is not so common an affection as the less dangerous and more chronic process.

Prognosis is always very grave in cases of septic endocarditis. Patients only recover as a rare exception, of which the following is a case.

A young man was under the writer's care in John Ward, May, 1893. He had suffered from rheumatism more than once, and had evidence of mitral incompetence. While in the hospital the temperature was high and very irregular, and blood appeared in the urine. The radial artery was next found to be blocked rather high in its course, but the collateral circulation soon compensated for this event. Then the anterior tibial artery was also obstructed low down, and a small aneurysm formed as the result, in the arteria dorsalis pedis of the right foot. This did not increase beyond the size of a large pea, and the patient's general condition improved, for he lost his fever and regained his appetite. At last he was so much better that, against advice, he insisted upon leaving the hospital, and

was last seen walking about, with his cardiac murmur as on admission, but saying that he was perfectly cured. The treatment had consisted in keeping him lying down, and giving him as much quinine and fatty food as he could take.

No efficient *treatment* is known, though quinine or arsenic is given in large doses. Dr Sansom recommends sodium-sulphocarbolate. Antistreptococcic serum has been injected in these cases, and in some instances with apparent benefit. The difficulty seems to be sure of the exact form of infective microbe before deciding on the corresponding kind of serum.* Another recent method of treatment, which is so far promising, is that of swallowing yeast or taking yeast extract. This was introduced by De Bacher (1893), and reported favourably of by Broadbent. Dr Vaughan, of Michigan (1897), has since introduced treatment by subcutaneous injection of solution of nuclein.

VALVULAR LESIONS.—The effect of inflammation of the endocardium where it lines the cavities of the heart is of little practical importance. The valves, however, are not only more often the seat of inflammation than the rest of the endocardium, but a very little damage to their delicate structure will interfere with their mechanical action. It is not always easy to say precisely how the early stages of acute endocarditis produce this effect. One would scarcely have supposed that a few minute nodules of lymph upon the edge of a mitral curtain would prevent its closing effectually when the ventricle contracts. Probably we must explain it by calling to mind the difference between acute inflammation of the skin, the eye, or the throat as seen during life, and its scarcely perceptible effects in the deadhouse. If we imagine the valvular curtains red, swollen, and oedematous, it is easy to understand how they fail in the nice coaptation upon which their efficiency depends. This at all events is certain, that in acute endocarditis the only physical signs met with are those which denote incompetence of one of the valves. Obstruction from contraction of an orifice can only be produced by a chronic process.

In the septic and ulcerative form of endocarditis above described, when cusps are eaten away or perforated, and chordæ tendineæ softened until they rupture, the resulting valvular inefficiency is easy to understand.

The effect of chronic or sclerotic endocarditis is permanently to pucker and deform the curtains, so that they no longer perform their part, while at the same time they become opaque and thick, less mobile and less flexible. In the case of the mitral and tricuspid valves the chordæ tendineæ also thicken and contract, so as to prevent the curtains floating up while the ventricle is filling.

Another process, moreover, is a frequent, one may almost say a normal, termination of the same series of changes. The circumference as well as the free edges of the valves slowly contracts after the inflammation is over, forming a true cicatricial fibrous tissue, which gradually narrows the orifice affected. When calcareous is added to fibrous degeneration, the narrowing is aggravated by the bulk of this new material. But beside the contraction of the whole aperture, the available opening for the passage of the blood is still more liable to be diminished by cohesion of the curtains or cusps, and to this process is probably due the most extreme stenosis of the auriculo-ventricular openings. In these circumstances the usual effect of the pressure

* See fourteen cases with three recoveries, given in tabular form by Sir Douglas Powell in his *Lumleian Lectures* (1899), p. 106.

of the blood-stream is to push the coherent valves into the ventricle or the outgoing artery, as the case may be; and thus to form a more or less funnel-shaped septum with a perforated apex. In extreme cases this perforation becomes a mere pin-hole and the orifice is all but obliterated, a condition seen in the case of the pulmonary orifice, as the congenital result of intra-uterine endocarditis of the right side.

Another form of deformity, which particularly affects the mitral valve, is for the orifice to become so stiff and narrow that at last it is a mere rigid slit, and receives the name of the "button-hole" valve.

When severe ulceration, with large adherent masses of fibrin is produced by septic endocarditis, the condition of the valve is liable to almost daily change. Regurgitation from extensive loss of substance of the curtains may be partially checked by deposit of fibrin, or this may still further shackle the cusp which has escaped ulceration; or one or more chordæ tendineæ may ulcerate through and break, allowing the curtain to which they are attached to break loose, like the detached sail of a ship, and to flap back into the auricle during systole.

Lastly, destruction of the valves may go so far, as is particularly seen at the orifice of the aorta, that one or more cusps are wholly eaten away, so that it sometimes seems as if almost the whole of the blood which leaves the left ventricle at its contraction must return with diastole, and one wonders how the circulation can have been kept on, however imperfectly, for an hour.

Effects of valvular disease on the heart.—We have seen that it is a physiological rule that obstruction in any passage causes hypertrophy in the muscular cavity next behind it (*supra*, p. 197), as seen in the case of the bladder from stricture, or in the intestine from a chronic obstruction; in like manner stenosis of the aortic orifice produces hypertrophy of the left ventricle, and stenosis of the mitral hypertrophy of the left auricle.

The effect of regurgitation through a valve is also to cause enlargement of the cavity next behind it, from its being continually over-full; it follows, therefore, that the left ventricle will be dilated in aortic regurgitation, and the left auricle in mitral regurgitation. In both cases there is habitual over-pressure of the blood in the cavity immediately behind the lesion, leading in most cases to both hypertrophy and dilatation (p. 195). The effect on the cavity next in front of the lesion will be habitual lack of due blood pressure and tension of its muscular walls.

Again, it is evident that any obstruction on the left side of the heart must affect the lesser circulation in the lungs, and so react on the right side of the heart; the effect on the right ventricle, tricuspid orifice, and auricle being the same as that of primary obstruction in the pulmonary capillaries from chronic bronchitis and emphysema; the right ventricle of the heart becomes dilated and hypertrophied, and as a consequence the right auricle also.

If we take the case of mitral stenosis, we find that the left auricle, in order to propel the blood through the narrow orifice, has more work thrown upon it, and becomes in consequence much hypertrophied, and at the same time its lining membrane opaque and thickened; the passage of blood through the lungs is in like manner retarded, and the right side also becomes tougher and thicker, as it does from long-continued obstruction in the pulmonary capillaries.

In the cases of disease of the sigmoid valves, in which there is usually an obstruction to the flow of blood as well as regurgitation, the left ventricle becomes hypertrophied and dilated to a monstrous size. The impediment to the escape of blood into the aorta impedes the flow through the mitral orifice, and so the left auricle next participates in the enlargement, and as a further consequence the right side of the heart also. Accordingly in such a case the whole heart is prodigiously enlarged, and is called "bovine." It frequently weighs 25 to 30 oz., and even heavier hearts are sometimes met with—up to 48 oz. in a case recorded by Dr Frederick Taylor.

Effect on the arteries and veins.—As the result of valvular disease of the left side of the heart the aorta is unaffected; its elasticity continues perfect, and it is not dilated; possibly it may sometimes be narrower and thinner than it would otherwise be, particularly in the case of growing children. The pulmonary veins are dilated. The over-pressure on the right side of the pulmonary circuit causes dilatation and thickening of the artery leading to the lungs, and also patches of atheroma as the result of the tension of its coats. The venæ cavæ are often found considerably dilated, but they do not appear to be thickened.

Effects upon other organs.—It will be convenient to mention in this place the changes which take place in other organs as the result of long-continued valvular obstruction or incompetence. Ultimately, at whatever point in the circuit it began, the final result is over-pressure in the systemic veins and low pressure in the systemic arteries.

In the case of the *lung* this venous congestion may be so great that blood is brought up during life, and the organ is found after death to have blood effused into the tissue, either in scattered foci, or as large circumscribed masses, described by Laennec and other French writers as "pulmonary apoplexy," and sometimes compared to damson cheese in appearance. This hæmorrhage is most often met with in the lower part of the right lung.

The mechanism of the pulmonary hæmorrhage in cardiac disease is probably not always the same. That it may be the result of mere mechanical distension of the pulmonary venules and capillaries is tolerably certain, but it is not common in cases of mitral regurgitation, and not so frequent in those of aortic valvular lesions as in those of mitral obstruction. Hæmorrhagic blocks are also seen in the rare cases of endocarditis of the right side of the heart when there is no extra pressure in the pulmonary area of the circulation. Hence it is probable that some other cause is at work in these cases, and the cause seems provided by embolism. Just as an embolus blocking an artery will cause hæmorrhage by reflux from the veins in the brain or the retina, the spleen or the kidney, so it will cause hæmorrhage in the lungs, when a venous thrombus is carried through the right chambers of the heart to the pulmonary capillaries, or when a clot from the tricuspid or right sigmoid valves or auricle reaches the same goal as an embolus.

If the blood do not actually burst through the vessels, the engorgement goes on until the capillaries are completely blocked and the alveoli of the lungs almost obliterated; the lung becomes very dense, hard, and fleshy, and sinks in water, the cut surface being smooth, without the granular aspect of hepatisation; this condition has been called "splenization." Sometimes, after a time a large quantity of pigment is formed, the alveoli are thickened, and the lung becomes dark and granular; this later stage is termed "brown induration." If inflammatory products are thrown out, and formation of

connective tissue takes place, the consolidation which then arises, combined with the granular pigment, produces an appearance which has been termed "brown indurated pneumonia." A close examination discovers the remarkable changes which have occurred in the vessels of the lung from the long-continued pressure; sometimes they have become varicose, and atheromatous degeneration of the branches of the pulmonary artery is frequently seen.

Hæmoptysis is a frequent result of valvular disease, whether from mere congestion or from pulmonary embolism.

The *liver*, which from the long-continued engorgement becomes much enlarged, is found to be in the state described as "myristicated," or like the mottled section of a nutmeg. The arrested flow of blood in the hepatic veins produces congestion of the portal circulation, as well as stagnation of bile in the ducts; the capillaries become choked, fatty degeneration of the cells takes place, and the secretion is checked. The appearance of the liver is altered in a most characteristic manner, the fatty degeneration of the circumference of the lobule giving it a white border, and this mingled with the red of the blocked hepatic veins and the yellow of the obstructed ducts, produces the nutmeg appearance. It is still a question whether production of new connective tissue may also take place. Several authors speak of the condition going on to cirrhosis, so that in course of time a structural disease of the liver is produced; but this is certainly an exception.

Some degree of jaundice is observable in most cases of chronic mitral disease, and when the hepatic congestion is continued, it becomes obvious. At the same time the liver is felt below the ribs; it is tender to the touch, and sometimes pulsates with each beat of the right ventricle (p. 263).

The *spleen* is found in cases of chronic valvular disease to be hard, dark, but very seldom swollen; sometimes it contains one or more fibrinous wedges ("infarcts") from emboli.

In cases of ulcerative endocarditis it is much enlarged and soft, as usual in pyrexia from any cause, and often contains congested wedges from the presence of septic emboli, which are afterwards found softened down into pyæmic abscesses.

The *kidneys* are large, hyperæmic, tough, and coarse-grained. In the end the congestion leads to a form of nephritis which, without producing the typical form of the large white kidney, may gradually cause shrinking, and thus simulate the appearance of the true cirrhotic kidney. Albuminuria with dark and scanty urine is the usual, hæmaturia the occasional effect of the congestion of chronic valvular disease.

The *stomach* is intensely injected, often with petechial spots or hæmorrhagic erosions, and its surface is covered with mucus. Dyspepsia, flatulence, and occasionally hæmatemesis, are the results.

The *pancreas* is indurated, but not obviously hyperæmic like the kidney and the spleen.

The *uterus* partakes in the universal venous congestion, so that its mucous membrane is red and velvet-like; and hæmorrhage sometimes takes place, as if from returning menstruation.

Cardiac dropsy.—A remarkable effect of long-continued venous congestion is that it produces transudation of serum by mechanical pressure in all loose areolar tissues and serous cavities,—both being alike, lymph-sacs lined with endothelium, and communicating by stomata with lymphatic vessels. The water, with the salts and albumen in solution, exudes through the walls of the vessels and collects wherever there are open spaces to receive it.

This is not a process of true exosmosis, for serum-albumen is a colloid; nor is it a process of inflammatory exudation, for there is no transmigration of leucocytes, nor is the transuded fluid coagulable. It is serum, not either pus or coagulable lymph. Such watery exudation or dropsy does not take place in the liver, spleen, kidneys, or brain, nor even in the spongy tissue of the lungs. By an exception which is not easy to explain satisfactorily, the result of the long-continued congestion of valvular disease in these viscera is, as above described, induration; or, in some cases, hæmorrhage. The globe of the eye is too completely filled to admit of dropsy, nor do we find hydrocephalus in cardiac cases, nor yet œdema of the muscles. In the brain such a result is prevented by the communication between the lymph-spaces of the brain and the vertebral canal, in the muscles by the lymphatic circulation, which is here probably at its best.

The chief seat, therefore, of cardiac as of other forms of dropsy is in the mesoblastic cavities lined with endothelium which form the areolæ of the connective tissue under the skin, and are expanded into the great lymph-sacs of the pleuro-peritoneal cavity. A considerable amount of fluid is sometimes found on one side or other of the chest. Less frequently there is notable ascites, and in most cases there is a moderate amount of fluid in the pericardium. In the tunica vaginalis the quantity must be very small, for one does not meet with passive hydrocele in cases of dropsy.

Cardiac dropsy is, as a rule, less extreme than that produced by some forms of renal and hepatic disease. Such excessive anasarca as goes with the large white kidney and the enormous ascites of cirrhosis of the liver are scarcely ever observed.

The mechanical character of cardiac dropsy is shown by its strict conformity to gravity. The face is occasionally swollen after lying down, but as a rule is free from œdema. The lower extremities are earliest affected, then the peritoneum, and one or both pleuræ. The abdominal walls are not œdematous as in renal dropsy, nor does the scrotum swell. There is often considerable œdema of the arms, which may vary from one side to the other as the patient lies in bed. Such œdema of one arm where there is no local cause of pressure on the axillary vein is very characteristic of cardiac dropsy.

Cyanosis.—In the pulmonary circulation we meet with a remarkable physiological result of the enforced stasis. This is dyspnœa, which is often the first sign of cardiac disease. The air finds free passage to the air-vesicles and pulmonary capillaries, but the blood is not sent through them with sufficient rapidity to ensure complete aëration. Hence ensues, first, rapid breathing, and next deep and laboured respiration. This is increased on exertion, like dyspnœa due to whatever cause, and is most observable when the patient ascends a hill or goes upstairs. The form of dyspnœa which compels the sufferer to maintain an upright attitude is very characteristic of cardiac disease, so much so that decided orthopnœa should always suggest disease of the heart.

Cyanosis as the result of dyspnœa is frequently present, but only to a moderate degree, and is usually masked by the anæmia and wasting which accompany heart disease. It is very rarely that we see the congested purple face and eyes and hands which are characteristic of severe capillary bronchitis and acute laryngeal obstruction. When such marked cyanosis is present, the cardiac lesion is almost always a congenital lesion of the right side.

Slight blueness, or (as the French writers call it) asphyxia, of the tip of the nose and the lobules of the ear may, however, often be seen, and sometimes of the chin and cheeks; that venous stasis is present is also denoted by the frequent occurrence in the more chronic forms of valvular disease of clubbing of the fingers and toes, with curving of the nails.

Symptoms due to arterial anæmia.—The anæmia of the arterial side of the systemic circulation produces the characteristic pallor of cardiac disease, a pallor which is most conspicuous in the face, and contrasts with the venous congestion of the cheeks and lips. It is also seen in the finger nails, which become yellowish instead of pink.

The brain is ill supplied with blood; syncope is often threatened, and sometimes proves fatal.

Other symptoms.—The patient suffering from heart disease, beside breathlessness, palpitation, flatulence, and dyspepsia, suffers greatly from disturbed and sleepless nights. As soon as he falls off, he starts up in fear of suffocation. The best position for the patient is the dorsal one with the head and shoulders raised, but this posture is unaccustomed, and therefore uncomfortable. A quiet regular circulation through the brain is necessary to cause sleep, but this is often impossible when a heart is beating too frequently and with various degrees of force. Moreover, the respiratory function of the bulb is blunted; for although it is said that the spinal system never sleeps, the statement is true only in a sense; during ordinary sleep the deep, slow breathing certainly indicates a change in the respiratory process. In disease of the heart a similar change, probably due to an altered vascular state of the centre of the vagus in the bulb, again and again produces suffocative symptoms and awakens the patient. The peculiar form of respiration described by Cheyne and Stokes is often present (cf. *supra*, vol. i, p. 1043).

The cardiac dyspnœa is further aggravated by congestion of the lungs; the patient coughs and begins to expectorate mucus tinged with blood, or sometimes enough to be called *hæmoptysis*. Dr L. E. Shaw, then Medical Registrar at Guy's Hospital, found, from an analysis made for the second edition of this work, of 262 adequately reported cases of valvular disease, that hæmoptysis was most frequent in those of mitral stenosis; much less so in those of aortic regurgitation, with or without obstruction; and least frequent in those of uncomplicated mitral regurgitation. Hæmoptysis occurred in 29 per cent. of the whole number of cases; in 45 per cent. of those with mitral stenosis, and in less than 20 per cent. of those without it. Or, put another way, of the total number of cases of hæmoptysis (76 out of the 262) 45, or more than 59 per cent., were cases of mitral stenosis.

It is a remarkable fact that while there is often evidence of local pneumonia (with pleurisy if it be near to the surface of the lung) round a patch of hæmorrhage, both at the bedside and in the deadhouse, it is very rare for the inflammation to spread and affect a whole lobe. Still more important is the fact that rupture of a vessel with hæmoptysis in the course of valvular disease is never followed by phthisis.

While bronchitis with occasional hæmorrhage is going on, we often find some dulness on percussion at the base of one or both lungs; as a rule, it is the lower lobe of the right lung which first becomes airless, of which no satisfactory explanation has yet been given. At this time the lung is in the condition above described as "splenization," and if blood has been expec-

torated there is probably effusion of blood in its tissue—so-called pulmonary apoplexy. But dulness at the base may depend on pleural effusion.

Hæmatemesis is a much less frequent symptom of valvular disease, and is seldom large in amount. The blood vomited is dark, venous, and more or less discoloured; sometimes digested, so as to resemble brown coffee-grounds.

The physical signs of valvular disease.—Palpation and percussion determine the size of the heart and the probable condition of its several cavities, and this furnishes us, as we have seen in the last chapter, with the best evidence we possess of their dilatation or hypertrophy. Dilatation and hypertrophy are most frequently the results of valvular lesion, and therefore aid in its diagnosis; but the direct evidence of the presence and nature of the affection of a valve depends upon auscultation.

Immediate auscultation is occasionally practised, and is as accurate, though less convenient, than other methods. The wooden stethoscope is most useful for listening through the clothing; but for critical observations it is desirable to employ the double as well as the single stethoscope, resting it upon the uncovered cardiac region, and taking care that it is not in contact with the patient's dress. It is also important, particularly in the case of mitral murmurs, to listen while the patient is lying down, as well as standing, and at rest, as well as after walking about the room.

The point of impulse is first ascertained by inspection, the patient lying on his back, or standing erect, or sitting up in bed and stooping somewhat forward. We then listen at that spot for the first sound, next at the junction of the third left costal cartilage with the sternum for the second sound, and afterwards trace any abnormal sound from the apex to the axilla and the back, or inwards to the ensiform cartilage, and from the base upwards towards each clavicle or down the sternum to the ensiform process.

The sounds of the heart may be modified in many ways, without there being what is technically called a murmur (*bruit anormal*).

(1) They may be *faint* or absent, due sometimes to the feeble action of the ventricle, sometimes to displacement of the heart, so that its sounds are not to be heard in a normal situation; sometimes to pericardial effusion or emphysema, bringing an ill-conducting medium between the organ and the ear. But often the cardiac sounds are feebly heard when the chest is perfectly healthy, owing to the thickness of its walls from great muscular development, or from subcutaneous fat.

The first sound of the heart is frequently deficient in loudness in cases of hypertrophy of the left ventricle; at the same time it is often prolonged and indistinct, so that it becomes impossible to say whether a "faint murmur," an "impure," or a "prolonged" first sound, or a "murmurish" sound, is the most appropriate description. A change in the opposite direction, to a higher, shorter, and clearer, *i. e.* more musical character, makes the first sound resemble the second, and is often observed in cases of cardiac dilatation.

(2) Increased *loudness* of the sounds, particularly the first sound, without other change, seldom points to organic disease. It is the result of violent action of the heart, and is constantly observed with the palpitation due to great exertion and temporary dyspnoea, or to that of mental excite-

ment. When this sign is present, the patient should be allowed to sit quiet or lie down for a few minutes before proceeding with auscultation.

The second sound is increased in loudness, and acquires a sharp, ringing, almost metallic character when the tension of the pulmonary or aortic cusps is much increased. This loud second sound ("accentuated," as it is called) is due either to high blood-pressure in the aorta compared with the left ventricle, or to the same high tension in the pulmonary artery compared with the right ventricle. Accordingly it is met with in chronic Bright's disease with high arterial tension, in mitral disease with low intraventricular tension, and in obstruction in the capillaries of the lung with increased tension in the pulmonary artery.

(3) If the pressure of blood in the two great vessels of exit from the heart is very different in degree, the pulmonary valves may close before or after those of the aorta, and thus the second sound becomes *reduplicated*. This is not nearly so common a thing as inexperienced auscultators suppose. The tendency is to call a healthy second sound "accentuated," and a loud, ringing one reduplicated; just as a timid auscultator will always find the first sound not quite clear at the apex, and the respiratory murmur somewhat harsh.

Reduplication of the first sound at the apex undoubtedly occurs, but in a well-marked and unmistakable form is decidedly rare. Apparently it is often confounded with a prolonged first sound, or with the first sound and a short presystolic murmur. There are considerable difficulties in explaining it by asynchronism in the contraction of the right and left ventricle; but the experimental results of the late Professor Roy, of Cambridge, and Prof. Adami, of Montreal, show that this is no impossibility. Clinically its most important significance is as a sign of mitral stenosis.

Intermission and irregularity of the cardiac sounds have been already discussed in connection with the pulse (*supra*, pp. 173-5).

*Cardiac murmurs.**—We now pass to the murmurs, *bruits* or abnormal sounds which are added to the first and second sounds. Sometimes they replace them entirely; sometimes one can more or less perfectly succeed in distinguishing the normal sound from the murmur which accompanies, precedes, or follows it.

The recognition of these murmurs was among the firstfruits of Laennec's discovery. He was naturally much struck by their occasional loudness and curious varieties of *quality*, and carefully described several of these varieties by comparing them to well-known noises. The commonest and most characteristic is the bellows murmur or *bruit de souffle*,† which exactly answers to the description of a continuous blowing sound, and is not unlike some forms of bronchial or amphoric breathing. Another frequent murmur has a higher pitch and more musical quality; it was named by Laennec the *bruit d'oboe*; and sometimes closely resembles a sibilant rhonchus. A harsh, grating, interrupted murmur he compared to the noise made by a coarse file scraping wood, and named *bruit de râpe*. Another quality was aptly disig-

* *Fr.* Bruits anormaux.—*Germ.* Herzgeräusche. It should be remembered that the French term *bruit* is applied to the natural sounds of the heart; so that what is called a "murmur" in English is always distinguished in French as a *bruit anormal* in general, or as a *bruit de souffle*, *bruit de râpe*, *bruit d'oboe*, etc., in particular.

† *Bruit de souffle* is literally a "blowing murmur;" the ordinary English equivalent, "bellows murmur," is a translation of the French *bruit de soufflet*, a phrase used by Laennec, and stated by Littré to be identical in meaning with the former.

nated by the term *bruit de scie*, or sawing murmur. This is usually to-and-fro or see-saw in rhythm, and is not at all unlike high-pitched and clear, tubular "in and out" respiration.

These comparisons, and many others which might be used, are chiefly of use for identifying a particular murmur. They tell us nothing or almost nothing with respect to the condition of the valves.

Nor is the *loudness* of a cardiac murmur of great importance; if distinctly heard, however faint, the message it conveys is the same as if it were loud, and sometimes an extremely loud murmur may be of little practical importance. A murmur may become faint or even disappear when the heart's pulsations are feeble, as in syncope; or may only become audible when its beats are rendered quicker and more forcible by movement or by excitement.

We must, moreover, remember that, in the healthy heart when palpitating from mental excitement, or when working under stress—as when a healthy young man runs a race, or a stout and elderly man goes too quickly up a staircase—a murmur may be developed which disappears after a short repose. It is only by repeated examination, and by the help of the pulse and other symptoms, that it is possible to decide between the diseased heart which under favourable conditions is without a bruit, and the healthy heart which, when overtaxed, produces one.

The *length* of the murmur is also of no great moment; but length renders it much more easy to be certain of, for a very short bruit may turn out to be a prolonged first or a reduplicated second sound.

The really important points to be observed concerning a cardiac murmur, as Skoda seems to have first definitely taught, are two—its *locality*, and its *rhythm*.

Locality.—It does not appear to be correct to say that any sound produced in the heart is "propagated in the direction of the blood-stream," for sonorous vibrations do not necessarily travel in the direction of the current of translation. Both direct and regurgitant murmurs are produced, not at the narrow passage, but beyond it, where the "fluid vein" is formed. Accordingly, a direct mitral bruit, having its origin in the left ventricle, is propagated to the chest wall, at the spot where the apex of the ventricle touches it; a regurgitant mitral bruit, being formed in the left auricle, is conducted by the auriculo-ventricular continuity to the apex, and, by the excellent conducting power of the ribs, along the axilla to the scapula. A direct or obstructive aortic (sigmoid) murmur, originating in the first segment of the aorta, is conducted by its walls to the part of the chest-wall nearest to it, and upwards towards the right clavicle; and a regurgitant aortic bruit, being formed just within the sigmoid valves, is usually best heard where the sounding-board formed by the sternum receives and conducts it. A direct pulmonary murmur, being formed in the pulmonary artery, is conducted, not by the stream of blood, but by the solid walls of that vessel, in the direction in which it passes, upwards and to the left. A regurgitant tricuspid murmur is not favourably situated for conduction, being in the right auricle, and is best heard either immediately over its place of origin or where the left auricular appendix comes in contact with the chest-wall, or along the sternum which transmits it downwards and to the left.

However explained by such physical conditions, we find as a clinical fact the following localisation of murmurs. Those produced at the *mitral*

valve are not transmitted directly to the surface of the chest unless they are unusually loud. The great depth at which this orifice lies explains the fact. They are conducted by the solid and uniform walls of the left ventricle to the point at which it is pressed against the chest wall in the systole. They are therefore apex-murmurs. Possibly owing to the conducting power of the fifth rib they are usually audible in the axilla and frequently along the wall of the chest as far as the angle of the left scapula. Only exceptionally can a mitral murmur be heard at the base of the heart or to the right of the sternum.

This applies to the most frequent of organic murmurs, that of mitral regurgitation. A temporary (*i. e.* in most cases a functional) apical systolic bruit is more confined to the place of cardiac impulse, it can seldom be followed into the axilla, and is never heard at the angle of the scapula.

The murmur of mitral obstruction is as a rule only audible at the apex-beat, and a little to the right of it towards the sternum.

Murmurs produced at the *aortic* orifice are audible directly over their seat of origin at the upper border of the third right costal cartilage at its junction with the sternum. If produced in the aorta by narrowing of its orifice, they are conducted by its walls to the chest wall in the second and first right intercostal spaces, *i. e.* upward and to the right from the base of the heart to the right clavicle. If produced in the left ventricle by leaking of the sigmoid valves, they are sometimes confined to the "aortic cartilage" and the third space immediately below it, but sometimes are conducted by the walls of the left ventricle to the apex, and more frequently down the sternum as far as the ensiform cartilage.

The far less frequent murmurs connected with the right side of the heart are localised as follows. One produced at the *tricuspid* orifice is audible over the sternum between the right and left fourth costal cartilages, and from that point downward to the ensiform cartilage and immediately neighbouring parts of the thorax. If audible, as it often is, to the left of the sternum, it does not reach so far as the point of impulse, where a mitral bruit is loudest.

A direct or obstructive murmur produced at the orifice of the *pulmonary* artery is audible at the junction of the third left costal cartilage with the sternum and in the second left intercostal space; it is carried upwards and to the left by the wall of the chest, so as to be audible in the second and first intercostal spaces. A very rare bruit, produced in the right ventricle by incompetence of the sigmoid valve, may be conducted a short distance down the sternum.

Time.—The rhythm of a cardiac murmur determines its connection with the contraction or dilatation of one of the chambers of the heart. There are two somewhat different systems of nomenclature, of which we will first state the older and then the more modern, leaving our readers to adopt which is thought preferable.

(1) A *systolic* murmur is one which coincides with the impulse and the first sound—that is to say, it accompanies the systole of the left and right ventricles. It often extends beyond the period of the normal first sound into the interval between it and the second, but it always begins at the moment of impulse.

A *diastolic* murmur is one which coincides with the second sound, the closure of the aortic and pulmonary valves, and the dilatation of the two ventricles. If it occurs after the second sound and separated from it by an

appreciable interval, although it still falls within the period of ventricular diastole which occupies the cardiac interval, yet it is for convenience named not diastolic, but post-diastolic—that is, occurring subsequently to the diastolic or second sound.

Lastly, if a murmur beginning in the cardiac interval is not separated from the first as well as from the second sound, but runs on until it meets the former, it is named a *presystolic* murmur.

This is the nomenclature formerly in general use and, as the writer believes, free from ambiguity and as minute as facts allow.

The only means we have of deciding the place of murmurs (or of tactile thrills) in the cardiac cycle is to refer them to the two ascertainable events: the closure of the auricular ventricular valves, marked by the first sound and the impulse; and the closure of the sigmoid valves, marked by the second sound. Systolic meant coincident with, presystolic immediately preceding, and post-systolic immediately following the first or systolic sound; and diastolic, post-diastolic, and prediastolic had a similar meaning with regard to the diastolic or second sound.

(2) Of late years it has become the practice to use the terms systolic and diastolic in a different, more physiological and etymological, less technical and less certain sense: to term a "systolic" murmur any abnormal sound occurring during the systole of the two ventricles, and to give the title "diastolic" to any murmur heard during the diastole of the ventricles. The objections to this nomenclature are as follows. Confining the term systolic to ventricular systole is after all a conventional though convenient limitation. Again, "diastole" includes two different successive events: the elastic expansion of the ventricle after the muscular contraction is over, and its passive continuance in an expanded state until the next cycle begins. Thirdly, we have no means of fixing the duration of the systole and diastole of the ventricles, nor of successive points of time in each, whereas we can absolutely refer murmurs to the first and second sounds, to the "interval" between them, or to the "pause" before the next cardiac cycle begins.

However, the nomenclature now more prevalent is to call any murmur coinciding with or immediately following the first sound "systolic," a murmur coincident with or immediately following the second sound *early diastolic*, one separated by an interval from the second sound and ceasing before the end of the "pause" *mid-diastolic*, and one beginning before the end of ventricular systole and running on to the first sound *late diastolic*, a term which therefore replaces and is synonymous with the original and more distinctive word "presystolic."

Tactile thrill.—The sonorous vibrations which are perceived by the ear are, when very loud, accompanied by a palpable vibration or tactile fremitus, just as we found that loud sonorous rhonchi can be felt as well as heard. This tactile vibration or cardiac thrill when well marked is extremely easy of detection, and gives a sensation to the hand which Laennec aptly compared to the tactile vocal fremitus which is felt when one lays one's hand upon the chest of a purring cat (*frémissement cataire*).

A tactile thrill, like an audible vibration, may be confined to the apex or base of the heart, and it may coincide with the systole, or with the diastole. It most frequently accompanies an apical and presystolic bruit, and often an aortic or aneurysmal bruit, but it may be felt along with any murmur; in fact, the tactile thrill and the audible sound are both alike

the impression on the senses of touch and hearing of the same physical vibrations of the chest walls.

Theory of cardiac murmurs.—Laennec's notion was that when one of the natural orifices was narrowed, the blood rustled in passing through, and it has been generally supposed that the rougher the surface the louder would be the noise. But comparisons taken from water running in contact with air do not apply to the passage of liquids in closed and completely filled tubes. It does not appear experimentally that roughness of surface produces audible vibrations, nor do we find clinically that roughening of the lining of an artery can be detected by the ear. Auditory vibrations in the vascular system are now believed to depend exclusively upon the formation of a *veine fluide*—that is to say, a narrow stream of liquid passing through an orifice or relatively smaller channel into a more spacious one. It can be imitated by filling a glass tube with water, and then injecting coloured liquid into it through a narrower tube or a constricted portion. The thin coloured stream passes into the wider space at first without mixing, then turns round and forms currents which eddy backwards upon the walls of the larger tube and set up in these walls vibrations which, if ample enough, may be heard by the ear and felt by the finger. Accordingly every cardiac or vascular murmur may be probably referred to the same physical cause.

The production of a *veine fluide* in pipes containing air or water, and the audible vibrations which result, is a well-established fact in physics; but the merit of applying it to explain cardiac murmurs, in opposition to the theory of Laennec and his successors, belongs to the late Sir Dominic Corrigan of Dublin, who, writing in 1829, says, "When an artery is pressed upon, the motion of the blood in the artery immediately beyond the constricted part is no longer as before. A small stream is now rushing from a narrow orifice into a wider tube, and continuing its way through the surrounding fluid. The rushing of the fluid is combined with a trembling of the artery, and the sensation to the sense of hearing is the *bruit de souffle*."

The conditions for the production of a fluid vein are demonstrable in almost every case of valvular disease accompanied with a murmur. If one of the cardiac orifices is contracted, the blood is forced through it into a wider space beyond; if one of the valves is rendered inefficient, the blood rushes through the aperture, under the pressure which closes the valve, into the space which it last quitted. In cases of dilatation of the right or left ventricle, the valves, although perfect, no longer suffice to close the enlarged orifice; leaking occurs, with the formation of a fluid vein, and a murmur is again the result.

It is, however, necessary in order that a *veine fluide* may be audible for the blood (like any other fluid) to be driven through a narrow into a wider space at a certain degree of velocity; otherwise, the mass being the same, the momentum of the vibrations excited will not be enough to reach the ear or the hand. Accordingly we find that the loudest sounds are those produced by a hypertrophied ventricle.

A direct sigmoid and a regurgitant mitral murmur are both the result of the contraction of the left ventricle. A diastolic sigmoid murmur is produced by the powerful elastic recoil of the distended aorta. A pre-systolic murmur is due to the contraction of a hypertrophied left auricle.

The chief difficulties in the practical application of this theory are as follows. In some cases of tricuspid regurgitation due to emphysema, we do not hear the systolic murmur we should expect; while a murmur is

sometimes audible at the apex in cases of hypertrophy from Bright's disease when there is no evidence of regurgitation. Moreover, murmurs heard in the great vessels are not easily brought under the same hypothesis of a fluid vein. The ordinary systolic basic bruit audible in the region of the pulmonary artery in cases of marked anæmia is certainly independent of contraction of the orifice or lesion of the sigmoid valve; and it is rather assumed than proved that, the trunk of the artery being temporarily dilated, the orifice which leads to it becomes relatively narrower, and therefore capable of producing a fluid vein. The well-known *bruit de diable* in the internal jugular vein must be explained by temporary dilatation of part of the vessel and relative narrowness of the part behind. The arterial murmurs which the stethoscope detects in anæmic patients probably depend upon compression of the vessel by the instrument, causing a constriction and a temporary fluid vein at that point.

The systolic murmur audible over an aneurysm or in the placenta of a pregnant uterus may be referred to the same cause without straining the anatomical facts. The difficulty is rather to explain why a murmur is so often absent in cases of aneurysm than to explain its presence as the result of a fluid vein.

No other theory of cardiac and vascular murmurs is supported by physical facts, or is nearly so applicable to clinical experience; but it must be confessed that we sometimes meet with difficulties in its application. It is possible that though roughness of the lining surface of a vessel is incapable of producing sonorous vibrations, it may reinforce those produced by a fluid vein; and we still seem to need some further physical explanation of the undoubted fact that in cases of anæmia murmurs are readily produced and are unusually loud.

In any case it appears to be certain that the vibrations which reach our ear are those of the walls of the vessel, and not those of the contained blood; that they start, not at the seat of obstruction or of leakage, but in the cavity immediately beyond; and that they are transmitted not by the stream of blood, and certainly not in the direction in which it flows more than in the opposite direction, but by the wall of the vessel or the chamber of the heart, which is thrown into vibration.

Having thus described the general pathology, symptoms, and physical signs of valvular disease, we will now notice the peculiar clinical symptoms which belong to each lesion.

1. *Aortic (sigmoid) stenosis with obstruction*.—This as an uncomplicated lesion is one of the most rare. It is most often met with along with sigmoid incompetence. It may occur under two conditions: as a result of rheumatic fever in youth, when, if not complicated by regurgitation, it is probably most often recovered from; and secondly, as a result of atheroma of the sigmoid valves in later life, when its import is grave. The origin of the chronic arteritis is in most cases the wear and tear of time and of laborious employment, particularly with the arms; but in younger patients—adults between twenty-five and forty-five—it is occasionally of syphilitic origin. (See 'Path. Trans.,' vol. xlvii, p. 26.)

The physical sign of narrowing of the sigmoid orifice is a systolic basal murmur propagated towards the right clavicle, sometimes accompanied by a tactile thrill.

The pulse is small, and, by an exception to most cases of valvular disease, slow: *i. e.* it is so in most cases, but the writer is inclined to ascribe the slow pulse, not to the aortic stenosis, since he has never found it in cases of the same stenosis from rheumatic endocarditis in children or young adults, but rather to the atheromatous inelastic state of the aorta; indeed a slow pulse may often be found in old men with atheroma, but no obstructive murmur. In some cases the pulse is anacrotic, and in others trigeminal, but neither is characteristic. The left ventricle is usually somewhat hypertrophied, but is not apt to dilate.

The general symptoms are not severe, but sudden and fatal syncope sometimes occurs;* and atheroma constricting the aortic orifice is one of the lesions found in fatal cases of angina pectoris, probably from atheroma narrowing the orifice of the coronary arteries. The difficulty of diagnosis is between structural disease and anæmia as a cause of a basal systolic murmur.

2. *Aortic (sigmoid) incompetence with regurgitation.*—This also occurs in two conditions: as a result of rheumatism in young adults, and as a result of atheroma in elderly people. It is rare in children, and much less common in women than in men. As with other cases of valvular disease, the origin of the lesion is most often due to rheumatic endocarditis or to atheromatous arteritis; and, as with sigmoid stenosis, the latter is the more frequent and the more serious. In some cases the arteritis is of syphilitic origin (see Dr Mott's article in the sixth volume of 'Allbutt's System,' p. 303), in others it is due to wear and tear, to gout, alcohol, lead, and the chronic Bright's disease they lead to; and occasionally the valvular lesion is due to sudden strain, raising the aortic blood-pressure and rupturing an already diseased valve. Whether rheumatic or atheromatous, it is commonly associated with aortic obstruction, and in the latter case with dilatation, or fusiform aneurysm of the aortic arch. It is, however, often met with alone.

Although the signs just mentioned denote incompetency of the aortic valves, it does not follow that they are themselves diseased, for their want of closure may arise from dilatation of the ascending aorta at its root. A yielding of the sinuses of Valsalva just above the aortic valves has been proved to interfere with their perfect action. See Dr Pitt's paper ('Path. Trans.,' 1898, and 'Brit. Med. Journ.,' vol. i, p. 369).

The physical sign is a basal diastolic bruit replacing and continuing the second sound, often very audible at the ensiform cartilage, and occasionally at the apex.† It is sometimes musical (*bruit d'oboe*), but more often sawing in character (*bruit de scie*), and is one of the very long and extremely loud murmurs. This aortic regurgitant murmur is the one which may occasionally be heard without a stethoscope by the ear at a distance from the patient's chest.

* Sir R. D. Powell has recorded a striking case of sudden death from this lesion in a schoolboy, while running.

† The second sound of the heart was ascribed to tension of the aortic valves by Sir Robert Carswell, 1831. In 1832 Dr Billing, in a paper at the Hunterian Society, published in the 'Medico-Chir. Review,' April 1st, 1833, stated for the first time in clear language that the first sound is caused by the tension produced in shutting the auriculo-ventricular valves; and the second sound is caused by the tension produced in the shutting of the sigmoid valves. Rouanet and Hodgkin had come to the same conclusion in the year 1832 (*Analyse des bruits du cœur*, August, 1832). In the same year (1832) Corrigan discovered the mechanism and diagnosis of aortic regurgitation.

The rate of the pulse is increased, the artery is large and compressible, the tension low. In systole it fills rapidly, and rapidly subsides again. It is characteristically the quick, short pulse—*pulsus celer*. In systole it is the strong, forcible pulse, in opposition to the weak pulse which is difficult to feel. It is, however, a weak pulse in diastole, *i. e.* weak in resistance, soft, compressible—the opposite of the hard, cord-like pulse. It exhibits the extreme of difference between systole and diastole, the greatest amplitude of arterial pulsation—the opposite of the characteristic pulse of chronic Bright's disease, in which there is little increase of calibre in systole, and little diminution in diastole.

This very striking pulse of sigmoid regurgitation was first recognised as its sign by Corrigan, and is still often named after him. It has also been described as a "locomotive," a "collapsing," and a "water-hammer" pulse.* The strong pulsation in the arteries is due to the reflux into the left ventricle during diastole. The elastic arteries are expanded with the ventricular systole, but instead of the column of blood being sustained by the resistance of the aortic valves, a portion of the contents of the ventricle which was pumped into the arteries returns as soon as the contraction of the ventricle relaxes, so that the blood-pressure in the arteries is unsustained and falls low between the pulses. The characters of the pulse are made more obvious by raising the patient's arm, since gravity then assists the rapidity of the collapse in diastole, and it is probable that not only in the aorta but in its branches for some distance a true reflex occurs; that the flow of blood is not only slackened or brought to a standstill at the moment of collapse, but actually moves back towards the heart.

If this is a fact, it explains a sign which is not frequent, but undoubtedly may occur, namely, the presence of a diastolic murmur over the femoral or brachial or carotid arteries in these cases of aortic regurgitation. It is improbable that any cardiac bruit can be transmitted by the walls of the arteries for more than a very short distance from its place of origin. A systolic arterial bruit appears to be always caused by compression of the vessel by a stethoscope producing a temporary stenosis; and thus a fluid vein and a diastolic bruit may be explained by the same temporary contraction, if we suppose that in the case in which it is heard there is an actual reflux in diastole. It is remarkable that when an anginal attack comes on in the course of sigmoid regurgitation, the radial pulse loses its full and jerking character, and becomes, to use Douglas Powell's words, "a small, hard, pulsating thread" ('Lumleian Lectures on Treatment of Heart Disease,' p. 29).

Beside the throb and the collapse, which are so striking to the touch, the eye detects the same exaggerated pulsation in the great vessels. As the patient sits before us the throbbing carotids almost tell the nature of his disease before we feel the pulse or use the stethoscope. The same visible pulsation is seen in the brachial artery at the bend of the elbow. It may enable one to diagnose aortic incompetence when examining the retina, from the obvious pulsation of the arteries seen with the ophthalmoscope. It extends to the smaller arteries and even the capillaries, so that we can sometimes observe a visible flush with each pulsation in the nails of the hand and upon the skin of the forehead, particularly if we cause relaxation of the vaso-motor nerves and injection of the capillaries by gently

* The comparison is to the suddenness of the fall of a column of water *in vacuo*.

rubbing the skin. Or, if we compress the lower lip with a microscopic slip of glass, we see the pulsation of the blood through it as soon as we relax the pressure.

The characteristic tracing of aortic incompetence is marked by a nearly perpendicular up-stroke, a sharp, and often tall, percussion-wave, a very narrow (*i. e.* short) tidal wave, and a notch, small and low down, on the descending line, or sometimes almost absent.

The regurgitant aortic murmur is most frequently combined with an obstructive one. The pulse in these cases retains the characters just described as those of the former lesion. There is audible at the base a very characteristic to-and-from—that is systolic and diastolic—double murmur, one of the loudest, least variable, and easiest to recognise of all physical signs; of the two murmurs the obstructive or systolic is usually the softer and shorter, the regurgitative or diastolic the longer, louder, and higher pitched. It is often audible in every part of the chest, and can sometimes be heard by the ear at a short distance from the chest, or even, it is said, by a person standing at the foot of the patient's bed.

The systolic bruit is often carried a considerable distance along the vessels, and sometimes the diastolic also. In the femoral artery, for example, a double bruit may sometimes be heard. If not, it may frequently be produced by using pressure with the stethoscope.

Austin Flint, of Philadelphia, observed that a presystolic murmur may sometimes be heard in cases of aortic regurgitation. He believed it to be due to the vibration of the mitral segments even when the valve is inactive, owing to the over-engorgement of the ventricle from the reflux of blood into it from the aorta.

The symptoms of sigmoid regurgitation (and of regurgitation with obstruction) are those of arterial anæmia rather than of venous congestion; for so long as the mitral valve is competent there is but little increased pressure in the left auricle and pulmonary veins or in the right side of the heart. Accordingly in uncomplicated cases we find the patient pale but without dropsy, albuminuria, or jaundice. He complains of headache, and is liable to faintness and giddiness, he shows dyspnœa on exertion and marked orthopnœa; but while tranquil and under favourable conditions of nutrition he suffers comparatively little. This depends upon what is known as *compensation*.

The ventricle is habitually over-full during diastole, receiving the reflux of blood from the aorta, as well as the natural onward stream through the mitral orifice. It also has the obstruction at the aortic orifice to overcome in systole. This habitual increase of pressure produces dilatation, and also hypertrophy. A moderate amount of the former change is probably beneficial in enabling the heart to accommodate an increased quantity of blood, and by help of the latter the force of the ventricular contraction is increased, so that as the result the blood-pressure in the arteries is kept up, just as by a more vigorous use of the handle we can obtain a free supply of water from a pump with a damaged valve. The stream of blood through the aortic orifice is diminished in calibre, but its velocity is increased, and thus the amount discharged in a given period of time may be as large as it was before the valve was injured. The compensation is, however, liable to come to an end in more ways than one; first, owing to insufficient supply of food, indigestion, anæmia, or other causes independent of cardiac lesions which interfere with muscular nutrition. The left ventricle does not perhaps

suffer more than the muscles of limbs ; but unlike them it is necessary to the organism, it can never rest. Hence the struggle is a difficult one, and though often successful for many years, increase of the valvular incompetence, or impairment of nutrition, or excessive calls upon the energy of the damaged organ may at any time break down the compensation ; and then comes what appears to be the natural end of abnormal hypertrophy, wasting and degeneration.

It has been supposed that the increased blood-pressure in the left ventricle during diastole which is the result of aortic regurgitation, would lead to dilatation rather than hypertrophy, and that the increased blood-pressure in the same cavity during systole in cases of aortic obstruction, would lead to hypertrophy rather than to dilatation. This is probably true ; but, in the first place, when passive dilatation has taken place, the ventricle will at the end of the cardiac pause be over-full of blood, for it will contain the contents of the left auricle, and the amount which has leaked back into it from the aorta. It will thus have more than its due volume of blood to expel with the next systole, and will thereby be stimulated to hypertrophy. On the other hand, we see from a study of the effects of obstruction in other hollow canals, that dilatation as well as hypertrophy may be the result. When the pylorus is obstructed we find the stomach dilated as well as hypertrophied ; above the stricture of the œsophagus or of the large intestine, there is a dilated pouch with thickened walls ; and the bladder in cases of stricture is often found dilated as well as hypertrophied. Moreover, in the case of the heart itself we have an instructive example of the effects of obstruction without regurgitation in cases of chronic Bright's disease. The precise nature of the obstruction in the systemic circulation is, we shall see, still a matter of controversy, but of its existence there is no question. Here, then, we have increased blood-pressure in the aorta, giving the ventricle increased work to empty itself in systole ; but the semilunar valves are perfect, and the ventricle receives only its due supply of blood from the auricle during diastole. The result is, as we should expect, hypertrophy of the ventricle walls, and this may continue for an indefinite time ; but in certain cases, and perhaps in the majority, if the process is continued long enough, the walls begin to yield, and instead of, or in addition to hypertrophy, we find dilatation of the ventricle after death, as well as its appropriate symptoms during the patient's life.

In cases of aortic incompetence the interval between the ventricular contraction and the radial pulse is, as a rule, lengthened, though there are exceptions to the rule ; and Dr Chapman, of Hereford, has given evidence by comparison of cardiographic and sphygmographic tracings, that this lengthening of the cardio-radial interval may be regarded as an index of good compensation (' Brit. Med. Journ.,' Feb. 16th, 1891). The prolongation appears to be partly due to slower contraction of the hypertrophied ventricle, partly to a slower pulse-wave in the arteries.

It has been thought that much of the compensatory hypertrophy we have been discussing does not bring increased power to the ventricle, because it is not all true muscular hypertrophy, but is accompanied by fibrous or fatty degeneration. This, however, seems to be very doubtful ; the most typical cases of fatty degeneration do not occur with hypertrophy of the heart, or with any form of primary cardiac disease, and fibrous degeneration, as we have seen, is also an independent process ; hypertrophy of the heart is a true muscular hypertrophy, and the muscle is often found in a

perfectly healthy condition. When this is not the case, what we do see is that the muscle is pale, soft, and flabby—looking, in fact, like the skeletal muscles in a patient who has long lain ill, but not showing either fibrous or fatty degeneration. Except in cases of atheroma of the coronary arteries, it is probable that the nutrition of the cardiac muscle depends almost entirely on that of the body generally.

The importance of the condition of the ventricle is obvious from what has been stated, and of late years there has been a disposition to make light of valvular lesions, in comparison with the state of the walls of the heart. But this might easily lead to practical mistakes. A valvular lesion is always a serious thing; for it is permanent, and, however good compensation there may be, is always liable to produce its full effects should the compensation at any time fail. On the other hand, hypertrophy or dilatation and fatty degeneration of the heart are comparatively seldom primary conditions, and they derive their importance in most cases from the fact of some antecedent lesion in the valves or elsewhere.

The clinical recognition of hypertrophy and dilatation of the left ventricle, as well as of the other cardiac chambers, has been already discussed (pp. 200—203).

At the mitral orifice we have again the two conditions of obstruction, which, although frequently combined, are also often met with separately.

3. *Mitral stenosis with obstruction* most often begins in one or more attacks of rheumatic endocarditis, followed by gradual thickening, sclerosis, and contraction of the edges of the opening. The semilunar orifice of the natural valve is converted into a short, straight, narrow slit, to which the not inappropriate name of the “button-hole mitral” has been given; or the two curtains may cohere, and, yielding to the pressure of blood from the hypertrophied auricle at the end of the cardiac diastole, may project in the form of a fibrous cone into the ventricle. This is called the “funnel-shaped mitral.”

The degree of obstruction may be roughly measured by the number of fingers which the orifice will admit, or more accurately by inserting graduated cones as far as they will go, or, again, by measuring the edge of the orifice after it has been cut open.

Sometimes a precisely similar contraction of the mitral orifice is found in persons who have never suffered from rheumatism—nor from chorea, scarlet fever, or other probable cause of acute endocarditis—and the whole process seems to be insidious in origin and gradual in progress. A certain proportion of these cases occur in later life, most often in women, and are associated with chronic Bright’s disease and atheromatous arteries. They appear, therefore, to belong to the group of degenerative changes which are more often associated with disease of the aortic valves; and this view is confirmed by our not infrequently finding calcareous nodules in the thickened fibrous tissue of the mitral curtains. Other cases, again, are met with in young adults and even in children with no history of rheumatic inflammation, nor even of chorea or scarlatina. Many of these are probably, notwithstanding this, rheumatic in origin, though the synovitis and the fever were, as is usual in children, so slight as to have been forgotten or overlooked. It is possible that other cases date from before birth. This was the belief of the late Dr Peacock, and is maintained by Douglas Powell, and it is difficult to disprove; but the extreme rarity of mitral stenosis in

early childhood makes it improbable that congenital stenosis of the mitral valve is also rare.

Dr Goodhart found in the post-mortem room of Guy's Hospital, mitral contraction in about a fourth of 192 cases of Bright's disease with granular kidney. Dr Pitt from experience of the autopsies at the same hospital came to the conclusion that most cases of contracting sclerosis of the mitral orifice in women which are not of rheumatic origin are associated with various uterine disorders, which set up cystitis and consecutive Bright's disease.

Certainly the connection between mitral stenosis and arterio-capillary fibrosis with atheroma and granular kidneys seems to be closer than that of the same cardiac lesion with chlorosis and other forms of anæmia; although the latter relation is maintained by some experienced physicians both in France and England.

Many French physicians have indeed maintained that "pure," *i. e.* uncomplicated mitral stenosis, and particularly the funnel-shaped orifice, is not the result of rheumatic endocarditis, and believe that its real cause is tuberculosis. This is quite contrary to the experience of the writer, whether in the wards or the deadhouse.

Mitral obstruction is common in children from five years old upwards, and in adults is decidedly more frequently met with in women than in men. In nearly 200 cases of marked mitral stenosis examined after death at Guy's Hospital during 1886—1895, Dr Samways found a history of rheumatism recorded in 120. There were 107 cases of mitral contraction in women to 89 in men.

The result of the narrowing of the mitral orifice is to diminish the supply of blood to the left ventricle, and to increase the blood-pressure in the left auricle. The latter hypertrophies in response to the call, and by a more energetic contraction succeeds in emptying its contents into the ventricle before the systole of the latter cavity begins. The process of dilatation, however, very soon accompanies that of hypertrophy, and since there are no valves guarding the entrance of the pulmonary veins the increased pressure in the auricle soon begins to tell upon the pulmonary circulation. Ultimately the right side of the heart and the systemic venous circulation may in time become affected. But this is a slow process; and, if the mitral obstruction be uncomplicated with regurgitation, only takes place, if at all, after many years.

The left ventricle neither hypertrophies nor dilates, and it would be surprising if it did. Some authors affirm that it becomes smaller instead of larger, but it would be difficult to prove that there is absolute atrophy; no doubt it looks smaller by comparison with the large auricles and right ventricle. As the heart of aortic disease is likened for its size to that of an ox, so the heart of mitral disease has been compared with that of a turtle from its breadth.

The physical signs of mitral obstruction are of great clinical and physiological interest.

The lesion was well known to Laennec, and he learnt to recognise a rasping murmur—or rather, as he expressed it, "a modification of the natural sound of the heart into a sound like that of a file"—which is sometimes due to a narrowing of one of the orifices of the heart, and is then much more loud than when it is caused "by the heart being too full of blood." In the former case he noticed that one could sometimes feel a thrill, and also that

the altered sound of the heart is much prolonged (1819). Latham, of St Bartholomew's Hospital, believed that practically all mitral murmurs were systolic in rhythm and regurgitant in origin.* Hughes, of Guy's Hospital, writing in the same year 1845 states the different mechanism of obstructive and regurgitant murmurs and the methods of diagnosing each, clearly and accurately; but he also failed to recognise the application of his rules to mitral obstruction. Those who more boldly carried out what they were taught naturally arrived at the conclusion that mitral stenosis would produce an obstructive murmur, apical in position and diastolic in rhythm; but this was seldom or never recognised by the bedside, and seems to have been admitted for the sake of symmetry. In practice, physicians agreed with Latham, Addison, and Walshe in recognising only one mitral murmur, systolic in rhythm, and produced by either obstruction or regurgitation. An apex systolic murmur denoted mitral disease, but whether the valve would be found too small or too large was quite uncertain. In 1845 Skoda stated that with narrowing of the mitral valve the second sound is replaced by a long murmur; but in 1843 Fauvel had shown that the characteristic sign of mitral stenosis is an apical murmur which is neither systolic nor diastolic, but, adopting a phrase previously used in another sense by Gendrin, "presystolic" in rhythm—that is to say, it is not separated from the first sound by any interval, but, beginning during the cardiac pause, it runs up to and unites with the first sound and the impulse. It therefore occurs during the ventricular diastole, but at the end of that period, and coincides with the auricular systole which immediately precedes that of the ventricle.

Apart from its characteristic rhythm, the presystolic mitral murmur is remarkably loud, harsh, and often grinding in quality, rarely blowing or musical. Moreover it becomes louder as it goes on, but not, as has been stated, of higher pitch, and is suddenly cut short at its loudest by the first sound and the impulse. Again, its locality is very limited, so that one is surprised to hear so very loud a sound, only audible in the immediate neighbourhood of the apex-beat. Lastly, it is accompanied by a sharp, loud second sound at the base, and not infrequently by the tactile fremitus which we have seen that Laennec recognised and named *frémissement cataire*.† This apical cardiac thrill, like the murmur which it accompanies, is presystolic in rhythm and ends in the impulse.

The cause of the murmur is probably that the hypertrophied left auricle, in forcing the last portion of its contents through the narrowed orifice into the ventricle, furnishes a fluid vein of sufficient velocity to produce audible and tactile vibrations of the ventricular walls, which are heard and felt at the point where it touches the surface of the chest.

The above explanation of the presystolic murmur is that first offered by Fauvel in 1843, and ably advocated by Traube and by Gairdner in 1861 ('Edin. Med. Journ.,' vol. vii, p. 438), and is generally accepted in this country and abroad. It was defended by Dr Fagge in his paper in the 'Guy's Reports' for 1871, and also in his article in 'Reynolds' System of Medicine.' It is, however, right to mention that another hypothesis, which referred the murmur to the ventricular systole, has been supported by Dr

* "The cases are so rare in which either the diastolic murmur alone, or the diastolic and systolic murmurs together, can be fairly imputed to the mitral valve, that they are a sort of clinical curiosity" ('Lect.,' vol. i, p. 38, 1845).

† "Quand (l'orifice rétrécie) est à gauche, on sent quelquefois à la main un frémissement analogue à celui qui accompagne le murmure de satisfaction que font entendre les chats lorsqu'on leur passe le main sur le dos" (Laennec, 'Ausc. Méd.,' tom. ii, § 634).

Ormerod (1864), by Barclay (1872), by F. C. Turner in the 'London Hospital Reports' for 1876, and by Dr Dickinson (1887) with characteristic skill in his collected papers, p. 217 (1896). The decision rests partly on physiological considerations, and partly on one's own auditory perceptions; and on both grounds the writer adopts the conclusions of Fauvel, Traube, and Gairdner.

The explanation of the loud, sharp, first sound which closes the presystolic murmur has been also a subject of controversy. Dr Sansom seems to have offered the best account of this fact in referring the clear first sound to the tricuspid valves closing under the tension of a hypertrophied right ventricle.

Whatever its physiological explanation, no one doubts that a presystolic murmur at the apex indicates mitral contraction, the only exception being the very rare condition associated with Flint's name (p. 252). The recognition of a presystolic murmur as a sign of mitral stenosis is, as shown by Dr Fagge in the paper above mentioned, undoubtedly due to Fauvel, whose article appeared in the 'Archives Générales' in 1843.

No cardiac murmur is more easy to recognise than the presystolic bruit of mitral contraction when the characters above enumerated are present. Nevertheless the diagnosis of this lesion is sometimes difficult, and for the following reasons:

First, it is often accompanied by mitral regurgitation; and a presystolic bruit ending, not in a first sound and interval, but in a systolic murmur, becomes difficult of detection, and is often taken for a long regurgitant bruit alone. One must try to recognise the difference of quality of the former and latter parts of this continuous murmur and to fix its precise rhythm by placing the finger upon the apex-beat. Another plan is first to apply the solid stethoscope so lightly that we only feel the impulse without hearing the sounds, and then to increase the pressure until it conducts the murmurs, which then fall into their proper place, the one before and the other after the impulse. One must never attempt to get the time by feeling the radial pulse, for this may be delayed. The carotid may be used for the purpose more safely; but the direct palpation of the heart by the finger or the ear, as just recommended, is the safest and easiest plan.

Secondly, the presystolic murmur is often absent in cases of undoubted mitral stenosis, and where it has been heard distinctly a short time before. On a single occasion the lesion of the valve may therefore pardonably be overlooked. Sometimes, however, one can feel a presystolic thrill when one cannot hear a presystolic bruit, or a ringing second sound may help us, or an apical reduplication of the first sound; and our conjecture will be all the more probable if the patient is a female, if hæmoptysis has occurred, and if there is no history of rheumatic fever.

In not a few cases of mitral stenosis, instead of the characteristic, rough, long, and ingravescent or *crescendo* presystolic murmur, a sound is heard which follows the second after a short interval and ceases before the first.* It is audible at the apex and is *diastolic* in rhythm, *i. e.* it occurs during the diastole of the ventricle. But it differs from the presystolic murmur in that it does not run up to the first sound and impulse; beginning after the second (or diastolic) sound it occupies the period of ventricular pause, and ceases before the first sound, from which it is separated

* A recent valuable monograph by Dr. Brockbank, of Manchester, has appeared ('The Murmurs of Mitral Disease,' 1899) in which he upholds the early ventricular-systolic hypothesis by some interesting experiments and some ingenious arguments.

by an appreciable interval. Its rhythm may be more precisely defined as either post-diastolic or mid-diastolic (cf. p. 247).*

The probable explanation is that in these cases the left auricle, being well hypertrophied, succeeds in emptying its contents through its narrowed outlet before the ventricle contracts, so that, as in health, there is a short interval between the auricular and ventricular contraction. When the bruit is presystolic, the auricle does not succeed in expelling its contents before the ventricular contraction at once reverses the direction of the inflowing blood-stream, and ends the obstructive murmur by the first sound or by a regurgitant systolic murmur.†

Whatever is the explanation, this diastolic apex-bruit, when associated with other signs of mitral stenosis, is a sure evidence of that lesion. The difficulty is to distinguish it from a diastolic regurgitant bruit from incompetence of the sigmoid valves, when the latter is, exceptionally, most audible at the apex. The other characters of the aortic bruit, which is usually less harsh and more blowing or musical, the evidence of dilatation and hypertrophy of the left ventricle, and the peculiar character of the pulse, are the best marks of distinction from the murmur of mitral obstruction, which is long, rumbling (*i. e.* unequal), low in pitch and very local.

Another sign of mitral stenosis is reduplication of the sounds of the heart. When the first sound is double, it is possible that the two ventricles are not working quite synchronously, and so the mitral and tricuspid curtains do not become tense at exactly the same time and are heard separately. More often perhaps what is called a double first sound by some auscultators would be described by others as a prolonged first sound, or as a first sound with a short and faint murmur following it.

When the second sound is double, it is frequently due to the diminished blood-pressure in the aorta and the increased pressure in the pulmonary artery causing the right sigmoid valve to close a little earlier than the left, and so the second sound, usually heard single, is split into its two constituents.‡ But in many cases it is probable that the later of the two second sounds described is really a short diastolic (mid-diastolic) bruit; and this is particularly likely to be true when the supposed doubling of the second

* It is sometimes called "*post-diastolic*," because it is heard not *with* or replacing the second (diastolic) sound, but *after* it. It is well to note that the term "*diastolic*" may be applied in three senses: to the period of diastole of the ventricle, to the diastole of the auricle, or to the second sound.

Hope, writing in 1832, remarked, "When the mitral valve is contracted, a murmur accompanies and sometimes entirely supersedes the second sound, being occasioned by the obstructed passage of the blood from the auricle into the ventricle during the diastole of the latter."

† Dr Humphry Rolleston has published a valuable criticism of this explanation, and his objections are not without force ('*Journ. Phys.*,' viii, p. 253); but those against any view which ascribes the diastolic apex-murmur to contraction of the ventricle are still more cogent. It is very difficult to say beforehand what derangement of mechanism may take place when the delicately-adjusted normal relations of blood-pressure in the cavities of the heart are altered. It certainly appeared unlikely that the right ventricle should beat asynchronously with the left, but Roy's observations establish this as a fact. That the hypertrophied left auricle drives the blood through the narrowed orifice with increased velocity, that the auricular systole will be prolonged in time, and that it must precede and be ended by the ventricular systole, seems to be certain.

‡ Sir George Johnson suggested that one of these sounds may be not valvular, but due to the auricular systole. Dr Sansom thinks that it may occur from a tension of the mitral valve at the time when the blood is thrown back during the diastole of the ventricle, and that therefore it is not a normal second sound. Dr Theodore Fisher has published an interesting paper on the *Bruit de galop* or three-fold sound in the '*American Journal of Medical Sciences*' for September, 1896.

sound is most audible at the apex. This double sound, or, combined with the first, this triple sound of the heart, is sometimes heard under other conditions of impediment to the circulation, but it is most common in the case of mitral stenosis. It is called in France by Bouillaud's name, *bruit de rappel*, and is like the sound of a hammer rebounding on the anvil.

Lastly, among the numerous other signs of mitral obstruction is a weakening of the first sound of the heart, so that it is scarcely noticed in contrast with the sharp and loud second sound. This seems to depend on the thickening of the mitral curtains no longer allowing them to become lax with diastole and tense with systole of the ventricle. Thus the valvular part of the first sound is abolished, and only the muscular bruit remains.

When the mitral valve is narrowed, we often hear, combined with its proper diastolic murmur, one of systolic rhythm, and sometimes this is heard when there is no other murmur audible. No doubt this systolic apical bruit is usually the sign of mitral regurgitation in addition to obstruction, for there is every reason to expect that a valve contracted by fibrous thickening will often fail to close the orifice when the ventricle contracts. Indeed, when in a woman of middle age and without a history of rheumatism we have cardiac symptoms and hear only a systolic bruit at the apex, we may suspect mitral obstruction and, after a time, may hear its characteristic presystolic bruit. But in other cases it is probable, as Dr Goodhart and Dr Sansom teach, that the systolic murmur heard in cases of mitral stenosis is not mitral but tricuspid, and depends on secondary dilatation of the right side of the heart and incompetence of the tricuspid valve. In seventy cases of mitral constriction observed after death in Guy's Hospital, Dr Samways found that a systolic bruit had been heard in forty.

The question still remains whether any other condition than mitral stenosis can give rise to the characteristic presystolic murmur; in other words, is the connection between this physical sign during life and a contracted state of the mitral orifice after death an absolute one? It appears that this is no exception to the rule that in medicine no rule is absolute, and no sign or symptom "pathognomonic." For there are the rare cases of aortic regurgitation, in which "Flint's murmur" is heard during life, and the mitral orifice is found uncontracted after death (cf. p. 252). The writer once met with a marked case in a boy of eight years old, in whom a presystolic murmur was heard at the apex of the heart during life, followed several days afterwards by a diastolic basal bruit. At the autopsy the sigmoid valves were puckered and incompetent, and the mitral orifice was dilated.

The *pulse* is generally, in uncomplicated cases, normal, except that it is more or less accelerated in time. If it is markedly compressible or irregular, this is probably due to concomitant mitral regurgitation.

The effect of mitral stenosis on the heart will naturally be to produce hypertrophy and dilatation of the left auricle, and ultimately of the right side. The left ventricle has less rather than increased work, and either continues of normal size, or may become somewhat atrophied, apart from comparison with the rest of the heart.*

The *compensation* for mitral obstruction is first hypertrophy and dilatation of the left auricle, and ultimately hypertrophy of the right ventricle.

* Dr Samways, among 173 cases of mitral stenosis in the post-mortem records of Guy's Hospital, found that the left ventricle was generally normal or small, rarely enlarged either by hypertrophy or dilatation ('Brit. Med. Journ.,' 1898, vol. i, p. 505).

The general symptoms are those of palpitation and dyspnœa without anasarca. The only one which is at all characteristic is hæmoptysis, which is certainly more frequent with uncomplicated mitral obstruction than with mitral regurgitation or disease of the aortic valves.

4. *Mitral incompetence* is the most frequent of all valvular lesions, but unlike the last three it is not always of organic origin. Hence we must distinguish (a) temporary and functional incompetence; (b) primary structural incompetence; and (c) secondary functional incompetence due to mitral stenosis or aortic regurgitation.

(a) Functional incompetence appears to depend upon the fact that the auriculo-ventricular orifice is not like that of the aorta, a fibrous ring, but is formed by the muscular lip of the ventricular cup. These fibres, as Ludwig proved, contract during systole so as to diminish the calibre of the opening.* Otherwise, the curtains do not suffice to close it, and regurgitation ensues. Want of due contraction of this muscular ring is probably the cause of mitral incompetence in many conditions, when a temporary systolic apical bruit is heard. It is, however, from the nature of the case impossible to prove this, and there are some difficulties which must be recognised.

One is that these functional apex-murmurs are not audible in the axilla, nor at the angle of the left scapula; whereas the physical conditions for the production and conveyance of a fluid vein would seem to be the same, whether the leakage is caused by the curtains being too small or by the orifice being too large. Again, an apical systolic murmur is sometimes heard in cases of chronic Bright's disease, not only when there is dilatation of the left ventricle, but when there is pure hypertrophy without dilatation and with the valves unaffected. If we find an apical bruit in cases of anæmia along with a systolic basic bruit, we attribute the latter to the pulmonary artery, and the former to dilatation of the mitral valve; but why such dilatation should sometimes occur and often be absent, even in severe cases of anæmia, remains a difficulty.

The apical systolic murmur of chorea is generally considered to be functional in character, and if so its mode of production is obscure (cf. vol. i, p. 882).

A systolic apex-murmur is believed by some authorities to be occasionally pericardial in origin, a true friction-sound, but it is sometimes audible in cases of adherent pericardium where there is no valvular lesion to be found after death.

The difficulty of explaining these apical murmurs is increased by the obscurity which still rests upon the nature of the normal first sound of the heart. If it is due, as taught by some physiologists, to tension of the mitral and tricuspid curtains alone, we cannot strictly apply the theory of the fluid vein to explain all functional systolic murmurs. If, as Laennec supposed, it is due to contraction of the ventricular muscle alone, its length and quality may be supposed to be variously modified enough to make of it a *bruit anormal*, without regurgitation. If, as seems most probable, it consists of two elements (the valvular tension which exactly corresponds to that of the sigmoid valves in the second sound, and a *bruit musculaire*, the addition of which gives the specific character distinguishing the first sound

* If a ligature is fastened around the arterio-ventricular groove in a living animal, it slackens with each systole, as the writer has found in repeating Ludwig's experiment.

from the second), we then have greater scope for explanation of the various systolic apical murmurs—but also greater difficulty in determining the true explanation in each case.

It has often been supposed that a temporary or functional murmur may be produced by failure or irregularity in the action of the muscoli papillares; Addison, for instance, suggested a spasmodic disorder of these muscles as an explanation of the choreic bruit. But we have no evidence of the occurrence of such a phenomenon; and in many cases of extremely irregular cardiac action no murmur is to be heard.

It must be added that none of the older explanations of either functional or organic murmurs which referred them to changes in the blood itself are compatible with physical facts; and though their admission would explain some difficulties, it would introduce many more.

Apart from temporary imperfection in the contraction of the ventricular ring, there may be permanent muscular dilatation, which will cause leaking at the mitral valve, although the curtains are normal. This occurs sometimes as a primary dilatation with more or less hypertrophy, such as was described in a previous chapter (p. 197), more frequently as a secondary result of increased blood-pressure in the left ventricle, consequent upon primary disease of the aortic valves or more distal obstruction in the systemic arteries, as in chronic Bright's disease. It may also occur as a more rapid process after scarlatina and other febrile disorders, particularly in children. This permanent dilatation with the consequent incompetence is marked by a systolic, apical bruit audible in the axilla, and leads to all the symptoms, to be presently noted, which follow the primary organic lesion of the mitral valve. Hence it is often extremely difficult in an advanced case of aortic regurgitation or of chronic Bright's disease, when mitral symptoms are superadded, to say whether the latter are due to independent structural changes in the valve or merely to consecutive dilatation. Practically the question is of minor importance.

(b) We now come to mitral regurgitation, caused by organic disease of the valve. Like other valvular lesions, it may depend upon infective ulcerative endocarditis, upon subacute rheumatic inflammation, or upon chronic sclerosis with atheroma. In the first case the symptoms and prognosis are those of the general disorder, and have been already discussed (p. 235). The third condition rarely leads to uncomplicated regurgitation; when it affects the mitral valve it usually produces stenosis. Mitral imperfection is by far the most frequent result of rheumatic endocarditis. It is common in children from an early age, but is met with frequently in adults at any age.

Its first sign seems to be the appearance, on the auricular surface of the curtains and close to their edge, of a slight ridge of fibrinous exudation, sometimes continuous, but often in separate nodules, which are arranged with remarkable regularity, forming a series of minute granules of equal size running along the opposite edges of the two curtains. It is somewhat difficult to understand how at this stage regurgitation is produced, but the presence of a typical regurgitant murmur leaves no doubt of the fact. Somewhat later the whole surface of the valves on the ventricular as well as the auricular aspect, and also the chordæ tendineæ, become affected with the same plastic inflammation. This may clear up and leave the valves uninjured, or only somewhat thicker and more opaque than before, like the pleura after recovery from pleurisy; but more often contraction takes

place, the delicate curtains are thickened, shortened, or puckered, and the chordæ tendineæ become thicker and shorter and adherent, or matted into a single deformed mass. Perforation never occurs, and rupture of a cord very rarely, except from septic inflammation supervening; but the valves often become so shrunken and tethered that they are as incompetent as if destroyed by ulceration.

The characteristic physical sign of mitral regurgitation is a bellows-murmur, systolic in rhythm, apical in locality, and audible in the axilla and at the angle of the left scapula. Sometimes the murmur is musical, and sometimes so high-pitched as to be almost a squeak. The regurgitant mitral bruit is formed in the left auricle, and conducted by the auriculo-ventricular continuity to the apex.

The pulse is characteristic: compressible, frequent, and irregular; not always small, and not dicrotic. Intermission is not common, and grouped beats are quite exceptional. It is the most irregular of all irregular pulses.

The second sound is accentuated, *i. e.* louder than usual and "brighter," *i. e.* more musical; but any clear tone, if increased in volume, makes a stronger impression on the ear, and so its characters seem more marked. It is occasionally doubled owing to the same cause—increased blood-pressure in the pulmonary artery compared with that in the aorta.

(*c*) Frequent as mitral incompetence is as a primary uncomplicated lesion, its frequency is much increased by the fact that, when the valves are thickened and the orifice contracted, their accurate adjustment during systole is often impossible. Hence the larger number of cases of stenosis are sooner or later complicated by mitral regurgitation. When this is the case, as when mitral regurgitation is consecutive to disease of the aortic valves, the clinical features of the case assume the character which belongs to cases of primary mitral incompetence. Indeed, the majority of fatal cases of mitral incompetence are found at the autopsy to be secondary to stenosis of the same valve, or to disease of the aortic valves, or to pericardial adhesions, or to chronic Bright's disease.

The *symptoms* of mitral incompetence are those common to advanced cardiac disease in general, as described above (pp. 241, 242), only somewhat modified. There is dyspnœa and orthopnœa, but the latter is usually less severe than in aortic cases; there is anæmia, but often modified by venous congestion of the lips, cheeks, chin, nose, and ears, and sometimes by an icteric tinge due to congestion of the liver. Hæmoptysis is less common than in either mitral stenosis or sigmoid disease. Dropsy, with albuminuria and slight icterus, is the most characteristic condition, while arterial anæmia with syncope and liability to sudden death is less marked than in aortic cases.

The left auricle, being habitually over-full, but with no obstacle to its contraction, dilates with but little hypertrophy. The pulmonary veins share in the change, and each pulsation of the left ventricle is transmitted to the pulmonary capillaries. Thus the lungs become congested, the blood-pressure in the pulmonary artery rises, and the right ventricle finds difficulty in expelling its contents. As a result, it hypertrophies to some extent, and dilates much more; its walls also become dense and tough. This may be the effect of hypertrophy, but it is perhaps partly due to congestion of the muscular tissue, from the cardiac circulation sharing in the general venous congestion. The tricuspid valve probably yields as soon as the ventricle begins to dilate. Its safety-valve action comes at once into

play, no doubt with relief to the heart. This regurgitation is beneficial under the temporary congestion of the right side, which occurs during exertion in a normal heart; but it now becomes permanent. The right auricle dilates as much as the left, and in chronic cases often more, as the increasing obstruction in the pulmonary capillaries leads to comparative diminution of pressure in the left ventricle and auricle.

The natural compensation of mitral regurgitation consists in hypertrophy of the right ventricle: The left ventricle is either unaffected or very slightly dilated, but it may hypertrophy if there is a complication of sclerosed arteries or chronic Bright's disease.

It is often stated that mitral regurgitation causes compensatory hypertrophy of the left ventricle, but this statement does not appear to be true in fact or reasonable in theory. Why a cavity in front of a leaking valve should have extra pressure is not clear; it must be easier to empty the ventricle (or any other chamber) by two exits than by one, and the pulse of mitral incompetence shows not high tension but low. It may be argued that the cardiac impulse is in these cases often displaced to the left, but examination after death shows that this is not due to enlargement of the left ventricle, but to the right side of the heart, which pushes the apex towards the left and downwards, or to aortic or renal disease.

The unguarded openings of the two cavæ allow the congestion of the right side of the heart to be at once transmitted to the systemic veins; each contraction of the right ventricle produces a wave which passes straight into the two cavæ and their branches, not only checking but reversing the natural current of the blood. This is seen in the external jugular veins, which become dilated and pulsate with each beat of the heart—an important sign of tricuspid regurgitation, which can be easily distinguished from the normal or exaggerated inspiratory pulsation, by its following not the pulmonary but the cardiac rhythm.*

The face and neck are generally preserved from dropsy, owing no doubt to the great assistance to the venous circulation afforded by gravity; but the arms are often found swollen, particularly over the dorsum of the hand, and the right or left limb will suffer according to the patient's position in bed.

The inferior cava is more rapidly and severely affected than the superior. The valveless hepatic veins which join it just before it enters the right auricle suffer immediately; the centre of each hepatic lobule becomes congested, the liver enlarges and is usually tender to the touch, and after a time biliary excretion is hindered, and a more or less decided jaundiced tint appears in the eyes and the skin; occasionally the hepatic channels become so dilated that the whole organ palpably throbs with each pulsation of the right ventricle, by the same mechanism as was just noticed in the case of the external jugular vein (see a paper by Dr F. Taylor, 'Guy's Hosp. Rep.,' 1875, p. 377). Even before there are symptoms of hepatic congestion, the stomach begins to suffer, dyspepsia and distressing flatulence greatly adding to the disturbance of the heart and the distress of the patient. After death the mucous membrane of the stomach is found swollen, deep red, often with minute ecchymoses, and covered with tenacious mucus. The intestines seem to be the least affected of the viscera in the portal region; but the spleen from continued congestion becomes, not swollen, as in

* Dilatation and pulsation of the internal jugular veins was recognised by Lancisi as a sign of ("aneurysm") dilatation of the right ventricle. Corvisart disbelieved this statement, but Lœnnec reasserted it ('Ausc. Méd.,' ii. 263-4).

cases of portal obstruction, but dense, dark coloured, and crisp to the knife.

The kidneys show their congestion by secreting scanty, high-coloured urine with raised specific gravity and an abundant deposit of lithates. Albumen is often present, but hæmaturia from cardiac disease of any kind is much less common. Hæmorrhoids if present are aggravated, although this is a less frequent effect of disease of the heart than it is of cirrhosis of the liver. The congestion of the lower extremities leads to anasarca, which begins about the ankles and dorsum of the foot, and spreads upward to the thighs. It is remarkable that the scrotum almost always escapes. Ascites is nearly constant among the later symptoms of cardiac dropsy, but is seldom so large as in cases of hepatic obstruction. Some amount of pleural and pericardial effusion is almost always found after death, but there is seldom enough to be detected during life, or to call for treatment. Hæmorrhage is a rare event, but occasionally it may occur either from the lungs or the gastric vessels.

Valvular affections of the right side.—These are seldom met with, and comparatively unimportant.

5. *Pulmonary or dextro-sigmoid stenosis with obstruction.**—This is the least rare of primary organic lesions of the right side. It is almost always congenital, though due in most cases to intra-uterine endocarditis, and will be described with other malformations (p. 274).

When, however, we hear a systolic basal murmur traceable upwards and to the left—that is to say, in the second and first intercostal spaces—it very seldom indicates congenital dextro-sigmoid stenosis. The murmur is temporary, not permanent, and is unaccompanied by signs of dilatation of the right side of the heart or of cyanosis. It is loud, blowing, often harsh, easily heightened by movement, and very restricted in locality. Occasionally it is heard in cases of phthisis, when it is probably produced by slight traction or pressure on the pulmonary trunk by pleural adhesions, and intensified by solidification of the lung. Much more often this systolic “pulmonary” murmur is heard in cases of extreme anæmia, in chlorosis frequently, in the anæmia of rheumatic fever and of Bright’s disease occasionally, and sometimes in cases of Addison’s idiopathic anæmia, of leuchæmia, and of Hodgkin’s disease.

Dr Foxwell, of Birmingham, has observed that the conus arteriosus of the right ventricle, as well as the pulmonary trunk, is dilated after death, and that the sigmoid valves are thus pushed up nearer to the more fixed bifurcation of the artery. Thus the vessel becomes tortuous, like the brachial artery affected with atheroma, and the constriction which results (as in bending an india-rubber tube) gives the condition necessary to form a fluid vein (Bradshaw Lecture, ‘Lancet,’ Nov. 4th, 1899).

* It is unfortunate that we have no word for the valves which guard the entrance to either the pulmonary artery or the aorta. The adjective “pulmonary” is naturally taken to apply to the lungs rather than to their artery or its valves, and the adjective “aortic” rather to the trunk of the great artery than to its valves. Some years ago the writer proposed to appropriate the terms semilunar and sigmoid by an arbitrary convention to the aortic and pulmonary valves respectively (‘Journ. of Anat. and Phys.,’ 1875). Dr Donald McAlister, of Cambridge, has suggested the words dextro- and lævo-sigmoid for the same purpose. Some such addition to our nomenclature would be of much practical value, but it seems almost hopeless to introduce changes of this kind, however much needed. Perhaps “sigmoid” as a synonym for aortic, and “dextro-sigmoid” as a synonym for pulmonary, have the best chance, and in the present and the last edition of this book they have been introduced, but not, it is hoped, so as to cause ambiguity.

A similar murmur is occasionally audible in the aortic area; and the arterial bruit heard in the great vessels of the neck, in the abdominal aorta, and wherever a superficial artery can be pressed by the stethoscope in anæmic subjects, is clinically and, in all probability, physically related to that of the pulmonary artery. The explanation above given, by which this murmur is reduced to an example of a fluid vein, is more satisfactory than any other.

In children a dextro-sigmoid systolic bruit may sometimes be heard, which is produced by the pressure of the stethoscope on the yielding parietes of the chest.

6. *Pulmonary or dextro-sigmoid imperfection* is the rarest of all valvular lesions. In the only cases seen by the writer, ulcerative endocarditis had affected the right as well as the left side of the heart. In 'Allbutt's System,' Dr Pitt has given the result of 99 collected cases of this lesion, of which 29 were from Guy's Hospital, and all verified by autopsy. In 57 the condition was one of septic endocarditis, and followed pneumonia in 5 cases, puerperal fever in 4, and gonorrhœa in 8. It often complicated congenital lesions, and was rarely confined to the pulmonary valves. Antecedent rheumatism was seldom noted. Its sign would be a diastolic basal murmur, traceable down the sternum from the third left costal cartilage; but Dr Pitt found this to be seldom heard—only a systolic murmur from concomitant stenosis. The usual symptoms were hæmoptysis and dyspnœa. The incompetence was occasionally due to dilatation of the pulmonary artery owing to obstruction in the lungs or left side of the heart.

7. *Tricuspid stenosis* with obstruction of the passage of blood from the cavæ and right auricle to the right ventricle and lungs is a condition occasionally seen, usually as a complication of mitral stenosis.

Dr Bedford Fenwick collected forty-six cases of tricuspid contraction, of which the majority occurred in young women, and only five in men ('Path. Trans.,' 1881, p. 48). In half there was no rheumatic history giving a clue to their origin. Every case was accompanied by a corresponding condition of the mitral orifice.

Among 87 cases collected by Dr Pitt from the *post-mortem* records of Guy's Hospital during twenty-six years, the majority were fatal between the ages of fifteen and fifty, and more than half under thirty. There was a preponderance of cases in women. In every case but two there was also mitral stenosis present. In more than half the cases there was a definite history of rheumatic fever, in twelve more of chorea, and in others of more or less doubtful "rheumatism."

A presystolic bruit was only heard in ten cases, and in many no murmur at all was audible.

The physical sign of tricuspid stenosis is a presystolic murmur audible at mid-sternum between the fifth costal cartilages, and traceable as far as the ensiform cartilage.

8. *Tricuspid incompetence with regurgitation*.—This is extremely common as a normal or almost normal adaptation to relieve temporary pressure on the right side of the heart. It probably occurs in every healthy man who runs a long race, or who dives and continues as long as he can under water.*

* For the explanation of this mechanism see the late Mr Wilkinson King's papers on the "Safety-valve Functions of the Tricuspid, or the Moderator Band of the Right Ventricle," 'Guy's Hosp. Reports,' vols. ii and vi of the first series.

It is also the probable explanation of the temporary systolic murmur which is frequently heard when apparently healthy persons have hurried upstairs to be examined for assurance ; or in young men who fear that they have disease of the heart, and are naturally excited under auscultation. The murmur always goes with accelerated pulse, often with irregularity and palpitation of the heart, and it speedily disappears after the excitement is over.

More permanent tricuspid regurgitation is frequently present, as above described, in chronic cases of mitral obstruction or incompetence, and also in those of dilatation of the right side of the heart, consecutive to chronic bronchitis with emphysema ; for in all such cases there is not only general venous congestion and dropsy, but after death the tricuspid orifice is found dilated, and its valve incompetent. But it is remarkable how seldom we can identify a bruit as the result of this regurgitation. In primary cardiac cases it may probably often be masked by an aortic or mitral murmur, and in dropsy and cyanosis of pulmonary origin rapid breathing and bronchial rhonchi may obscure it, or emphysematous lungs may prevent its reaching the ear.

Nevertheless, its comparative rarity remains a difficulty. Possibly when there is decided dilatation of the tricuspid orifice the leak is so wide that it nearly approximates to the dimensions of the auricle itself, and thus the conditions of a fluid vein would not be fulfilled. This explanation would agree with the much less frequent but still undoubted absence of a mitral murmur in some cases of regurgitation, and with the constancy of a diastolic bruit in those of aortic regurgitation, where the difference in calibre between the largest gap in the sigmoid valves and the left ventricle in diastole must be far greater than that between a badly leaking mitral orifice and the left auricle.

If carefully sought for, we may occasionally detect a tricuspid bruit supervening in a case of chronic mitral disease ; and its presence is not infrequently recognised in the later stages of bronchitis with emphysema, of cirrhosis of the lung, or of extremely chronic fibroid phthisis. Its characters are systolic rhythm, and a limited seat in the mid-sternum or over the ensiform cartilage. Its quality is usually that of a bellows murmur. The second pulmonary sound is less loud than in health.

The radial pulse is not directly affected, but is usually frequent and often irregular from mitral disease. Beside the murmur, and more constant than it, is the symptom of jugular venous pulsation synchronous with the cardiac syncope. Tricuspid incompetence from primary lesions of the valve is a rare condition, and most often occurs along with ulceration of other valves in cases of septic endocarditis.

Combined cardiac lesions.—The most common combination is that of aortic (sigmoid) regurgitation and obstruction, giving the characteristic to-and-fro murmur at the base. Its effect on the heart and on the pulse and its clinical course are those of sigmoid incompetence.

Often associated with these as an organic lesion is mitral incompetence, and still more frequently secondary dilatation of the mitral orifice with regurgitation. Indeed, the natural course of aortic valvular disease when it is not cut short by sudden death is towards mitral incompetence with congested lungs, dilated right side of the heart, and general dropsy. Clinically such cases are mitral ; the pulse loses its water-hammer character and

becomes small, weak, and irregular, and the attacks of anginal pain are often relieved. Aortic lesions rarely follow those of the mitral valve.

Mitral stenosis is, as above mentioned, often associated with regurgitation; usually the latter is the complication, but early imperfection of the mitral valve from rheumatic endocarditis may gradually be converted by successive attacks into a cicatricial stenosis. In such cases the pulse is that of regurgitation, and the general symptoms indistinguishable from those of primary mitral incompetence. Mitral regurgitation as the result of dilatation of the left ventricle in the latter stages of chronic Bright's disease is frequently met with, and leads to important clinical complications, to be hereafter described.

Tricuspid incompetence as the result of dilatation of the right ventricle is, we have seen, extremely common in the final stages of heart disease whether aortic or mitral in origin, and it is equally so as the result of chronic pulmonary obstruction from emphysema, bronchitis, asthma, or the most protracted forms of phthisis. Primary tricuspid incompetence is nearly unknown.

Tricuspid stenosis, though not so rare as was once thought, is almost always combined with mitral stenosis, due to the same causes, and helping to produce the same effects.

Pulmonary (or dextro-sigmoid) obstruction is a congenital defect and often associated with an incomplete ventricular septum.

Dextro-sigmoid incompetence is occasionally seen as a complication of left sigmoid or mitral disease of a septic kind.

Comparative frequency of valvular lesions of the heart.—The relative frequency of the several valvular lesions of the heart is not easy to determine: partly because during life it is often a question whether a murmur is due to functional or organic disease; partly because, even after death, there may be a question as to the perfect competence of a valve; and partly because there are so frequently combinations of more than one valvular lesion.

Clinically, an apical systolic bruit is undoubtedly the most frequent; a to-and-fro basic bruit comes next, and a presystolic murmur third. Mitral regurgitation, therefore, is by far the most common form of heart disease, as judged by physical signs; but mitral stenosis is decidedly more frequent in adults as shown in the deadhouse, and even before adult life the same is probably true after the seventh and eighth year. Mitral is far more common than sigmoid disease in women, and mitral stenosis more common in women than in men.

From the *post-mortem* records of Guy's Hospital, Dr Shaw ascertained that out of 147 cases the mitral valve alone was diseased in 41, the aortic alone in 26, and both in 69. On the right side there were 14 cases of tricuspid stenosis, 7 of its dilatation and thickening, and 5 of disease of the pulmonary valves. In only 3 cases were there lesions on the right side without the left being also affected.

Prognosis.—According to Wilks, "as regards the relative gravity of the valvular diseases, probably aortic obstruction is the least serious; next to this, stenosis of the mitral valve, which with a compensating hypertrophied auricle may endure for years. Regurgitant diseases are far more serious, but mitral regurgitation is less so than aortic, for the latter condition often leads to sudden death."

This opinion was corroborated by Peacock and by Bristowe, but there are authors who regard stenosis as more serious than incompetence, both for the sigmoid and mitral valves.

If we exclude cases of acute infective ulceration, which proves fatal not so much by its mechanical effects as by its septic emboli and toxines, undoubtedly the gravest of all valvular lesions is aortic incompetence with dilatation of the left ventricle. Nevertheless, well-compensated aortic leaking may go on with care for years, and when mitral dilatation and incompetence become added, the danger is rather diminished than increased.

The least grave single valvular lesion is aortic stenosis, with moderate compensating hypertrophy. Still, in the case of elderly men with atheromatous and inelastic arteries, these cases are liable to be cut short by sudden and fatal syncope.

Intermediate in danger between the two forms of aortic valvular diseases are mitral obstruction and regurgitation. The former is more often met with accidentally, without symptoms; and in all likelihood will last longer. The latter causes severe symptoms, but of all valvular lesions is most efficiently relieved by appropriate treatment. The most common mitral cases are those of stenosis with secondary regurgitation, and these are the gravest; next to cases of aortic incompetence they offer the shortest prospect of continued life. The danger of sudden death is greater in uncomplicated mitral stenosis than when there is regurgitation either primary or secondary.

Other considerations, however, modify the prognosis derived from the anatomical character of the valvular lesion. One is that of age, another is mode of life as to exposure, intemperance, and occupation. A rheumatic lesion is, other things being equal, of better outlook than one of atheromatous origin, partly because the former occurs in younger, the latter in older patients.

Another important consideration is the state of nutrition of the cardiac walls. A healthy hypertrophied auricle will often overcome the impediment of a narrowed left ostium, a healthy hypertrophied right ventricle, a leaking mitral valve; and a healthy hypertrophied left ventricle will compensate a narrowed or even a leaking aortic valve. But when thrombosis and anæmia, fatty or fibrous degeneration, or dilatation befall the muscle, then the hydraulic effects of the valvular defect become apparent and too often ingravescient.

Right side.—Of lesions of the right side, pulmonary stenosis is almost always a congenital defect; it leads to cyanosis, and life is rarely protracted beyond childhood, except in the slighter forms of the disease, and even then the patients usually die as young adults from phthisis. Occasionally, however, they may live to be fifty years old.

Tricuspid stenosis is almost always a concomitant of the same change in the mitral orifice, and, if recognised during life, would add to the gravity of the prognosis. It was found after death in 32 cases out of 196 at Guy's Hospital by Dr Samways, and most of these were cases in which the mitral orifice admitted only one finger ($2\frac{1}{4}$ inches in circumference). Tricuspid regurgitation is very common, but only as the result either of pulmonary obstruction or of primary valvular lesions on the left side of the heart. It is probably a beneficial event, by allowing the high blood-pressure to be distributed through the systemic veins and relieved by anasarca.

Left side.—It is only in the latter stage of aortic valvular disease that

dropsy and general venous congestion with albuminuria and jaundice occur, when the left ventricle and the mitral valve have yielded and become dilated. Hæmoptysis and epistaxis belong particularly to its early stages. Severe cardiac pain, urgent dyspnoea with orthopnoea, and excessive palpitation, are miseries which attend the later stages of aortic incompetence. Hæmoptysis is far more often from mitral stenosis than from mitral regurgitation.

Fatal syncope.—Syncope and sudden death are usually and justly associated with sigmoid rather than mitral disease. In mitral disease sudden death occurs more often in cases of obstruction than in those of incompetence.

In 14 consecutive cases of sudden death by syncope from valvular disease of the heart at Guy's Hospital, there were five due to aortic regurgitation, fatal at ages between twenty and seventy, and nine due to mitral obstruction, three of the patients being between twelve and fourteen, and the other six between twenty-six and forty-eight. None were due to mitral incompetence alone. The total number of mitral compared with aortic cases was very much larger, perhaps three times as large.

Probably atheroma of the aortic orifice, with considerable stenosis and incompetence, is the form of valvular disease which most often leads to absolutely sudden death. Even severe cases of aortic regurgitation, occurring in younger patients as the result of rheumatism, produce comparatively slight symptoms while well compensated; but when dilatation occurs, the mitral valve gives way, pulmonary congestion and dropsy soon follow, and the case becomes a mixed one. In such cases death may still supervene by terribly sudden syncope, but there has been ample warning beforehand.

With respect to *sudden death* in general, Dr Shaw found that there were 34 cases in the hospital in the ten years 1875—84, from all causes, excluding injury and hæmorrhage. Of these, in 14 the cardiac valves were found normal, in 20 they were diseased; the aortic in 13, the mitral in 7, and in all these seven mitral cases the left ostium was constricted. Of the above 14 cases with healthy valves, the cardiac muscle was degenerated in 11, viz. fatty in 8 and fibroid in 3.

An independent supplementary search, made for this edition by Dr Walter Telling for the years 1894-5-6, shows that of 16 cases in which death might be called sudden, *i. e.* with serious symptoms of less than ten hours' duration, there were two due to aortic regurgitation (at eight and thirty-one), one to a fatty heart at sixty-six, two to rupture of an aortic aneurysm (at twenty-nine and sixty-two), and eight to cerebral hæmorrhage. Of the remaining three, one was caused by profuse hæmoptysis in the course of phthisis, one by uræmia, and one by perforation of a stercoral ulcer above a cancerous stricture of the colon.

Of *compensation* as influencing prognosis of heart disease, we have already stated the importance of hypertrophy of the muscular walls next preceding the valvular lesion, the left ventricle for the aortic (sigmoid) valves, the left auricle for mitral obstruction, and the right ventricle for mitral regurgitation. Dilatation is the opposite process, in which the cavities act only as elastic, not as contractile tissues; but a moderate degree of dilatation is probably not only inevitable but advantageous behind a leaking valve.

We may, however, exaggerate the value of a compensatory hypertrophy. If there are no symptoms, the absence of hypertrophy shows that the heart

does its work well in spite of the valvular lesion by its own natural powers.

With regard to *duration* of valvular disease, judging by the patient's age at the time of death, Dr Shaw found that of 95 fatal *aortic* cases at Guy's Hospital (26 without and 69 with concomitant mitral lesions) 1 died under ten years old, 14 between ten and twenty, 40 between twenty and forty, 33 between forty and sixty, and 7 above sixty. Of 41 fatal *mitral* cases, none died under ten, 6 between ten and twenty, 14 between twenty and forty, 20 between forty and sixty, and only 1 above sixty. The general experience is that the duration of aortic disease is considerably less than that of mitral, *i. e.* the time of death is earlier.

Taking clinical instead of pathological data, he found that among 80 patients (excluding cases of ulcerative endocarditis), the shortest period which elapsed between the appearance of marked cardiac symptoms and death was in *aortic* cases two weeks, in *mitral* four. The average period (not very important, since it may be so much influenced by a single exceptional case) was only two and a half years in "mitral" cases in general, one year and a half in aortic cases with mitral regurgitation also, and as much as four and a half years in simple, *i. e.* well-compensated aortic cases. In cases of mitral stenosis without symptoms or sign of regurgitation, the average duration was three and a half years.

The average age in 53 persons dying of mitral stenosis (with or without regurgitation) was found by Broadbent to be about thirty-eight for women and thirty-three for men; in 42 persons observed by Hayden about thirty-eight; in 61 cases (many of them children) recorded by Samson, thirty-three; and in a much larger number of cases (196) collected from the *post-mortem* records of Guy's Hospital, Dr Samways found the average age of death was thirty-eight, without any difference between males and females ('Brit. Med. Journ.,' 1897, vol. i, p. 197, and 1898, vol. i, p. 364), in 1896, Nov. 28th, and 1898, Feb. 5th.

In the most favourable form of valvular disease, aortic stenosis, Peacock recorded a case exhibiting the most extreme degree of obstruction, in which compensation was so perfect that the patient died at seventy-five after an operation for strangulated hernia. For aortic regurgitation the same accurate observer estimated as the longest period of survival after the existence of the lesion was ascertained, five or perhaps seven years. Happily, however, this period is often exceeded.

So far as prognosis is related to the *cause* of the cardiac lesion, it is better in rheumatic than in atheromatous cases. The most favourable cases, apart from the nature and extent of the lesion, are those in young subjects whose hearts have been damaged by rheumatic endocarditis, but whose nutrition and renal depuration are vigorous. The worst cases are those of ingravescient atheroma with confirmed gout and granular kidneys.

Indeed it may be generally said that, other things being equal, the younger the patient the better the prognosis; but the forecast must always be doubtful until a year or more after signs of valvular disease have appeared, so that we can judge of the amount of compensation and the absence of ingravescient symptoms.*

As to *sex*, the prognosis is better for women than for men, because in the majority of cases their lives are easier and more tranquil.

* There is in early life a special power of repair and self-adjustment in the heart which warrants our expressing a more cheerful prognosis than would be justifiable in cases of cardiac disease occurring in a grown person (Chas. West). (In children,) as long as the cardiac lesion gives rise to no symptoms, the prognosis is very favourable (Eustace Smith).

Apart from the nature of the lesion, a most important element of prognosis is the kind of life which the patient is able and willing to live. Hard work and exposure, dissipation, starvation, and drink bring otherwise favourable cases to an early fatal result. Moderation in all things, a gentle life, an equable temper, with exemption from attacks of rheumatism, from bronchial catarrh and pneumonia, from muscular strains, and from the excitement of passion, are the conditions which prolong otherwise unfavourable cases to advanced age, and that not infrequently after a far from useless or unhappy life.*

On the whole, the hospital student will find that in private practice he will succeed far better with the same treatment than he could anticipate from his previous experience. Sir Andrew Clark, Professor Gairdner, and others have published remarkable facts as to the long duration of life in cases not with cardiac murmurs only, but with conclusive symptoms of organic disease of the heart ('Brit. Med. Journ.,' Feb. 5th and 12th, 1887).

The two following cases have come under the writer's observation:

An old gentleman, the subject of atheroma of the left sigmoid valves with obstruction and regurgitation, was advised to live with the precautions necessary to his condition, and from about sixty-five to nearly eighty continued to enjoy good health. He was of a placid temper, and so obedient a patient that he would let a train start before his eyes rather than hurry to catch it. He died at last from cerebral hæmorrhage at a little over eighty.

A young man of thirty-one had been the subject of cardiac disease since an attack of rheumatism when a child. He had been educated at home, was never suffered to play like other boys, and at college lived the same careful and moderate life. When the writer saw him he was married, with healthy children, in active business, with good breath and healthy complexion. There was a loud diastolic basic murmur and a splashing pulse, with scarcely any signs of dilatation or hypertrophy, *i. e.* the nearly normal ventricle was sufficient for the regular calls upon its power.

From the point of view, however, of *insurance*, the risk is great and difficult to estimate; so that few offices will accept on any terms "lives" which are weighted with organic disease of the cardiac valves. The only duty of the advising physician is not to mistake functional disturbance, with or without murmurs, for organic disease.†

Treatment.—The objects of treatment in valvular disease of the heart are—first, to spare the damaged organ as much work as possible, and therefore to avoid all strain on the circulation, whether of muscular exertion or mental agitation; secondly, to maintain the nutrition of the myocardium, and particularly of the right or left ventricle, in the best condition, so that (with or without compensatory hypertrophy) it may suffice to make up for the damaged valve; thirdly, to moderate the rapidity and increase the force of the cardiac contractions, and to lengthen the period of repose afforded by the interval between one beat and the next; fourthly, to prevent or relieve dyspepsia and flatulence, and to keep the bowels and the kidneys to their work; lastly, to succour the final stage of venous congestion and dropsy by mechanically relieving the circulation of the load of stagnant blood or exuded serum.

The first indication is met by enjoining a quiet, self-restrained life, with avoidance of the exposure which leads to bronchitis, and the intemperance which may end in Bright's disease; the second by prescribing animal food with moderate use of stimulants, and the addition of tincture of steel, or

* The late Sir Stafford Northcote was a striking instance of this result.

† The writer may be allowed to refer to a paper "On some Points in the Prognosis and Treatment of Cardiac Disease," read before the Hunterian Society ('Brit. Med. Journ.,' November, 1890), and a lecture on Life Assurance, printed in the 'Guy's Hospital Gazette' for 1897.

any other preparation of iron which suits the patient better. In some cases arsenic is even more useful, and acts in much the same way, by improving the blood and the muscles. To fulfil the third indication, we possess a direct cardiac moderator, making irregular action rhythmical, rapid ineffectual pulsation slower and more efficient, and feeble contractions strong; steadying the pulse, and raising the blood-pressure in the systemic arteries.* This invaluable drug is digitalis. We use either the dried and powdered leaves of foxglove, or the infusion and tincture prepared from them. The condition most favourably influenced by digitalis is one of mitral regurgitation, with a dilated heart, and a rapid, irregular, compressible pulse. In cases of mitral stenosis this drug is far less constantly successful, though it is still the best we have. Nor is digitalis of so much service in functional disorders of the heart or in any condition attended by fever. In disease of the sigmoid valves with compensating hypertrophy of the left ventricle it is often useful, but when there is much dilatation it may be dangerous. In cases of aortic regurgitation especially, it must be used with great caution, and never when the pulse is slow. When, however, primary sigmoid disease or mitral stenosis becomes complicated with mitral regurgitation, and the pulse acquires a "mitral" character, digitalis is invaluable. In fact, in all cases of cardiac dropsy it is our most important remedy. Digitalis should not, however, be given when compensation is good and symptoms absent. All that it could do is done by natural means, and it is much wiser not to interfere.

Beside its direct action on the heart, digitalis acts as a powerful diuretic, probably by raising the blood-pressure in the renal artery. For this object it is much assisted by combination with mercury and squill. The pill so composed—"the pill," as Addison called it—is the most valuable of remedies for cardiac dropsy. It appears to have been introduced by Matthew Baillie. Other diuretics may also be usefully prescribed—acetate of potash, cream-of-tartar lemonade (the "imperial drink"), sweet spirits of nitre, decoction of broom-tops, and resin of copaiba.

Diaphoretics are not of much service in these cases, and if the kidneys are unfortunately diseased we must depend on the action of hydragogue purgatives. In other cases more gentle aperients help the action of the diuretic drugs.

When there is great ascites, it is well to tap at once, and to prescribe the pill when the kidneys have been thus relieved. For general dropsy acupuncture is a still better remedy. The patient should be got out of bed and the effused serum allowed to gravitate into his legs—a great relief in itself. They should then be washed, smeared with weak carbolic oil, and punctured with a needle or lancet quite through the skin into the lymph-spaces beneath. The benefit of the free flow of serum thus obtained is often most striking. The patient finds his breathing easier; he can sleep again and take food; the kidneys begin to secrete; the liver is relieved of its congestion; and a man who was at the point of death by suffocation is able to go about free from dropsy, and in comparative health.

Occasionally engorgement of the lung will come on so quickly, and the whole venous system with the right side of the heart be so blocked, that *venesection* is called for, and may be practised with the greatest benefit; the indications for this treatment are orthopnoea, lividity of countenance, great

* This last vaso-motor action of digitalis was ascertained by Brunton, and in this it differs from strophanthus (Fraser, 1885, Brunton and Tunnicliffe, 'Journ. of Phys.,' 1896).

distress of breathing, and pulsation of the jugular veins. After ten ounces or perhaps not until a pint of blood has been removed, the lividity will pass off, the breathing become tranquil, and often refreshing sleep ensue. Moreover, diuretics and digitalis will now act with effect, which before they could not do.

If the prejudices of the patient or his friends forbid bleeding, cupping between the shoulders is the next best thing,—certainly more efficient than leeching. But a purge of calomel or blue pill, followed by a full dose of magnesium sulphate, is always a good sequel, and often a very tolerable substitute for letting blood.

Hæmorrhage from the lungs in cases of mitral stenosis is a valuable means of relief, and should never be stopped, even if we could do it.

For a most distressing symptom of heart disease—sleeplessness, opiates may be given, not only with safety, but with the greatest benefit. Five grains of the compound soap pill, or one sixth or one fourth of a grain of morphia may be taken at bedtime, or, if it produce gastric disturbance, it may be injected hypodermically. If, from the presence of renal disease or any other cause, opiates are unsuitable, we may use Hoffmann's anodyne (sp. ætheris comp.) or full doses of hyoscyamus. Chloral hydrate is counter-indicated, and paraldehyde is unpleasant; but a fluid drachm of tincture of henbane in a little hot brandy and water at bedtime is a safe hypnotic; and particularly in elderly people is preferable to bromides. Of all the stronger hypnotics, except opium, the writer has found chloral-amide and sulphonal most useful in cardiac cases.

Extract and tincture of the flowers and stem of lily of the valley (*Convallaria majalis*) have been of late years revived as a remedy in similar conditions to those in which digitalis is valuable. The drug is undoubtedly efficacious in raising blood-pressure, and thus producing diuresis. It appears to be devoid of danger, and sometimes proves useful when digitalis fails or is counter-indicated. It was employed for dropsy in the seventeenth century, long before Withering introduced the use of digitalis, and it is still a popular remedy in Russia.

Citrate of caffeine is another useful cardiac remedy, and is much praised by some experienced physicians. To obtain its good effect it should be given in full doses.

Strophanthus, an African arrow poison (*S. hispidus*, nat. ord. Apocynaceæ), was introduced by Fraser, of Edinburgh. A tincture is prepared from the seeds, which yield an active glycoside, strophanthin. Fraser found that this drug increased the length of the cardiac systole in animals while slowing the pulsations, and that it was more powerful than digitalis. It appears not to have a cumulative effect. After thorough investigation in the laboratory it was tried in cases of cardiac, particularly mitral, disease (in doses of five or ten minims of the tincture or more), and with good results. Its remedial action is like that of digitalis, and it sometimes appears to succeed better, at least for a time; but at present strophanthus has shown no title to supersede the older remedy, and not infrequently fails where digitalis succeeds. See Prof. Fraser's paper (in the 'British Medical Journal,' November 14th, 1885), and one by Dr Quinlan (*ibid.*, August 27th, 1887).

Increasing experience of strophanthus has raised the writer's estimation of its value. It is safe and useful, particularly in cases of mitral disease or of functional tachycardia and palpitation. It suits children well, and is often useful in chronic cardiac cases in adults. He has never seen the

striking and admirable results of digitalis in the worst cases quite attained by strophanthus or any other drug; but he has found the addition of strophanthus to digitalis succeed where neither drug was so useful alone.

In cases of sigmoid regurgitation, where digitalis is useless or counter-indicated, we depend chiefly upon iron or belladonna in the early stages, and on ammonia, senega, and ether in the later.

One important object in most cases of cardiac disease is to prevent flatulent dyspepsia by careful diet, and by the use of soda and nux vomica, bismuth, tincture of cardamoms, peppermint, and other carminatives. Strong coffee, spices and pepper, wine, aromatic spirits of ammonia and brandy are each of them useful.

In many cases of disease of the sigmoid valves, usually before general dilatation of the heart and dropsy have set in, symptoms appear like those of angina pectoris. For this severe paroxysmal pain nitro-glycerine and nitrite of amyl are the appropriate remedies, with repeated small bleedings from the arm. Belladonna is sometimes given in these cases with good effect; also iodide of potassium, which has a double application in disease of the heart, anodyne, by diminishing tension, as in aneurysm, and diuretic in cases of cardiac dropsy.

Prof. Oertel has advocated treatment of disease of the heart by a system of carefully arranged gymnastics, including ascent of hills. This plan has been criticised both in Germany and elsewhere. The heart can never be an idle muscle, and when there is a valvular lesion present, the difficulty stimulates it to increased exertion. To add to this stimulus seems unnecessary, if not injurious; and to urge a patient to tax his heart beyond what he can do with comfort is meddling practice; we have ample experience of its bad effects from what we see in patients whose necessity compels them to practise the cure without knowing it.

More recently what is called the Schott treatment, as practised at Nauheim, has become more fashionable. It consists in a combination of hot saline baths (90° F.) with regulated muscular exercises. That the cautious and tentative exercise of the arms against moderate resistance may be useful in certain cases is possible, and that hot baths temporarily divert blood to the surface of the body and for a time raise the arterial blood-pressure is demonstrable. But good of this kind can be obtained by the use of baths anywhere, and whether warmed in the earth or in a boiler. Some of the statements made as to the rapid and permanent diminution of the cardiac dulness can only be attributed to the difficulties of percussion and the remarkable power of expectation over the faculty of observation.*

CONGENITAL DISEASE OF THE HEART.—This frequently depends on a primary defect of development, but also upon intra-uterine inflammation, sometimes perhaps of a true rheumatic character.†

Malformation from arrest of development without evidence of endocarditis.—When the heart consists (like that of a fish) of only two cavities, the auricle and ventricle, without any septum making a right and left divi-

* See on this subject Douglas Powell's judicious criticism (Lumleian Lectures, 1899, pp. 80-90). Also some remarks by Dr Hale White on the Oertel-Schweniger and the Schott treatment in the 'Guy's Hosp. Gazette,' Sept. 15th, 1900, pp. 104 and 105, and the earlier papers by Dr. Poore 'Brit. Med. Journ.,' 1895, vol. ii, p. 1195), and Sir William Broadbent (ibid, 1896, vol i, p. 769).

† The most important contributions to the pathology of this subject are by Rokitsky. Rauchfuss, and Kussmaul in Germany, and by Norman Chevers and Peacock in England.

sion, life is rarely sustained after the cessation of the placental circulation at birth. One infant, however, with this defect is recorded to have lived seven days, and two others three days.

With two completely formed auricles and one ventricle, from which an aorta arises and supplies both lungs and body (as in a frog), life may be prolonged for weeks or even months (see Dr Peacock's collection of cases in his monograph on 'Malformations of the Human Heart,' 1866); and when the pulmonary artery and aorta are normal, adult life may be occasionally reached even though there is no trace of a septum in the ventricle.

A mere imperfection of the septum between the two ventricles (as in the chelonian reptiles) is a less important defect of development. It almost always occupies the "undefended space" between the base of the left ventricle and the sinus of the right. Persons with this abnormality have lived to adult age in apparent health, and died of some independent disorder. They usually, however, suffer from cold extremities, are unable to take active exercise, are liable to dyspnoea on exertion, and to cyanosis in cold weather.

Results of intra-uterine endocarditis.—It appears from twenty-three cases tabulated by Schipmann that this is rare before the fourth month of foetal life. By far the most common and important effect of inflammation of the foetal heart is *stenosis of the pulmonary orifice*. The limitation to the right side of the organ really comes under the same law as the corresponding limitation to the left side in extra-uterine life; in both cases the cavity which has most work to perform suffers most. Why the tricuspid valve should so frequently escape is less clear; probably it may depend on part of the incoming foetal blood being diverted to the foramen ovale. Cases, however, of congenital stenosis of the right auriculo-ventricular valve are recorded.

The effect of this obstruction, occurring before the septum between the ventricles is completed, will be to perpetuate the aperture of communication in the undefended space; the ductus arteriosus will remain permeable, and the foramen ovale will continue unclosed.

The first case of this remarkable condition was recorded by Sandifort at Leyden in 1777, and the next was by William Hunter at Glasgow in 1783. In each the patient was a boy who lived to the age of about twelve years. Numerous cases have been since observed, in most of which the aorta communicates with the right as well as the left ventricle. In the most extreme degree of the lesion the pulmonary artery is completely obstructed (*atresia*), so that not only some but all the blood which reaches the right ventricle is expelled through the aorta, and the lungs are supplied through the ductus arteriosus. The septum ventriculorum is open, and under such circumstances life may be prolonged for months or possibly for more than a year. Sometimes the constriction is not at the orifice of the pulmonary artery, but at the junction of the sinus with the rest of the right ventricle, so that that cavity of the heart is divided into two, like the ventricle and *bulbus arteriosus* of a frog's heart. From the position of the opening in the undefended space above described, there is still free communication between the right and left ventricles, and so the results are much the same as those which accompany moderate stenosis of the pulmonary orifice.

Lastly, the effects of endocarditis of the right side may be combined with one or any of the remarkable *transpositions* which occur in the origin of the great vessels of the heart. These combinations have been particu-

larly investigated by Kussmaul, and are of great interest from a morphological point of view, but they are not frequent enough to be of clinical importance. Moreover, as we shall see, the physical signs and symptoms of congenital lesions of the heart are rarely capable of leading to more than a general recognition of their presence, and we must be content to base further diagnosis upon the age which the patient has attained, and our knowledge of the relative frequency of the several lesions.

Prognosis.—In many cases it is remarkable how adaptation and compensation by dilatation and hypertrophy prevent the results which beforehand would appear inevitable. The greatest mortality occurs in the first days or weeks of extra-uterine life. All the worst cases end in death before the infant is a month or two old, and if he survives the first year there is good probability of his being reared. With the development of the body at puberty fresh stress is put upon the heart, and often severe symptoms then first show themselves. Even if adult life be reached, the dangers of exposure to cold producing bronchial and pulmonary inflammation, and of muscular exertion breaking down the compensatory power of the heart are such, that of those who survive childhood few live beyond the age of thirty. The same conditions, exposure and exertion, sufficiently explain the undoubtedly worse prognosis for males. Among the longest survivals in cases of (usually very moderate) congenital pulmonary stenosis, two cases were fatal at thirty, one at thirty-seven, two at forty-four, one at fifty-seven, and two at sixty.

The writer met with a very marked case of morbus cæruleus in a young man of twenty-five; and a mild case in a lady who was living and tolerably well when past fifty.

Rokitansky believed that cyanosis protected from tuberculosis, Lebert thought it favoured consumption. In 56 cases of malformation of the heart with more or less cyanosis, where patients reached the age of eight, Peacock found that nine died of tubercular disease of the lungs. When puberty is attained (and from thirteen to twenty-five) phthisis is undoubtedly the most common cause of death, a remarkable contrast to its rarity in stenosis of the orifices of the *left* side of the heart. The chief peculiarity of the disease is the frequency of severe hæmorrhage. It does not often run a rapid course, and tubercles of the larynx are said to be rare.

Symptoms.—The most important clinical results of congenital disease of the heart are dyspnœa and cyanosis; to these anasarca succeeds, and the other results of venous congestion and arterial anæmia which have been detailed in the present chapter.

Cyanosis, or the blue disease (*morbus cæruleus*), was once supposed to be characteristic of congenital cardiac lesions, and to depend on mingling of the arterial and venous streams of blood. So William Hunter originally taught, and he was followed by Meckel, by Gintrac and Bouillaud, by Hope, Williams, Walshe, and Chevers. But it is not an uncommon result of chronic bronchitis, especially of bronchiectasis in children; and, on the other hand, it may be absent for many years, notwithstanding grave malformations of the heart. Still, in its extreme form it is seldom seen except in cases of malformation; and if absent it often appears on exertion or crying.

The question has been much discussed whether cyanosis, if admitted not to be due to actual mixture of venous and arterial blood, depends more upon non-aëration of the blood or upon venous congestion. The latter

view was taken by Louis and Cruveilhier, Rokitsansky, Stillé, and Peacock. Both conditions coexist in most of the cases now under discussion; but of the two, deficient aëration is probably the more constant. See a valuable paper by Dr Lees in the 31st volume of the 'Pathological Transactions.'

Cyanosis is most marked in the lips, the tip of the nose and ears, the fingers and toes; in bad cases the whole face is of a leaden colour, the eyes are bloodshot, and the mucous membrane of the mouth purple in tint. The blueness is increased by exertion, by coughing, and by cold. The fingers and toes are clubbed and the nails incurved. Though the patient is usually comfortable while at rest, exertion brings on dyspnœa. Orthopnœa is less constant and marked than in acquired disease of the heart.

The patient suffers much from the winter's cold, and in marked cases the hands and feet feel chill and clammy even in warm weather. In the slighter cases the body is often well nourished, while the muscular strength and mental faculties are not perceptibly impaired.

Peacock quotes the following graphic description by Dr Wm. Hunter of his original case (1783): "Though he was remarkably thin, he had not the look of being emaciated by consumption; on the contrary, it appeared to be his natural habit. If a man had never seen any of the canine species but the bull-dog, he would be struck at the first sight of the delicate Italian greyhound. This young gentleman put me in mind of that animal, and when I looked at his legs especially, I could not but think of the legs of a wading waterfowl."

In 101 cases analysed by Peacock, he found that symptoms were first noticed at or shortly after birth in 74, within the first year in 15 more, between one and two years after birth in 4, between two and five years in 5, and in the remaining 2 cases at thirteen and fourteen.

The *physical signs* of congenital pulmonary stenosis are as a rule—(1) increased cardiac dulness transversely and to the right from hypertrophy of the right ventricle; (2) a long and loud bellows murmur, systolic in rhythm, basic in position, but often unduly diffused, and frequently accompanied with a tactile thrill. The form of the chest, the seat of the apex-beat, the character of the second sound, and the rate, volume, and regularity of pulse when the patient is at rest, are usually unaffected. But the pulse is liable to be easily rendered frequent, irregular, feeble, or intermittent. Occasionally a diastolic bruit has indicated pulmonary regurgitation; and, perhaps more often, there has been no abnormal sound whatever.

Treatment.—The first indication is to keep up the temperature of the patient's body. A cyanotic child should be wrapped in cotton wool, and the room kept at an equable temperature. If it survives infancy, it should be clothed from head to foot in flannel, and carefully shielded from exposure to inclement weather, even a degree of cold or of wind which is only healthful to an ordinary child. Fits of crying should be as much as possible prevented during childhood, and all exertion should be carefully restricted. Such children instinctively sit quiet by the fireside, although unfortunately they are sometimes of an excitable temper.

Removal to a warm climate when practicable is a most valuable means of treatment.

The convulsive attacks to which cyanotic persons are subject are often relieved by the application of a few leeches to the temples or behind the ears. Palpitation, dyspnœa, bronchitis, and flatulence must be treated by the same measures as are useful in acquired disease of the heart.

CARDIAC DISEASE IN CHILDREN.—Apart from the congenital lesions just described, children are frequently the subjects of acquired disease of the heart, and in them it presents certain peculiarities of practical importance.

The parts involved are almost always the pericardium and the endocardium. Primary disease of the myocardium and lesions of the aortic valves is rare. With the doubtful exception of chorea and, occasionally, of adherent pericardium, there is scarcely any condition but organic lesions of the valves which produces a bruit. Even in cases of marked anæmia, it is very rare to hear murmurs in the heart, the arteries, or the veins. The pulse is naturally frequent in children, and the heart's action is easily quickened by excitement. The apex-beat is usually higher in childhood than in adults, and sometimes a little further out.

Ætiology.—The origin of valvular disease as observed in childhood is in one of the following causes:—(1) Congenital lesions from malformation or intra-uterine endocarditis of the right side. (2) Rheumatism. This is common in children, but the synovitis and pain are so slight and the fever so moderate, that it is easily overlooked. On the other hand, there is great likelihood of the heart suffering, since there is no doubt that fewer children affected with (acute or subacute) rheumatism escape cardiac inflammation than adults attacked in the same way. (3) Chorea. This is often combined with cardiac murmurs, and these are probably organic, but chorea is so often preceded by rheumatism that certainly many, and perhaps the large majority, of these cases are due to the latter cause. (4) Scarlatina. Valvular lesions may not very infrequently be traced to an attack of scarlet fever; but this disease is so often followed by multiple synovitis of, in most cases, true rheumatic character, that here again most post-scarlatinal cases of endocarditis are really due to the latter malady. (5) Occasionally cardiac disease in children has followed measles, enteric fever, smallpox, or diphtheria; but more often dilatation than valvular lesions.

In the great majority of cases, disease of the left side of the heart in children means previous rheumatic endocarditis. Girls are more liable to valvular disease than boys (167 to 81 of Dr Goodhart's cases).

Acute ulcerative endocarditis with infective emboli may occur before puberty, usually as a second attack on the valves previously damaged by rheumatism; but atheromatous degeneration of the valves is unknown in childhood.

Anatomy and locality.—The nodules of coagulated lymph, the puckering, contraction, and adhesion of the valves, are what has been already described in the adult. Parrot (quoted by Dr Eustace Smith) observed minute hæmatomata and fibrous nodules on the mitral curtains in infants dying under a month old; these seem to gradually disappear without leading to further changes.

In children, incompetence is a more common result of endocarditis than obstruction, but the latter is a not infrequent lesion of the mitral orifice after five years old. An apex systolic bruit is the commonest physical sign, and the lesions are probably, in order of frequency—mitral insufficiency, mitral obstruction, aortic regurgitation, and aortic obstruction.

Adherent pericardium is also very common, and quite as grave as mitral disease. The resulting hypertrophy and dilatation of the cavities does not differ from that seen in adults.

Symptoms.—It is remarkable that we rarely see the familiar type of either aortic or mitral disease in children, except in the final stages of

their illness. The aspect of a child with cardiac disease is rather that of phthisis. He is pale and thin, with dilated pupils, a delicate skin, and a quick, easily excited, but usually regular pulse; he has often a short, dry cough, and gets out of breath when he runs.

Sudden death from disease of the heart is very rare in children. The last stage of their illness is marked by dyspnoea and exhaustion, or sometimes by general dropsy.

Prognosis and treatment.—For the most part, moderate valvular lesions are well compensated in childhood, and for many years there may be no symptoms whatever. Even when pallor and dyspnoea on exertion lead to an examination of the chest and discovery of the malady, it is often long before palpitation appears, and longer still before signs of venous congestion are added to those of arterial anæmia. The period of puberty and the five or six years that follow it are very critical, and the greater exposure and liability to the strain of labour and of emotion which adult life brings are unfavourable, so that few who have acquired serious lesions of the valves in childhood reach middle age. Some of the worst cases are those of adherent pericardium.

The general treatment consists—first, in fostering the nutrition of the patient by helping the appetite and digestion, and giving a due amount of meat with, in most cases, the addition of a little ale or port wine; secondly, in protecting him from damp and changes of temperature, while at the same time seeking to increase his power of resistance by constant woollen clothing, by well-ventilated dwellings and bedrooms, and by life as much as possible in the open air; thirdly, in prohibiting violent games, and all but the most equable and moderate bodily exercise; lastly, in giving the heart long periods of physiological rest by sleep.

In these cases residence in the south of Europe during the winter and spring, and, for those who are old enough, a long sea voyage, if possible on a sailing vessel, often prove most salutary.

Of drugs, steel is the most constantly useful. *Digitalis*, in small and long-continued doses, is next in importance. *Strophanthus* has not served the writer so well with children, and sometimes entirely fails to reduce the number of pulsations. In one case of advanced mitral regurgitation with dropsy, in a girl of about twelve, *convallaria* acted as a most useful diuretic after *digitalis* had apparently lost its power.

CARDIAC DISEASE IN OLD AGE.—Valvular disease above sixty is usually due to atheroma, for most of the cases which follow rheumatism do not survive middle life. Aortic valvular lesions are most common in elderly men, sometimes complicated by aneurysm or renal disease, while mitral stenosis is more common in women. Fibrous and fatty degeneration are not infrequent in both sexes. The pulse is usually large, somewhat jerking and tortuous from loss of elasticity in the aorta, even when there is no disease of the sigmoid valves; it is often intermittent. The treatment should not be active, but small doses of *digitalis* are valuable. An occasional blue pill, aloes, senna, and other warm laxatives are indicated, with such cordials as cardamoms, ether, and paregoric.

ANEURYSM OF THE AORTA

Ἀρτηρίας ἀναστομωθείσης, τὸ πάθος ἀνεύρυσμα καλεῖται διαγιγνώσκεται ἐκ τὰ τοιαῦτα παθήματα ἀπὸ τῶν σφυγμῶν τῶν ἐργασαμένων ἀρτηρίων.—GALEN, *De Tumoribus* cap. ii.*

Aneurysm—Definition of the term—Varieties—Anatomy—Pathology—Origin in strain, in syphilis, and in embolism—Symptoms of two kinds.

Aneurysm of the ascending aorta—of the arch—of the descending aorta—Diagnosis—event—prognosis—treatment.

Abdominal aneurysms—Symptoms—origin—diagnosis—treatment.

Dissecting aneurysms—Statistics of aortic aneurysm.

ANEURYSM (ἀνεύρυσμα, from ἀνευρύω, to widen out, to dilate—*Fr.* Anévrisme) is a term used by Galen and Paulus Ægineta to denote what we should now call a traumatic, false or secondary aneurysm. Its Latin equivalent is *dilatatio*, and it was applied by Corvisart to dilatation of part or the whole of a chamber of the heart. The word occurs in its modern restricted application at the end of the seventeenth century (Cook's 'Marrow of Chirurgery,' 1675; Salmon's 'Ars Chirurgica,' 1699; Blancard's 'Lexicon Medicum,' 1702); but aortic aneurysms were first investigated by the great Italian writers of the 18th century, Valsalva, Morgagni, and Scarpa.

An aneurysm may be defined as a circumscribed tumour containing blood, communicating directly with an artery and limited by a tunic, called its sac. It was long questioned whether the term should include cases in which the whole calibre of the vessel is dilated, and whether in the circumscribed form it should depend on the number of arterial coats involved. A mere bulging of one side of the aorta is spoken of as a *dilatation*, whereas a general dilatation of the whole circumference of the vessel, if limited in length, is called a *fusiform* aneurysm. If one side of the aorta causes symptoms by pressure on neighbouring parts, the term "aneurysmal dilatation" is often used, and applies to a case where the symptoms are referable both to the impeded circulation due to dilatation, and also the mechanical pressure of a circumscribed aneurysm. When we speak of "aneurysm" without qualification, we imply the existence of a circumscribed swelling of

* When an artery dilates it is called an aneurysm, and this kind of disease is recognised by the pulsation of the labouring arteries.

the *sacculated* kind, communicating with the vessel by a comparatively narrow aperture.

The division of sacculated aneurysm into two kinds, *true* and *false*, according to the number and nature of its coats, is now discarded, for the terms have been used in opposite senses, and the same aneurysm may in the course of time vary in the number of these tunics.

Scarpa, one of the earlier and most original writers on the subject, ascertained that in most saccular aneurysms some of the coats are wanting; and he called these "true," in distinction from mere dilatations of an artery with all its tunics complete, or "false" aneurysms. Subsequent writers used the terms true and false as indicative of the perfection or absence of some of the coats of the vessel. It is better, therefore, to avoid these terms altogether, or to use the word "false" as synonymous with *diffused*—that is, to denote the case of a ruptured artery when the effused blood has formed out of the surrounding tissues a pulsating swelling with new walls.

Anatomy.—Aneurysms begin in a chronic inflammation, which starts in the deep layers of the intima, and involves the whole arterial wall. Thus weakened, it yields before the constant pressure of the contained blood; and so a bulging occurs which pushes the media and adventitia before it. Before the aneurysm has reached a large size, the intima becomes attenuated or in parts atrophied. It may extend over the whole of the inner surface, continuous with that of the artery; in other cases it ceases abruptly at the neck of the sac, or is only in patches and shreds discoverable over other parts of the interior. Frequently we find the whole intima smooth, but the endothelium is absent, and it is closely connected with the middle coat. While the internal and middle tunics are thinned or destroyed, the adventitia becomes much thickened, and often forms alone the sac of the aneurysm.

In the fusiform aneurysm with smooth walls the blood does not coagulate, but in the circumscribed or saccular form, with its narrow orifice and roughened interior, coagulation is ever ready to occur. This is promoted by the sluggish motion of the blood within it. The aneurysmal sac is filled wholly or partially with fibrin arranged in concentric layers,—the outer thin, hard, pale, and adherent to the arterial coats; whilst the inner, which were bathed with blood during life, are thicker, softer, and darker. This shows that the deposition is due to the coagulation of blood, and not to any exudation from the walls of the sac. Nor does there seem to be any vascular connection between the sac and the fibrous clot. In the cases of rapid cure by pressure the sac is merely filled with a uniform coagulum; so the sac need not be filled layer by layer; indeed the lamination appears to take place in the coagulum after it has been deposited.

Ætiology.—Aneurysms are scarcely ever seen under the age of twenty, and are only occasionally met with in young men, while they are almost as rare in old age as in young adult life. They are by far most common in men in the prime of life (30—50), and are very rare in women at any age.

The process which sets up an aneurysm is a true chronic arteritis or inflammation of the deepest part of the intima, and is quite distinct from fatty degeneration of the endothelium, which is always superficial. Arteritis spreads first to the middle coat, and then ruptures the endothelium of the intima, forming what used to be called an "atheromatous ulcer." The first histological products are leucocytes, and fatty and calcareous degene-

ration follows, producing a "gruel-like" detritus (*atheroma*). (See Dr Moxon's paper in the 16th volume of the 'Guy's Hospital Reports,' Third Series.) Whether the term *atheroma* be used, or Virchow's term *Endarteritis deformans*, or *arterio-sclerosis*, it is important to remember that the process is one of chronic inflammation followed by degeneration, not of primary fatty or calcareous transformation.

The origin of this arterial inflammation is to be sought first in the wear and tear, the strain on the arteries which is caused partly by mechanical stretching—particularly in the artery of the ham, in the subclavian and innominate—partly by varying and excessive blood-pressure. Thus it may occur early in life as the result of excessive stretching of the aortic branches and the femoral arteries in walking or climbing, or in lifting weights, or in rowing; or as the result of great efforts made with the breath held, the thorax fixed, and the muscles contracting to the utmost of their power. The former cases will more affect the arteries of the limbs, the latter the aorta.

Again, the same increased strain from high blood-pressure may be produced in chronic Bright's disease—not by the arterio-capillary fibrosis which affects the smaller vessels, but by the high tension which accompanies this condition, and produces its effects on the aorta and its primary branches. Thus, atheromatous arteritis becomes connected with gout, with plumbism, and with alcoholic intemperance. We cannot say that *atheroma*, still less that aneurysm, is the direct result of Bright's disease, or of gout; but all these morbid conditions are found together, and have more than an accidental connection.

Atheroma, however, appears as a purely senile change due to the normal and necessary wear and tear of the circulation, and this natural arterial degeneration does not appear to lead to aneurysm. In addition to the predisposing disease of the arteries, it seems necessary that a local strain, like that of extreme extension of the knee in walking, should determine the bulging of the weakened arterial coats.

As in other cases, we must consider the tissues acted on as well as the irritant. The amount of strain which sets up extensive *atheroma* in one man has no such effect in another; thus we must regard aneurysm from the point of view of vulnerability of the arterial system, which is probably often hereditary. A sudden strain or violent exertion may be the direct precursor of aneurysm, by producing an actual rupture of the coats. It is a well-known fact that *atheroma* and aneurysm of the thoracic aorta are frequent in those who use their arms in rowing, or climbing the masts of a ship, or hauling ropes. The aortic valves, too, are apt to be affected under the same conditions. These are the causes in operation in lightermen and sailors. Soldiers are also very liable to aneurysm; but it must be remembered that, besides their drill and accoutrements which, no doubt, may strain the great vessels, the lamentable frequency of syphilis in the army may have a share in the result.

That muscular exertion is productive of aneurysm is shown in the greater liability of men than women to the disease; also by the labouring classes being most obnoxious to it, particularly porters, and others who work hard with their arms. Moreover when we find aneurysm or sigmoid disease in a woman, she is usually one of masculine build and doing men's work, or else she has signs of lues.

That *syphilis* may be a cause of aneurysm had long been conjectured,

and there can now be no doubt from careful observation in many countries that it is by no means an infrequent antecedent.* It seems not to produce atheromatous arteritis, but a more patchy and more softening form of inflammation, which leads to aneurysm without extensive deformity and thickening elsewhere. It is allied to but not identical with the endarteritis obliterans described as affecting the cerebral vessels (vol. i, p. 719).

In the smaller arteries acute aneurysms have been demonstrated to arise as the result of the softening arteritis produced by a septic *embolus*. In a case under the writer's care in 1883, ulcerative endocarditis was seen to produce first embolism and then aneurysms of the radial and anterior tibial arteries successively. The patient was a young and otherwise healthy man, and he recovered of the acute disease, having only a well-compensated imperfect aortic valve. Each aneurysm was nearly as large as a cherry. (See Langton and Bowlby's important paper, 'Med.-Chir. Trans.,' 1887.)

Two clinical forms.—Dilatation or fusiform aneurysm of the ascending aorta as the result of atheroma leads to loss of elasticity in this part of the vascular system; the pulse is not sustained in diastole, and the shock on the smaller arteries is greater; hence there may in these cases be a typical "water-hammer" pulse without implication of the sigmoid valves. Moreover the same atheroma frequently befalls the aortic orifice, leading to valvular disease. Clinically this form of "aneurysm" is quite distinct from saccular aneurysm of the aorta, although anatomically they are, as we should expect, often found together. The symptoms of saccular aneurysm depend upon its size and position, and connect it clinically, not with cardiac disease but with new growths within the thorax.

THORACIC ANEURYSM.—It is convenient to treat of thoracic and abdominal aneurysms separately, and to divide the former into those of the root of the aorta within the pericardium, those of the ascending, transverse, and descending thoracic aorta.

(1) If the aneurysm forms a pouch in the sinuses of Valsalva, the valves become involved, their closure may be prevented, a diastolic bruit be produced, and all the other signs and symptoms dependent upon regurgitation follow. These may indeed be the only symptoms, with none indicative of the presence of an aneurysm. The sac never grows to a great size; it usually bursts into the pericardium, and thus causes instant death.

(2) When the aneurysm is situated outside the pericardium its course is very different. It will continue to increase, sometimes without much inconvenience, until its presence is apparent by a pulsating swelling. Unlike aneurysms of the rest of the arch, they tend towards the front of the chest, and therefore do not implicate the important structures which aneurysm of the transverse and descending aorta must do. Previous to its appearance pains in the chest may have been experienced, but they are often absent, and seldom severe. In fact, here symptoms are few and slight, while physical signs are obvious. When it has come forward, it may be recognised as a bulging of the chest, to the right of the sternum and usually in the second space. When the hand is placed over it a distinct pulsation is felt; this is synchronous with the heart's action, as we ascertain by placing the other hand over the apex.

Sometimes the touch detects a thrill (*frémissement*, tactile fremitus),

* In China, among European residents (young unmarried men), aneurysm is said, on good authority, to be very common—much more common than in England, and is probably to be ascribed to the frequency of lues.

and if the swelling projects so as to be grasped by the hand, a distinct expansion is felt, and this is the most important and distinctive sign of an aneurysm. Without it there is little certainty of its nature, for a pulsating tumour may be nothing more than a growth situated over an artery, and if a bruit is present it may be produced by pressure on the vessel; but no dilatation would be felt by the hand which grasps the swelling. The expansion may sometimes be well shown by covering the swelling with a piece of plaster in which a slit has been cut; if this be narrowly watched the slit will be seen to widen at every beat of the sac. If pulsation is felt, but not seen while looking directly at the chest, it may often be clearly observed by placing the eye on a plane with the patient's chest, either by looking over his shoulder when he is in the erect posture, or by stooping to a level with his body when he is supine. Or the stethoscope may be placed on the spot as a lever, and the pulsations recognised as they are magnified at its distal extremity.

On listening with a solid stethoscope, a marked throb or jar is communicated to the ear with each beat of the heart. Sometimes a systolic murmur is heard, but this is by no means always the case, for it depends upon the relation of the sac to the vessel, and is due to the existence of a constricted opening, through which the blood passes into a larger space of the sac beyond. It is very rare to hear a diastolic bruit; if it exists, it must depend upon some peculiar and exceptional circumstance, or it may be a transmitted cardiac murmur from aortic regurgitation. Indeed, it would be strange if the diseased and distended coats of an aneurysm, which have long lost their original elasticity, should be capable of expelling blood from the sac during diastole with sufficient force to cause an audible fluid vein. As a rule the second sound is not only clear, but loud, with a shock or "thud."

The heart is not enlarged as the direct result of saccular aneurysm of the aorta; it is only when an aneurysm exists at the very root that enlargement occurs, as in primary disease of the valves, or from fusiform dilatation of the aorta. The apex-beat is, however, often found somewhat lower than natural, owing to the direct downward pressure of a large aneurysm. Occasionally the tumour presses upon the superior vena cava, and then some enlargement of the veins of the neck may be observed, or the surface of the chest may be seen covered with distended veins. In some remarkable cases a communication has been formed between an aneurysm of the ascending aorta and the superior vena cava, in others between the aneurysm and the pulmonary artery; but the latter event is less rare when the aneurysm is in the transverse aorta. If large, the sac may press upon the trachea or bronchus, and impede the entrance of air into the lung. Sibilus and rhonchus may thus be produced, and if ulceration takes place into the bronchus, hæmoptysis. If the aneurysm approach the axilla, the subclavian vein and the nerves of the brachial plexus may be involved, so as to cause swelling and pain of the arm.

As a rule, however, aneurysms of the ascending aorta push forward and, absorbing the ribs or passing through the sternum, project outside the thorax as circumscribed tumours—sometimes very large and irregularly bossed; often of the size and rounded form of an orange. The skin may become thin and red, but the aneurysm very rarely bursts externally. Blebs may form with thin sanguineous contents, the surface may become discoloured, and at last a raw surface is formed, which from time to time exudes blood. But even then, the elastic corium long resists pressure, and

after alarming hæmorrhage has again and again been stanchèd, the sac usually bursts inwards.

The percussion-note over an aneurysm is of course dull, the extent varying with its size and forward growth.

(3) *Aneurysm of the transverse part of the aortic arch* gives rise to symptoms more numerous and varied than those of the ascending part, while the physical signs are often absent. An aneurysm of this part is usually saccular and produces all the mechanical effects of a tumour. Hence new growths and aneurysms are constantly mistaken for one another, for if the latter be deep-seated pulsation may be altogether wanting. If we consider the close packing together of the aorta, trachea, bronchi, œsophagus, pneumogastric, recurrent laryngeal, and sympathetic nerves, it is evident that any tumour like an aneurysm forming amongst them must interfere with some of these structures. The vertebræ may be eaten away; or the pulmonary artery may be pressed upon and even opened. The parts usually involved, however, are the trachea and left bronchus. These being for some time pressed upon, subsequently ulcerate until the mucous membrane is reached, and an oozing of blood takes place. If the aneurysm contain much fibrin, and the solid portion be in contact with the air-passages, this oozing of blood or occasional hæmoptysis may continue for a long time, for weeks or months, and in some exceptional cases for years. Sooner or later, however, the blood breaks through in large quantities, and causes immediate death by stifling or by syncope.

The most frequent seat of aneurysm of the arch is at the origin of the innominate artery, and this vessel is often dilated so as to form part of the sac. In such cases the right subclavian may be occluded by the external pressure of the aneurysmal sac, or plugged by a detached clot, so as to make the pulse of the right wrist weaker than that of the left.

In such cases a pulsating swelling can often be felt in the root of the neck on the right side.

If an aneurysm forms in the transverse aorta behind the sternum, it will probably soon show itself on the left side as a pulsating tumour about the second left costal cartilage. If there have been no symptoms, this, when first observed, may be mistaken for the left auricle. As in the case of the aneurysm of the ascending aorta, it may be seen and felt to pulsate, and occasionally a bruit is heard.

If the aneurysm grows downwards from the hollow of the arch, no external signs are apparent, and the symptoms are all due to its pressure on the parts which surround it. The most constant symptom is *dyspnœa*, which may arise either from pressure on the trachea or bronchus, or from pressure on motor nerves. In the former case the dyspnœa is more or less constant, and is often accompanied by sibilus, and by blood-tinged expectoration. In the latter the difficulty of breathing is due to paralysis of the vocal cords. It is usually the left vagus which is involved, and it is sometimes found after death closely incorporated with the coats of the sac, and perhaps much thinned, while the muscles of the larynx, especially the posterior crico-arytænoid, are atrophied. The symptoms due to pressure on the vagus, or its recurrent laryngeal branch nerve, differ from those caused by direct pressure on the air-passages in that the difficulty of breathing is paroxysmal, and the cough like that of a croupy child, ringing or "brassy." In the intervals the breathing may be free and natural. If the laryngoscope be used, it will be found that one of the vocal cords,

generally the left, is paralysed and motionless; but sometimes, although only one nerve is involved, both cords become paralysed, causing great difficulty of breathing, and threatening suffocation (*supra*, p. 109). That the left cord is as a rule affected is due to the course of the left recurrent nerve under the arch of the aorta.

Pressure of an aneurysm on the œsophagus may occasionally cause difficulty of swallowing; and cases have been recorded in which an attempt to pass a sound has caused death by rupture of the sac.

Pressure on the vagus or pulmonary plexus may set up an insidious form of *pneumonia*. Frequently after the appearance of febrile symptoms it is found that one lung is becoming solidified; and after death it is found to be hepatised or in a state of purulent infiltration, probably, as Sir William Gull and Dr Budd long ago surmised, by implication of the trophic nerves of the lung.

Pressure on the sympathetic nerve in the neck may cause *myosis*, or contraction of the pupil, in the same way as paralysis of the third nerve causes dilatation. The left pupil is more often found contracted.

Sweating on the same side as the contracted pupil has been recorded in cases of aneurysm by Professor Gairdner ('Edin. Med. Journ.,' 1856), and also by Dr Bramwell (*ibid.*, 1878); but in the latter case the hyperidrosis had preceded the symptoms of aneurysm by years, and in cases of undoubted injury to the cervical sympathetic, as well as of its experimental division in animals, the secretion of sweat is checked, not increased, on the affected side when the pupil is contracted (cf. 'Journ. of Physiology,' vol. viii, p. 26).

The writer had a marked case of this unilateral sweating on the same side as the contracted pupil in a patient who died in John Ward of aneurysm and sigmoid regurgitation in December, 1892.

Pain is a more constant and severe symptom in aneurysm of the transverse than in that of the ascending aorta; indeed, it is a pain in the chest or down one arm which first brings the patient to the physician.

It is very rare for aneurysm to run its course without pain, and in these rare cases it is most apt to appear early as a pulsating tumour.

The *pulse* sometimes indicates the existence of an aneurysm of the transverse arch, by the left pulse being smaller than the right. This may arise from three causes: the left subclavian artery may be compressed by the sac from without; or of the sac involves its origin from the aorta, its mouth may be closed by fibrin; or again, and more frequently the artery is open, but the aneurysm, as we saw before, in the case of the right subclavian, prevents pulsation in the vessels coming off from it. It must be remembered that when the blood is delivered by the ventricle to the aorta, the elasticity of the latter does not prevent each shock being felt in the distant arteries; but when the aorta is converted into a large sac the impulse of the heart is lost in this space, and the blood flows out through the efferent vessels in a more continuous stream. A case once in Guy's Hospital exemplified this: the pulses at the wrists became quite imperceptible, but the warmth of the hands and their vascularity showed that the pulse flowed as before.

It is the exception, however, for the pulse, as in this instance, to cease completely; usually it becomes more feeble and the artery smaller in one wrist than in the other. When the aneurysm involves the innominate, the right radial pulse is, of course, enfeebled; if the left subclavian, the left.

The sphygmograph is found useful in demonstrating on paper the difference between the normal tracing from the one wrist and the imperfect tracing from the other.

Retardation of the left radial pulse or in the left radial and carotid, compared with the arteries of the right arm and neck, may sometimes help to the diagnosis of an aneurysm of the transverse arch between the innominata and left carotid.

(4) Aneurysms of the *third part* of the arch and of the *descending thoracic aorta* may be taken together. They are almost always saccular, not fusiform dilatations as is often the case in the ascending aorta. They soon involve the vertebræ, particularly the fourth and fifth, which they erode, and at last expose the spinal nerves, and before this the sac may press on the left intercostal nerves. Pain, therefore, is one of the commonest symptoms, and often the earliest. It is sometimes confined to the course of a particular intercostal nerve, and so the seat of the disease may be accurately determined. A descending thoracic aneurysm may also involve the lung, or press upon a bronchus. The œsophagus, too, is more often compressed by aneurysm of the descending than the transverse part of the arch, and by the continuation downwards of the thoracic aorta. Cases of compression of the thoracic duct by an aneurysm of the descending aorta are also recorded, in which great emaciation followed. The aneurysm sometimes makes its way backwards, erodes the ribs, perforates the thoracic wall, and appears as a pulsating tumour to the left of the vertebræ in the back. It may even enter the vertebral canal and, compressing the cord, produce paraplegia (cf. vol. i, p. 614).

Diagnosis.—The recognition of a thoracic aneurysm is sometimes easy and obvious, sometimes extremely difficult.

When a pulsating tumour can be seen and felt, it must be either an aneurysm, or an abscess or tumour which receives an impulse from the heart, or an extremely vascular (in fact erectile) growth.

When no tumour or expansion or even pulsation can be discovered, we must depend entirely on the symptoms of pressure above detailed. Of all of them pain is the most constant, and should never be carelessly put down to "neuralgia," still less to "hysteria" or to malingering. When a man in middle life and sound in mind complains of a constant fixed pain in the chest, the probabilities are that he has an aneurysm.

The most frequent difficulty, after due care and observation have been used, is to distinguish between an aneurysmal and a solid intra-thoracic tumour. Here we are helped by the sex and age of the patient. Aneurysm is unlikely in a woman, and is very rare in a patient under thirty, while its presence is probable in a labouring man, in a soldier or a sailor, and in a case where we find (with or without a history) evidences of syphilis. On the other hand, enlarged lymph-glands above the clavicle, or in the axilla, indicate a new growth, and so does pleural effusion; pleurisy and hydrothorax are not produced by an aneurysm.

A valuable symptom of aneurysm of the transverse aorta we owe to Surg.-Major Porter and the physicians of the General Hospital at Montreal. When the trachea is drawn firmly and steadily upwards, the pulsation of the aneurysm on the left bronchus causes a rhythmical pull, which is readily appreciated and very characteristic. The best plan is to stand behind the patient seated in a chair, and with both hands gently draw up the cricoid cartilage until unmistakable "tracheal tugging" is felt. Carefully per-

formed, the manœuvre causes no pain to the patient, and the sign does not appear to be simulated by violent functional palpitation, nor by the collapsing pulse of aortic regurgitation. If absent, it probably denotes that an aneurysm, if present, does not affect the hollow of the aortic arch.

The use of the Röntgen rays is a valuable recent addition to our means of diagnosis.

Results and prognosis.—It is very rare for aneurysm to undergo spontaneous involution. That this is possible is proved by some solid and cured sacs being accidentally discovered in the deadhouse or dissecting-room. They are usually found in bodies wasted by phthisis or cancer (Moxon, 'Guy's Hosp. Rep.,' vol. xii). Occasionally an aneurysm may in its growth compress its own artery, and thus cure itself. With such rare exceptions, when the dilatation has once begun it goes on until the sac ruptures. Even if an aneurysm fills with clot and becomes contracted and solid, fresh sacs are apt to be pushed out from a neighbouring atheromatous surface.

Aneurysm of the first part of the ascending arch opens into the pericardium or into the pulmonary artery. In the latter case death may be long delayed and peculiar murmurs may be heard.*

Aneurysm of the ascending aorta opens into the superior vena cava or the right auricle or the right pulmonary artery.

Aneurysm of the rest of the arch usually opens into the trachea or the left bronchus, rarely into the œsophagus or left pulmonary artery.

Aneurysm of the descending thoracic aorta sometimes opens into the lung, but more often bursts into the pleura by a gaping fissure, which never allows oozing as a mucous or cutaneous surface does, but empties the whole sac at once and kills by sudden syncope.

A valuable paper by the late Dr Peacock in the 'Pathological Transactions' for 1868 (vol. xix, p. 111), gives a table of 33 cases of aneurysm of the aorta which had ruptured into various adjacent cavities.

The duration of thoracic aneurysms after they are recognised must be reckoned by months rather than by years; but they sometimes grow for two years or even more before they prove fatal, and an accidental compression may, as a rare exception, lead to a natural cure.

Death often results from disturbance in the pulmonary circulation, causing congestion of the lung or œdema, sometimes from disease of the left sigmoid valves, and occasionally from some intercurrent disease. If none of these events intervene, rupture and death by sudden or by repeated hæmorrhage is the natural end of the disease.

Treatment.—Our object in treating aneurysm of the aorta is the same as that aimed at in treating aneurysm of a limb—to retard the movement of blood in the vessel and favour the deposition of solid fibrin on the walls. This is more likely to be effected when the opening of the sac is small and the interior rough.

One method of cure is to lessen the rapidity and force of the circulation by keeping the patient absolutely at rest in the recumbent posture, and subjecting him to a strictly limited diet.

The soundness of the principle on which this treatment is founded was well shown by the late Dr Moxon in an article in the 'Guy's Hosp. Reports' for 1866. He there remarks on the few and perhaps doubtful cases of

* When an aneurysm of the ascending arch presses on, or opens into, the pulmonary artery, inflammation may ensue, which renders the pulmonary valves incompetent. Of this Dr Pitt found eight examples in the Guy's Museum.

spontaneous cure of aneurysmal tumours described by Porter (and also referred to by Heberden), and quotes Liston's case of an axillary aneurysm cured by a second sac forming above it and obliterating the subclavian artery. He then gives an account of a case of aortic aneurysm cured by the supervention of cancer, and quotes six cases from the 'Pathological Transactions' in which consolidation of an aneurysm was found after death from phthisis, or other causes of anæmia and wasting.

The method of cure first practised by Albertini and Valsalva, was bleeding the patient repeatedly, and keeping him on the smallest possible amount of food. This treatment was revived by Tufnell in a modified form with remarkable success. His object was to reduce the intra-vascular pressure, and to keep the blood in a highly coagulable state. He allowed three meals a day* and a small amount of fluid, with absolute rest for at least two months. The value of rest in the recumbent posture is shown by the fact that in one of his patients the difference of the number of beats of the heart between the sitting and recumbent position was thirty-five per minute, equal to more than 50,000 in the twenty-four hours. It must be remembered that his experience was chiefly with peripheral aneurysm.

Of the two constituents of this treatment, *rest* and *low diet*, the former is the more efficient and indispensable. The patient must not sit up to take food, nor rise to have his bed made or to relieve his bowels. He should not even suddenly turn over, and should move his arms as little as possible. Sleep should be procured when necessary by bromides or chloral, or, if there is much pain, by opium or injection of morphia. Opiates are useful in the early part of the treatment by stilling hunger as well as pain, but after a few days the pain usually subsides and the patient sleeps well.

The restriction of solid food is comparatively easy; that of liquid is more difficult. Thirst may be relieved by ice, by effervescing lozenges, or by sucking slices of lemon. While aiming to reduce the water taken to half a pint a day, the diminution must be gradual, and must sometimes stop short of that limit.

After two or three weeks of this regimen the pulse is apt to become again rapid and feeble, with palpitation of the heart. The strict dietetic treatment must then be relaxed, opium must be given for a time, and then the food cautiously diminished again. In certain cases all attempts in this direction are baffled, and it is then wise to let a patient eat and drink moderately, and to trust to the effects of absolute rest.

In favourable cases pain rapidly subsides and then disappears, pulsation is less violent, the swelling diminishes, and after two or three months there may be neither physical sign nor symptom of the aneurysm. This occurred in a patient under the writer's care, after the front of the thorax had been perforated in two places. Both the sacs filled with clot, ceased to pulsate, and gradually diminished in size until they were no longer to be felt. (For sequel, see below.)

As soon as the tumour has disappeared the patient's food should gradually be increased; and next he should be allowed to sit up. Lastly, he may be restricted from laborious occupations only, and suffered to walk about as usual; but avoiding bodily efforts or mental emotion.

* Tufnell's bill of fare was as follows:—For breakfast, two ounces of bread-and-butter and two ounces of milk or tea; for dinner, three ounces of mutton, three ounces of potatoes or bread, and four ounces of claret; for supper, two ounces of bread-and-butter and two ounces of tea: the total *per diem* being ten oz. of solid food and eight oz. of liquids.

Of drugs, neither digitalis nor strophanthus seems to be followed by any benefit, and the same may be said of lead, ergotine, and many other reputed remedies; the only one which can be said to be efficient is the *iodide of potassium*. This is given in large doses, fifteen to thirty or even sixty grains, three or four times a day for several weeks; and sometimes with marked success. Under its use the aneurysmal sac becomes hard, and the pain subsides.*

There is no evidence that potassium iodide has a specific influence on syphilitic arteritis, or that it increases the tendency of the blood to coagulate. But we know that, like other potash salts, it depresses the action of the heart, brings down arterial blood-pressure, and thus mechanically favours the formation of fibrin. Dr Balfour published some remarkable cases of benefit by treatment with iodide, Dr Bramwell recorded several others in the 'Edin. Med. Journ.' of 1878, and it is very generally used at the present time. But like many remedies, iodides were most successful when first introduced. The present writer had some excellent results, but they have not improved with further experience.

The best of these was an elderly man who, after most patiently keeping the horizontal position, living on restricted food and liquid, and taking potassium iodide in large doses, recovered as above mentioned. All his previous symptoms disappeared, the two pulsating tumours subsided, and at last disappeared, and after more than three months he was allowed to get up, and at last returned home to all appearance cured. He continued well for nearly two years, but symptoms of deep-seated thoracic pain then came on; he was re-admitted to hospital, and very soon died from sudden and profuse hæmoptysis. At the autopsy the original aneurysm of the ascending arch was lined with thick, firm fibrin, and the two sacs which had perforated the chest-wall were firmly contracted and solid. But the disease had spread to the transverse part of the arch, and there had softened and burst into the left bronchus.

This seems to be the great difficulty. Aneurysm of the aorta is not as that of the popliteal artery often is, a local disease in an otherwise healthy artery; but there is almost always extensive atheroma, or syphilitic arteritis, which is ready to form fresh pouches when the original one is occluded by coagulation. Still the iodide is the only effectual drug we have, and when a patient will not or cannot carry out the treatment by absolute rest and restricted food, its free exhibition certainly diminishes pulsation and relieves pain.

Wooldridge, from his experiments on coagulation in the lower animals, concluded that a diet of chiefly fatty constituents is theoretically best adapted to produce the formation of clot in an aneurysmal sac. Dr Wright's proposal to treat aneurysm by calcium chloride in fifteen to thirty-grain doses, has been followed with apparent success at Netley, and by Dr Whitla at Belfast. Lancereaux' treatment by subcutaneous injection of gelatin has proved beneficial in some cases.

Sometimes a case has gone too far for treatment before it is seen, or the patient may be intractable; or the treatment, after success for a time, is followed by a more obstinate return of the disease; or the tumour threatens immediate rupture. To relieve urgent symptoms which cannot

* This shows that the pulsation of the sac produces symptoms not due to the tumour alone; for when the latter has become hard and inert they disappear. It is true that the cured aneurysm is smaller than the active one, but this alone will not account for the effect. In cases of popliteal aneurysm the pain and tenderness immediately cease after the arrest of the circulation through the sac.—C. H. F.

wait for the effect of rest, and indeed prevent it, the most efficient remedy is small and, if needful, repeated *bleeding*. Four or six ounces taken from the arm, as recommended by the late Dr Hughes Bennett, rarely fail of effect. More than once the writer has seen a patient who had suffered many days and nights of unrelieved pain and sleeplessness, with frequent attacks of agonising suffering—after ice-bags, morphia in large doses, and other means had failed—obtain complete relief and sound sleep after a single venesection.

For the radical cure of thoracic aneurysm, when the plan of treatment above described is inapplicable or has failed, various local measures have been attempted, but not with much success. One of these is *galvanism*. The poles of a battery have been passed into the sac so as to produce coagulation of the blood. The needles must be well insulated, and of course must not touch; but probably the best plan is to use the positive pole only, so as to procure the firm non-gaseous clot which forms on the anode, and to place the cathode as a large wet sponge on an indifferent part in the neighbourhood. This practice was successful in the hands of Ciniselli, and partly so in those of the late Dr John Duncan ('Edin. Med. Journ.,' April, 1866), but is now generally abandoned.

Another plan, introduced by the late Mr Moore, is to pass several yards of fine iron wire into the sac. At Guy's Hospital this has been tried with imperfect success, and horsehair has also been used. The danger is setting up inflammation or sloughing of the sac. Only two patients survived the operation more than a fortnight; and about twenty fatal cases are recorded.

In one case under the writer's care, this plan was adopted in fear of rupture. The effect was to produce inflammation of the sac, to increase the patient's sufferings, and to hasten his end.

Ligature of the distal vessels has been several times performed, and in some cases with temporary benefit.

Prof. Macewen, in an able paper on the treatment of aneurysm, has advocated the introduction of needles into the sac, so as to irritate its internal surface, and produce a firm parietal clot ('Brit. Med. Journ.,' Nov. 15th, 1890). The results in three cases were very satisfactory.

ABDOMINAL ANEURYSM.—Aneurysms of the abdominal aorta occur most frequently (in 133 out of 177 cases collected by Sibson) just below the diaphragm, near the coeliac axis, which is often itself involved; they are much less common at the origin of the superior mesenteric artery, and are seldom found lower than this point. The form of aneurysm here, as in the descending aorta in the thorax, is always saccular. Its anatomy agrees with what has been already stated of thoracic aneurysm; and it also is most often seen in elderly men with general atheroma of the arteries. When it occurs in younger men or in women, it is in most cases due to syphilitic arteritis.

Symptoms.—The earliest symptom of abdominal aneurysm is abdominal pain, dull and aching in character, or piercing and paroxysmal. Patients sometimes describe it as encircling the body like a girdle. It is probably due not to the nerves involved in the sac, but to those pressed on by the pulsating tumour, or to erosion of the vertebræ.

If the aneurysm lies in the upper part of the abdomen, pulsation will be usually felt a little to the left of the median line. A distinct tumour under the hand, and communicating obvious movements to the stethoscope, is not infrequently detected; but even then it is only an expansion that is felt,

when the diagnosis of aneurysm is established. A thrill can often be perceived; and a systolic bruit will almost always be heard on auscultation.

If the aneurysm is lower down, it can more easily be grasped and its nature made out. If it grows backwards, it may occasionally be detected in the loin, both by its pulsation and by a bruit being heard; and this bruit cannot be produced by pressure of the stethoscope, as is frequently the case when it is heard in front in a thin and anæmic subject. Various other symptoms may be present owing to the pressure of the sac. Actual obstruction of the intestine has been recorded; but the constipation which is frequently observed is not due to this. A very careful examination of the femoral arteries will sometimes show a slight retardation of their beat as well as a diminished tension.

The result of an abdominal aneurysm is generally rupture; this may take place directly into the peritoneal cavity, causing instant death, or behind the peritoneum, whereby the blood becomes effused into the areolar tissue, and a coagulum is formed for the time. This new or false sac soon, however, gives way, with the result of fresh and probably fatal hæmorrhage. In very rare cases the coagulum becomes hard, and, being incorporated with the original aneurysmal sac, a cure is effected. A celiac aneurysm sometimes opens into the stomach, causing profuse hæmatemesis and more or less sudden death, or into the transverse colon. Other exceptional terminations of abdominal aneurysm are sometimes met with, as in a patient in Guy's Hospital, in whom a loud whizzing bruit was heard in the lower part of the back, and who also had dropsy of the lower extremities. After death a small aneurysm was found communicating with the vena cava, so as to make an aneurysmal varix; the venous blood was retarded in its flow upwards, and so the dropsy was produced.

Ætiology.—The causes of abdominal aneurysm are the same as in the thoracic form. Violent exertion is no doubt a frequent cause, and this is the reason why it is so much more common in men than in women; for in women it is exceedingly rare. In several cases there has been well-marked syphilis, and no doubt this is a frequent cause, particularly in women. A case was recorded at Guy's Hospital by the younger Babington as early as 1853; the patient was a prostitute, and under treatment for syphilis, when she died suddenly from rupture of an aneurysm of the celiac axis.

Diagnosis.—This is not always easy, since pulsating tumours may exist in the abdomen which are not aneurysmal, and murmurs may be discovered in the abdominal aorta without disease of its coats. In anæmic women especially pulsation in the abdomen often occurs, and bruits are heard, which suggest aneurysm. Again, a tumour close to the aorta may pulsate, and a bruit may be heard, but these two signs do not warrant the diagnosis of an aneurysm; nothing but the rhythmical expansion of the sac can do this. If the patient places himself on his hands and knees, and the tumour is felt, any falling forwards would be in favour of a morbid growth; moreover, pulsation conducted from the aorta to an adjacent tumour would disappear, while an aneurysmal pulsation would still remain. A murmur audible over the first and second lumbar vertebræ is a very characteristic symptom.

Treatment.—The general treatment of abdominal aneurysm is the same as that of thoracic—restricted diet, absolute rest, and potassic iodide; but an opportunity sometimes occurs of using mechanical pressure as a local method. The plan was first successfully carried out by Dr Murray, of

Newcastle, in 1863, and has since been followed by many others. He found that a very short period may be sufficient to cause coagulation in the sac. Unfortunately the more common position, at the coeliac axis, is too high to allow room for any instrument being applied above the tumour. In Dr Murray's first case ('Med.-Chir. Trans.,' vol. xlvii, p. 187) pressure was used for two hours, and again for five. The aneurysm was rapidly cured, and the man remained well for six years afterwards. It was found after death that the aneurysm came off from the aorta at the origin of the inferior mesenteric artery, which had been obliterated, and the circulation carried on through collateral branches. This case was followed by Dr Greenhow's, in which one application was not sufficient; pressure was repeated again and again for a week, and at last a perfect cure resulted. Durham reported a case about the same time of an abdominal aneurysm cured by pressure, which was kept up with the patient under chloroform for ten hours. Pulsation of the sac ceased, and the circulation also stopped in the femoral arteries, and the legs became cold. Pulsation afterwards returned in the aneurysm, but in a less degree. The treatment was continued, but pulsation was not completely arrested in it for a month, when perfect recovery at last ensued.

Another case occurred in Guy's Hospital where pressure was kept up for some hours on the *distal* side of a high aneurysm. No apparent effect upon it was produced; but collapse came on, and in twenty-four hours the patient died of peritonitis. The duodenum was found bruised and covered with lymph; but the sac contained a coagulum which appeared to have been deposited during life.

The bold plan has also been adopted of opening the abdomen, exposing the aneurysm, and inserting iron wire (Pringle and Morris, 'Med.-Chir. Trans.,' 1887).

Dissecting aneurysm.—Instead of expanding into an aneurysm, the aorta sometimes ruptures from disease. It is rare for the whole of the coats to give way simultaneously; more usually the intima is lacerated, the blood then finds its way between the tunics and produces a "dissecting aneurysm," finally the adventitia ruptures, with fatal result. When the blood is effused in this manner, it tears asunder the layers of the middle coat, so that part is found united with the intima and part with the adventitia. The following cases illustrate this remarkable form of false aneurysm.

A former sister of Petersham Ward, about sixty years of age, was seized a month before her death with a violent pain across the chest and abdomen; her heart beat quickly and tumultuously, and she seemed to be dying. She rallied, however, and in a few days was doing duty in her ward. She had another similar attack and again quickly recovered; but two days afterwards she fell dead whilst dressing. The autopsy showed the pericardium to be full of blood, which had proceeded from a rent at the origin of the aorta. On the inner side the rent was seen to be an inch long just above the valves, and through this blood had passed between the coats of the vessel; the external coat was ruptured, but this did not correspond to the internal opening. Some distance above this, and at the beginning of the transverse arch, was another fissure, an inch longer and older in date; it had smooth edges connected by bands, and was quite healed. Proceeding from this was effused fibrin in the coats of the artery, which were separated from one another to the end of the abdominal aorta, in the whole circumference of the vessel.

Another case was that of a patient of Dr Fagge about sixty years old. After returning from the City and eating his dinner, he was seized with a severe pain in the chest, which continued all the evening until he went to bed. He was found afterwards dead at the side of his bed. The autopsy showed the pericardium full of blood, which proceeded from a fissure in the aorta. A laceration of the inner coat was seen to have occurred half an inch

above the valve, and ran almost completely round the vessel. Blood had passed through and separated the coats throughout the arch, the descending thoracic, and the abdominal aorta. The clot was quite recent. The laceration in the outer coat was at right angles to the internal one; it was an inch and a half long, and opened into the pericardium.

A third case was reported by Dr Fagge in the 52nd volume of the 'Med.-Chir. Trans.' It occurred in a gentleman aged seventy-one, who had long suffered from signs of cardiac disease, and died very gradually. The rupture in the intima was less than an inch above the valve, and opened into a large aneurysm which communicated again with the aorta in the third part of the arch; but the false channel continued side by side with the descending aorta to beyond its bifurcation, and it opened into the common iliac arteries.

Dr Peacock, who thoroughly investigated the subject of dissecting aneurysm ('Path. Trans.,' 1863), speaks of a variety occasionally met with, in which a long time elapses between the rupture of the internal and external coats, so that a distinct pouch may form like an ordinary aneurysm, and become in time lined by a smooth membrane. The third of the above cases is an example of this rarer form.

Statistics.—During the ten years from 1875 to 1884 inclusive, there were ninety-two cases of death in Guy's Hospital from saccular aneurysm of the aorta, or nearly 2 per cent. of the total autopsies. An abstract of the *post-mortem* records was made for the writer by Mr Alfred Parkin, now of Hull, and eight cases occurring next after the 92 have been added so as to make the number a hundred.

As to the *sex* of the patients, 93 were men and only 7 were women.

As to their *age*, one patient was eighteen at the time of his death, 5 were between twenty and thirty, 29 were between thirty and forty, 33 between forty and fifty, 25 between fifty and sixty, 3 between sixty and seventy, 1 was seventy, and 1 (a woman) was eighty-three.

All the cases were *saccular*, but in sixteen there were one or more other pouches, some of which were obsolete and solid; and in several others there was more or less diffused dilatation of the aorta. In three cases the inner coat only had ruptured and had formed a *dissecting* aneurysm.

The part of the aorta affected was in 55 cases some part of the arch, and in 28 of these in the ascending part before the origin of the innominate artery; in 7 it was the descending thoracic aorta; in 2 the sac was just at the orifice in the diaphragm, and in 15 it was below that boundary, 11 above and 4 below the origin of the superior mesenteric artery. It is, however, often difficult to decide where a bulky sac, with perhaps some diffused dilatation around it, should be reckoned to belong.

In 47 cases there was rupture of the sac, including six in which it had opened into the pulmonary artery. In 52 the sac had not ruptured, and death was caused by pneumonia, bronchitis, or pulmonary oedema, by syncope, by embolism, or by some independent disease.

Of the thoracic cases in which rupture occurred, the sac burst into the right pleura in 4, into the left in 7; into the trachea in 4, the left bronchus in 4, and the left lung in 1; into the œsophagus in 4, and the posterior mediastinum in one; into the pericardium in 4, and into the left auricle in 1, beside the six above mentioned into the pulmonary artery.

The abdominal aneurysms ruptured nine times into post-peritoneal tissues, once into the peritoneal cavity, and once into the stomach.

In the whole number of 100 cases there were none of external rupture, although in two the threatening signs described above had appeared.

The following figures are taken from a series of cases of aneurysm of the

aorta above the diaphragm which have been under the writer's care, and of which he has sufficient notes. Some of them were in his wards during the ten years above mentioned, and some died in hospital, and so would appear in Mr Parkin's numbers from the *post-mortem* records. But these are only a few ; the rest begin with the year 1865 and come down to the year 1895 inclusive. They all concern patients seen alive, and are thus clinical as distinguished from anatomical records. The number of patients is 70, 59 with aneurysm of the arch of the aorta, and the remaining 11 with the abdominal aorta affected.

Of the 70 patients only three were women, all those with abdominal and 56 of those with thoracic aneurysm being men.

The ages of the patients with thoracic aneurysm were as follows :—The youngest was twenty-eight, and another (called a young man) was also under thirty. Each of these patients had suffered from chancres, and one certainly from secondaries. Fourteen men and one woman were between thirty and forty, and the youngest of these, under thirty-one, had contracted syphilis. Seventeen men and one woman were between forty and fifty, 10 men and 1 woman were between fifty and sixty, 2 men were between sixty and seventy, and 1 was seventy-two.

The ages of the 11 patients with abdominal aneurysm were : 2 twenty-eight, who both had certainly constitutional syphilis ; another under thirty, also a case of syphilitic arteritis with perforation ; 3 between thirty and forty, 4 between forty and fifty-five, and 1 an old man of eighty, who came under treatment for eczema ; an aneurysm of the abdominal aorta high up was discovered by accident, for it gave him no pain ; and he left the hospital apparently in perfect health.

Of the 70 cases, twelve only ruptured : 2 into the pleura, 1 into the left auricle, 1 into the œsophagus, 2 into the pulmonary artery, 2 into the trachea, 1 into the left bronchus, 1 into the peritoneum, 1 into the pericardium, and 1 into the inferior vena cava (' Path. Trans.,' May 18th, 1880, vol. xxxi, p. 85). The other 58 were relieved under treatment or died at last from dyspnœa, from pressure on the trachea, bronchitis, pneumonia, acute nephritis, or other intercurrent disease. One died after ligature of the common carotid, 1 after galvano-puncture, after inserting wire into the sac, and 1 after an attempt to compress it through the abdominal walls.

In 2, an aneurysm of the ascending arch ruptured into the tissues of the right breast, but did not break through the skin ; and in 1 an aneurysm immediately above the coeliac abscess burst into the left lumbar muscles. This caused great relief, but some months later it eroded the last dorsal and first lumbar vertebræ so as to cause paraplegia ; and finally broke into the peritoneum.

The majority of the patients were either soldiers, sailors, or men who used hammers. As above noted, most of the younger men had suffered from syphilis ; and the 3 women had probably also, for one had repeatedly miscarried, and her husband was syphilitic ; another had definite history of secondary lesions several years before ; and the third showed pigmentary choroiditis in both eyes.

DISEASES OF THE ALIMENTARY TRACT

AFFECTIONS OF THE MOUTH, THE THROAT, AND THE GULLET

Τοῖσι μὲν μικροῖσι καὶ νεογνοῖσι παιδίοισιν, ἄφθαί.—HIPPOCR., *Aph.*, iii, 24.*

"In Anginam ego me nunc velim verti, ut veneficæ illi fauces prehendam."
PLAUT., *Most.*, i, 3, 61.

"Interminato cum semel fixæ cibo
Intabuissent pupulæ."

Hor., Ep. v.

Affections of the mouth—Vesicular ulcers of the tongue and lips—Ulcer of the palate—Phagedænic ulcerative stomatitis—Gangrenous stomatitis or cancrum oris—Thrush—Aphthæ—Ptyalism—Xerostomia.

Affections of the fauces and pharynx—Sore throat: catarrhal, ulcerative, and herpetic inflammation—Subacute follicular tonsillitis—Acute tonsillitis or quinsy—Hypertrophy of the tonsils—Granular pharyngitis—Adenoid growths of the naso-pharynx.

Diseases of the œsophagus—Minor disorders—Spasmodic stricture—Simple stricture—Cancerous stricture—its anatomy, symptoms and event.

THE diseases of the alimentary canal vary in their pathology more even than those of the brain or the chest. The only classification attempted will be the anatomical or topographical one of dealing with the several organs as they are most prominently the seats of disease; and we will begin with the diseases of the alimentary canal above the diaphragm.

AFFECTIONS OF THE MOUTH.—*Simple or herpetic ulcers.*†—Among the trivial affections to which children, and sometimes adults, are liable, is the formation of minute, shallow, round or oval ulcers in the mouth. They begin as raised white spots, looking like vesicles; but in a few hours the roof is lost, apparently as a consequence of maceration in the buccal mucus and saliva. The ulcers which result have an ash-grey or yellow surface and a bright red border; they are as large as a pin's head or rather larger,

* *Synonyms.*—Aphthous ulceration of German and of some English writers—Follicular, vesicular, or herpetic ulcers—*Ulcera mitia familiaria*—*Herpes oris*.

† *Aphthæ* affect new-born infants and young children.

painful, and very sensitive to the contact of particles of food, especially sugar and salt. A frequent seat is within the lower lip, especially where it joins the gum; they also occur upon the mucous membrane which lines the cheek, and perhaps most often on the tongue. Some persons are more liable than others to these little ulcers, and are long troubled with them at intervals of a few weeks or months. Their presence is attributed by children to "telling stories," and by mothers to a "disordered stomach;" their true cause is unknown. In a day or two these ulcers heal of themselves without treatment, but touching them with a stick of lunar caustic removes the pain at once.

Single ulcer of the palate.—In marked contrast with this affection is one which is described by Vogel (in 'Ziemssen's Handbuch') as occurring in weakly infants, especially those brought up in lying-in or foundling institutions. It consists in the formation of a single flat ulcer at the back of the hard palate, just where the velum joins it. There is not usually any tendency to spread deeply, but neither is there any disposition to heal, and the ulcer remains until the child's death, which usually results from diarrhoea.

*Phagedænic and ulcerative stomatitis.**—Another form of ulceration of the mouth is a grave disease. It chiefly affects the gums, the edges of which are reddened and swollen, more or less detached from the teeth, and finally seem to break down into a grey pulp. So complete may be the destruction that the alveoli are sometimes exposed, and the teeth become loose and fall out. In the worst cases the whole of the lining of the cheeks and lips is occupied by ash-coloured ulceration, and sometimes the tongue also, which is swollen, marked at its edges by the teeth, and thickly furred. A large quantity of fluid escapes constantly from the mouth, running out upon the pillow while the patient is asleep. All movements of the mouth are painful, and food is taken with much difficulty, while the breath is horribly fœtid.

At one time this affection was not infrequent among the patients of Guy's Hospital, in children between the second dentition and puberty; once two sisters came with it at the same time. Among the soldiers of the French army it used to be frequent, occurring epidemically when they were overcrowded in close quarters. It probably depends on some local infective microbe, but this has not yet been ascertained. Bergeron, who described the disease in 1859, is said to have inoculated himself successfully with it in the lower lip.

In a milder form the same condition is not uncommon in children or in adults. Ulceration of the buccal mucous membrane, with spongy and bleeding gums, but without other signs of scurvy, is ascribed by dentists to the fluids of the mouth being acid instead of alkaline, and the writer has found this to be verified by test-paper in some cases of the kind in children.

The remedy for ulcerative stomatitis is chlorate of potass, which may be given in ten-grain doses at frequent intervals, dissolved in water. Or lozenges containing the salt, or Wyeth's compressed tablets, may be used with the object of securing its local action upon the mucous membrane, although this appears not to be so essential as its absorption into the blood. It is surprising in how short a time the affection is brought to an end; within three or four days the diseased parts begin to show a clean, healing

* *Synonyms.*—Phagedænic gingivitis—Putrid sore-mouth—Stomacace, a French term, "mouth-ill," often applied to scurvy.

surface. An example of this action in an adult was given by the writer in the 50th volume of 'Virchow's Archiv' (1870), p. 462, where, under the title "Gingivitis," three cases of ulcerative stomatitis are related.

Gangrenous stomatitis* is a disease almost entirely confined to young children. It differs from the last-described affection in the fact that ulceration, or even phagedænic stomatitis, begins in the mucous membrane, whereas noma begins as a slough in the submucous tissues, commonly in the cheek or the lower lip. Its cause is unknown, but it often follows measles or other exanthems. Its treatment consists in destroying the diseased tissue by strong nitric acid or other surgical means, and in supplying stimulants freely.

In a case seen by the writer in a little girl of twelve, convalescent from measles, no caustics, though early and freely applied, checked the progress of the disease, which proved fatal in a few days by hæmorrhage. Even when recovery ensues there is often dreadful deformity of the face and retraction of the lips.

Thrush.†—The term *aphthæ* is applied in England to a condition of the mouth, different from what is known abroad as *aphthous ulceration*. The latter corresponds to the *herpes oris* above described, the former is applied to the affection of children known by the vernacular term "thrush."

When thrush is setting in, the mucous membrane lining the cheeks and mouth becomes redder than usual, hot, and painful. Soon minute milk-white spots appear upon its surface. These rapidly increase in size, and run together; and in a day or two the whole surface may be covered with a nearly uniform adherent layer. At first there is some difficulty in detaching the white material; but after a time it becomes loose, and can be peeled off in large flakes without any bleeding. Microscopically it consists of layers of squamous epithelium, mingled with the spores and mycelium of a fungus.

This fungus is commonly known as the *Oidium albicans*, a close ally of *Oidium lactis*, which is the active agent in the souring of milk. The presence of oidium in the mouth is not peculiar to cases of thrush. It has been found upon diphtheritic membranes, and in portions of fur taken from the tongue in fevers. Nevertheless the essential cause of thrush is a vigorous and rapid growth of this fungus, leading to inflammation of the mucous membrane and detachment of epithelium, just as the fungus of ring-worm produces a red and scaly condition of a part of the skin on which it grows. A necessary condition, however, before the oidium can germinate actively in the mouth appears to be an acid state of the buccal secretions; and according to Vogel the mouth always gives an acid reaction from the very first, before any white spots are visible. The preponderance in infants of mucus, which readily turns acid, over the alkaline salivary secretions, which are undeveloped in infancy, is perhaps the reason why thrush is so much more common in them than in older children.

In many cases thrush comes and goes with little disturbance of the health, and without any danger to life. But in others it is associated with severe and even fatal diarrhœa; and the popular notion is that the disease passes through the child and comes out at the anus. Although it is true that the œsophagus is sometimes affected in its entire length, there is no reason to believe that the *Oidium albicans* is capable of germinating on any

* *Synonyms.*—Noma—Cancrum oris.—*Germ.* Wasserkrebs—Wangenbrand.

† *Synonyms.*—Aphthæ (ἄφθαι), a good Greek word, and used by Hippocrates probably rather in the English than the German sense.—*Fr.* Muguet.—*Germ.* Soor.

surface which is not covered with squamous epithelium. Thus it never enters the nose or the larynx; but it does sometimes appear in the lowest portion of the rectum and upon the vulva, and it may also be seen where there are sore spots on the skin of the face and neck. The relation of thrush to diarrhoea in young infants probably is that they are both effects of a weakly state from bad feeding. In these cases there is usually superficial dermatitis (or "eczema") of the nates, which must be carefully distinguished from a syphilitic rash.

The treatment of aphthæ in children is to wash out the mouth at frequent intervals with a weak solution of an alkaline carbonate or of borax, or to apply the *glycerinum boracis* freely to the surface of the mucous membrane.

In adults thrush rarely occurs except in those who are reduced to a state of extreme marasmus by a chronic malady (such as consumption or cancer), or who have passed through several weeks of pyrexia from enteric or puerperal fever, or septicæmia. Thrush in adults is generally taken as of most unfavourable prognosis; but patients may, nevertheless, recover after it has appeared.

Stomatitis with salivation or ptyalism.—In patients under mercurial treatment the mouth is very apt (unless the teeth are kept scrupulously clean and the gums well washed with alum, borax, or tincture of myrrh) to present an appearance like that above described as "ulcerative stomatitis." But in addition there is also a profuse flow of saliva, the condition known as ptyalism or salivation. Salivation may occur independently of the administration of mercury, sometimes as a result of the action of other drugs (as iodide of potassium or pilocarpin), sometimes from irritation starting in distant organs (as the uterus or the stomach).

The quantity of secretion that used to be poured out by a patient undergoing mercurial salivation is astonishing. The usual daily amount was one or two quarts; but as much as five quarts is said to have been collected in twenty-four hours. The patient was incessantly spitting it out, or allowing it to dribble into a spittoon; and at night it saturated his pillow. It is more or less viscid or glairy in consistence, and is said to have sometimes a specific gravity as high as 1059; but as the case goes on its density falls till it is scarcely above that of water. It contains little sulphocyanide of potassium, and less ptyalin.

Mercurial salivation is now seldom seen. In administering mercury one watches the patient's mouth carefully, and the medicine is at once stopped when any disagreeable odour is perceptible in his breath, or when his gums become in the least degree inflamed, or his teeth tender on pressure. If one is using mercurial inunction, or calomel vapour baths, or full doses of blue pill instead of minute doses of the bichloride, the patient may often obviate salivation by frequently using the tooth-brush and taking chlorate of potass. When once salivation has developed itself the chlorate appears to have little direct influence upon it, although it more or less quickly brings the gums and the mucous membrane of the mouth into a healthy state. While salivation lasts, the patient generally becomes thin, his urine is scanty and his bowels are constipated. Relief may be given by washing out the mouth with astringent solutions, as of alum or gallic acid, to which tincture of myrrh may be added.

Xerostomia.—In the 21st volume of the 'Clinical Transactions' Mr

Hutchinson and the late Dr Hadden described independently two cases in which there was "deficiency of the salivary and buccal secretion," causing a permanently dry "mouth." Both patients were widows about sixty years of age, and a third case recorded by Dr Rowlands ('Lancet,' Jan. 14th, 1888) also occurred in a woman of sixty. Several cases have since been published but they are certainly uncommon. The condition had in two of these cases begun suddenly after nervous emotion, and it seems probable that the secretory, without the vaso-motor, nerves are primarily affected. In Hadden's case tincture of jaborandi was exhibited with relief.

AFFECTIONS OF THE FAUCES.*—Many names have been given to affections of the throat, all of which are attended with difficulty or pain in deglutition, and with more or less marked inflammation of the pharynx, palate, and tonsils. We have already spoken of the special forms of sore throat which characterise scarlatina, diphtheria, and syphilis.

Ordinary *catarrh of the fauces* is sufficiently characterised as redness and slight swelling of the soft palate and of the posterior wall of the pharynx, coming on after exposure to cold. In persons who have repeatedly had attacks of catarrh, a "relaxed sore-throat" is apt to be of frequent recurrence; the fauces feel dry and painful (especially in the morning on first waking), but the symptoms all pass off after breakfast, when nothing is to be seen beyond elongation of the uvula, slight œdema of the palate, and pallor rather than redness, with perhaps some dilated venules in the mucous membrane.

What is called *ulcerated sore throat* is apt to occur in nurses and medical students who are in close attendance on the sick, or in other persons if weakened by unhealthy surroundings. In this variety small white superficial ulcers are seen on the surface of the tonsils and other parts of the fauces, which are vaguely called follicular or aphthous, and resemble in appearance the minute yellow ulcers of the mouth and tongue described above (p. 296). There is a considerable general disturbance, the breath is foul, and the tongue furred. Wine, quinine, and guaiacum or tincture of iron are indicated, and generally bring the affection to an end in a few days. If not, a few days at the seaside is usually effectual.

It is important to distinguish from ulcers on the tonsils certain circumscribed whitish-yellow patches, which are really masses of inspissated and protruding secretion.

Follicular tonsillitis is the name given to what may perhaps be regarded as a slighter and sporadic form of the epidemic if not infective sore throat just described. It is marked by diffused redness and moderate swelling of the fauces, with numerous yellow points dotted over them. These are supposed to be the mouths of mucous crypts; but of this there is no evidence. Probably some are swollen lymph-follicles, and others inflammatory vesicles, which at first do not ulcerate. This kind of angina is always bilateral, and is not attended by the severe pyrexia of true quinsy. It is best treated by quinine, port wine, and fresh air, but astringent gargles or lozenges have their use.

Another faucial affection called *herpes of the pharynx* consists in an eruption of opaline vesicles, sometimes few in number, but sometimes so

* Sore throat.—*Fr.* Mal de gorge.—*Germ.* Halsweh. The terms *angina* and *cynanche* are often applied somewhat indiscriminately to all inflammatory affections of the fauces.

thickly crowded together that they may easily be mistaken for diphtheritic membrane. Sometimes it recurs again and again in the same patient; and sometimes it alternates with an herpetic eruption on the skin or on the genitals. There is not satisfactory evidence that these vesicles are due to retained mucus, as those of the skin called sudamina are due to retained saliva. More likely they are inflammatory like the vesicles of herpes labialis or herpes preputialis.

Quinsy.*—The most acute and severe kind of sore throat is one that mainly affects the tonsils, and is well known by the popular name of “quinsy.” Mackenzie’s analysis of 1,000 cases showed that it is far more frequent in persons between fifteen and twenty-five years old than at any other age; it is comparatively seldom seen in children, or in adults beyond the age of forty. Some persons are exceedingly liable to quinsy. It is, like most inflammations, ascribed to a chill, or to slight disorder of the stomach, or (in the case of women) to menstrual irregularity. Acute tonsillitis sometimes precedes rheumatic fever, so that in certain cases it deserves the name “rheumatic” by a better title than a doubtful dependence upon cold.

Not infrequently the pyrexia precedes the local inflammation by a day or two, so that in young adults the possibility of quinsy coming on should always be remembered when the temperature rises high without obvious cause. In many persons there is habitually a chronic enlargement of the tonsils, and upon this acute inflammation from time to time supervenes.

Quinsy appears to be rather more common during autumn than at other seasons. It is attended with great swelling of the tonsil, which forms a red, shining, globular mass, projecting into the fauces, and also distinctly to be felt in the neck at the angle of the jaw. When both tonsils are involved, they may come into contact in the middle line, being flattened and even (as Bristowe remarks) ulcerated from mutual pressure. But as a rule one side is alone affected; and sometimes, just when the inflammation is subsiding in one tonsil, the other is attacked in its turn. The uvula, the soft palate, and the pillars of the fauces all partake more or less in the swelling. The patient complains of severe pain in the throat whenever he moves his jaw, especially in attempting to swallow, and the pain may radiate to the ears. Pyrexia comes on abruptly and rises high, higher than seems proportionate to the severity of the local affection. When there is no actual rigor, the patient experiences alternate chills and flushes of heat, and complains of headache, malaise, and pains in the limbs; the pulse ranges from 100 to 120 in the minute, and is full and bounding; the temperature rises to 102°, 103°, or even 105°. The tongue is thickly furred and the breath foul.

Dr Fagge once made an autopsy in the case of a child who had died in Guy’s Hospital during the previous night of suffocation as the result of severe swelling of the tonsils from quinsy. Such an occurrence, however, is exceedingly rare. The disease almost always ends favourably, either subsiding more or less rapidly, or else advancing to the formation of an abscess in the tonsil, which breaks and discharges a thick foetid pus that is generally swallowed. This brings immediate relief to all the symptoms, the temperature falls rapidly, and the patient soon feels perfectly well. The

* *Synonyms*.—Cynanche—Acute suppurative tonsillitis or amygdalitis—Angina tonsillarum—Angina phlegmonosa. *Κυνάγχη* or *Συνάγχη* refers to the choking sensation produced by this and similar inflammations of the throat. *Angina*, from *angio*, has a similar meaning. The French *Esquinancie* and English *Quinsy* are corruptions of Cynanche.

ordinary duration of the disease is three or four days; but if the two sides of the throat are attacked in succession, it may be prolonged over a week or ten days. In some cases it is said that suppuration starting from a tonsil extends down to the neck, and burrows until it may reach the mediastinum. Cases also have been published in which tonsillar abscesses have eroded the carotid artery and given rise to fatal hæmorrhage. Mackenzie reports one case of suffocation as the result of the abscess breaking and filling the upper air-passages with pus.

The writer had a patient in 1879, a young man of twenty-four, who died from the effects of hæmorrhage due to lancing a quinsy, several hours after the bleeding had ceased. The internal carotid was not touched, and the wounded vessel was found to be the ascending pharyngeal artery.*

In the *treatment* of quinsy, sucking ice sometimes gives more relief than anything else, particularly at the onset. But when an abscess is in process of formation, the course of the disease may apparently be hastened by the use of fomentations or poultices externally and by steam inhalations. As soon as fluctuation can be felt, an incision should be made with a guarded bistoury. Störk remarks that the best way of detecting a soft spot is to push the parts inward with one forefinger placed at the angle of the jaw, while the other forefinger in the mouth is carefully passed down over the inflamed structures from point to point. In opening a tonsillar abscess the cutting edge of the knife must be directed inwards and not outwards, lest the internal carotid artery should be wounded. When the patient refuses surgical interference, an emetic has led to the immediate rupture of the abscess.†

In some cases, however, it would seem that by suitable treatment quinsy may be made to abort, and the occurrence of suppuration be prevented. Aconite has been recommended for this purpose, but guaiacum is more efficacious. Mackenzie prescribed the lozenge (three grains of the resin) every two hours, and seldom saw it fail to arrest the disease if begun at its first onset.

Chronic enlargement of the tonsils.—A not uncommon affection of the tonsils is chronic overgrowth of their substance. They may be as large as chestnuts, or even larger, and are very firm and fleshy; smooth on the surface, but sometimes with cheesy or calcareous matter projecting from their interior. There is true hypertrophy of the adenoid tissue of the tonsil, the follicles increasing both in size and number. It is therefore strictly analogous to the hypertrophy of the lymph-glands in delicate children.

The tonsil becomes scarred by the caseous follicles falling out as they soften, and thus the surface looks worm-eaten, and may remain so long after the swelling has disappeared and the tonsil has atrophied by the natural process of age.

The affection may exist from infancy, or may develop itself during childhood or at puberty. In most cases it subsides when adult life is reached, and it seldom persists after the age of thirty.

In children it is attended with many inconveniences; the mouth has to

* Some French writers, especially Maingault and Gubler, have maintained that tonsillitis is now and then followed by paralysis of the soft palate, like that which is so often seen after diphtheria. But one would rather be disposed to think that in such cases the specific disease was really present, for marked swelling and even suppuration of the tonsil is by no means rare in diphtheria.—C. H. F.

† The reader will remember the absurd story told of Abernethy's eating plum porridge against a patient with quinsy till he made him laugh and burst the abscess; and the Ingoldsby legend of how another case was cured by a less hilarious movement.

be kept open during sleep, and rhonchus accompanies the breathing. A pigeon-breast may result, and often goes with narrow and high-arched palate, with crowded teeth and a narrow passage through the nostrils. Such children snore at night and breathe through the mouth during the day, and thus constant faucial irritation is kept up. The obstruction to the posterior nares leads to breathing through the mouth, and this again aggravates the faucial swelling. There is something about the physiognomy of such patients that enables one to see at a glance what is the matter with them; Mackenzie speaks of their "open mouth, drooping eyelids, and dull expression;" and in addition to these peculiarities the voice is thick and nasal, while the act of deglutition (as in swallowing the saliva that accumulates in the mouth) is performed clumsily and with obvious effort.

Another effect of chronic enlargement of the tonsils is deafness, due to swelling of the mucous membrane obstructing the Eustachian tube; and sometimes the sense of smell (and so of what we call of taste) is impaired. The tonsils, however, may be greatly enlarged without giving rise to any obvious symptoms, and they should therefore always be inspected when a child is for the first time examined.

The *treatment* consists in the administration of cod-liver oil and iron, while the tonsils are every day brushed over with a solution of perchloride of iron, or smeared with powdered alum or tannin. But if there is much overgrowth of solid tissue, excision by means of the guillotine is necessary. If hæmorrhage follows the operation, it is generally checked by making the patient suck ice; if not, Mackenzie recommended slowly sipping half a teacupful of a solution of tannic and gallic acids, containing $\mathfrak{z}\mathfrak{v}\mathfrak{j}$ of the former and $\mathfrak{z}\mathfrak{i}\mathfrak{j}$ of the latter to the ounce of water. The wounded parts remain sore for some days; during this time the food must be soft and bland, and the use of marsh-mallow lozenges is recommended as soothing.

Granular pharyngitis.—When we examine the throat in a good light, we often notice that the pharyngeal surface is dotted over with small projections the size of millet-seeds or more. They may be either scattered, or closely packed together, or confluent in ridges. The venules of the mucous membrane are dilated and tortuous; and usually the back of the throat is dry, there being apparently a deficiency of mucus. With regard to the nature of the granulations, it is generally supposed that they consist of enlarged muciparous glands, while, according to Störk, the mucous membrane generally is thinner than natural. Microscopically the granulations show an absence of the superficial stratum of epithelium, and large round swollen cells lie uncovered.

Mackenzie describes an "exudative form" of the affection in which viscid mucus is seen adhering in patches to the follicles, or in which their orifices are filled with a white caseous material. This is identical with "pharyngeal herpes," and closely related to the subacute follicular sore throat described above (p. 300).

Dr Horace Green, of New York, who gave a graphic account of granular pharyngitis, called it "follicular disease" and "follicular inflammation" of the throat and air-passages. It is by no means limited to the regions directly visible at the back of the mouth; but often spreads upwards towards the vault of the pharynx, or downwards towards the larynx. Michel insisted on the importance of thoroughly exploring with the laryngeal mirror every part of the fauces; for example, when the rest of the surface is quite

healthy, there may be a small patch of disease just behind one or both of the posterior arches of the palate; this is easily overlooked, and yet it may be the cause of great suffering to the patient, as is shown by touching it with a probe.

The subjective symptoms of granular pharyngitis vary widely in different cases. Often it causes no discomfort at all. Sometimes it gives rise to a troublesome feeling of stiffness or dryness in the throat, with a constant need for swallowing, to a pricking pain during deglutition (especially deglutition of the fluid secretions of the mouth), to a tickling sensation compelling a frequent and often painful cough, or to incessant hawking, in the hope of getting rid of mucus, until retching or actual sickness may occur.

In other cases speaking or singing may be attended with a sense of painful effort; the patient may be obliged to stop from time to time, in the middle of a sentence, to swallow or to clear his throat. Such cases are very common in preachers and public singers, as well as in street hawkers and costermongers (cf. p. 122).

Talking in the open air plays the principal part in causing or in aggravating the symptoms of granular pharyngitis; but irritation of the fauces by tobacco smoking is another exciting cause.

Granular pharyngitis appears to be sometimes due in part to an inherited predisposition, a vulnerability of the fauces and pharynx. Dr Green speaks of three brothers, all clergymen, whom it compelled to give up their official duties; and he refers to many cases of two or three members of a single family having been treated for it.

Some patients seem unable to forget, even for an instant, the morbid feelings in the back of the throat; they are always running from one doctor to another, and, in fact, become hypochondriacal.

Granular pharyngitis is most apt to occur from twenty-five to thirty-five, and in men more often than in women. But Mackenzie observed it in children who were eight, six, or only three years old.

In its *treatment* the first object is to destroy the granulations. Sprays, gargles, and inhalations are not sufficient, and so treated, granular pharyngitis is often very intractable, and runs on for years. In fact, as gargling with fluids is commonly practised, it seldom brings them into contact with any part of the fauces behind the anterior pillars.

Guinier, of Caunterets, showed in his 'Etude sur le Gargarisme laryngien,' that when a person with his head thrown backwards makes fluid bubble about in the back of the mouth, while he goes on inspiring at regular intervals through the nose, the fluid is supported by the base of the tongue, the uvula, and the anterior pillars of the palate; any portion of it which passes further backwards is instantly swallowed. He has, however, shown that it is possible to teach patients to gargle in quite a different manner, and that the fluid then passes not only into the pharynx, but even into the larynx itself, resting directly upon the upper surface of the vocal cords. The directions are that the head should be slightly raised, the mouth but little opened, the lower jaw thrown forwards, so as to lift the chin. Having taken a small quantity of fluid into his mouth, the patient is to draw a deep breath through the nose, and then to allow the fluid to fall back into the fauces while he endeavours to emit the sound of the vowel *é* (or the English *a* long). This means that the cords are brought together, and that the act of expiration is begun; while at the same time the epiglottis is raised so as to throw widely open the upper part of the laryngeal cavity. A bubbling sound is produced by the thin stream of air which passes outwards between the cords; this sound, as Guinier says, is quite unlike that of ordinary gargling; it resembles rather the rattle in the throat which precedes death. The act of gargling within the larynx can be continued only so long as a slow expiration is being maintained. Before a fresh breath can be taken the fluid must be thrown up into the pharynx, whence it often passes out through the nostrils. I have myself seen Dr Guinier demonstrate with the laryngoscope the presence of a layer of fluid resting upon his own vocal cords.—C. H. F.

Mackenzie used to apply a caustic paste to each granulation separately, touching on the same day only two or three, and sometimes only one of them; and it is easy to imagine how tedious this practice must have been to the patient. But in 1873 Michel, of Cologne, drew attention in the 'Deutsche Zeitschrift f. Chirurgie' to the success attained in about seventy cases by the use of the galvanic cautery. The method consists in applying a heated platinum loop to the granulations, so as just to destroy their surface; and, as a rule, the operation has to be repeated only three or four times, inasmuch as the effect is not limited to the part immediately cauterised, but extends to some distance around. There is little or no pain at the time, except when the pillars of the fauces are the parts touched by the instrument; the inflammation which follows can be kept within bounds by making the patient during the first few hours suck ice at intervals; and there is nothing to prevent his continuing his daily business. Dr Foulis, of Glasgow, employed a small gas cautery for the same purpose; but this must be heated before being passed into the mouth, whereas the platinum loop of the galvanic cautery is cold at the times of its introduction and of its withdrawal.

Adenoid vegetations in the vault of the pharynx.—In 1869 Dr Wm. Meyer, of Copenhagen, drew the attention of the Royal Medical and Chirurgical Society (vol. liii) to this affection, which he was almost the first to recognise. It is exceedingly common in children, and consists in vascular lymphomata of various shapes and sizes, occupying the posterior wall or the roof of the pharynx, or sometimes its sides, or the upper surface of the soft palate, but never the posterior nares. They are sometimes soft and sometimes firm. They consist chiefly of veins and lymphatic tissue—a scanty areolar network, its meshes filled with lymph-cells. The epithelium which covers their surface is either ciliated or of pavement form, according to their exact seat. Their colour is generally the same as that of the more or less congested mucous membrane, or they may have a yellowish hue. In most cases the disease begins as a hypertrophy of the third or pharyngeal tonsil. Pathologically it is allied to enlargement of the tonsils and lymph-glands, and to lymphadenoma.

One can often tell by the way a child speaks, and by the expression of his face, that he has adenoid growths in his pharynx. The peculiarity of the speech is an inability to utter the nasal sounds *m*, *n*, *ng*—so that instead of "common" the patient says "*cobbod*;" instead of "nose," "*doze*;" instead of "song," "*sogg*." This depends upon the occlusion of the posterior nares preventing the nasal cavity from acting as a resonator, so that the explosive vocal sounds take the place of the continuous vocal or "nasal" sounds. The same effect is produced by closing the anterior nares, or by occlusion of the passage from the swelling produced by a cold in the head.

The peculiar facial expression depends also upon the obstruction to the passage of air through the nostrils in breathing. This causes them to appear narrow and collapsed, and the nose itself looks thinned and flattened from side to side. It also compels the patient to keep the mouth more or less constantly open; and since the orbicularis oris no longer gives support to the other muscles of the face, the countenance acquires a dull and vacant aspect, often increased by an odd trick of twisting and pouting the lips.

Other symptoms are a feeling of fulness in the upper part of the fauces, as though there were a foreign body there, a secretion of thick mucus which

glides down the pharynx and compels the patient to be constantly clearing the throat, the presence of blood in the mouth, especially on first waking in the morning, and frequent headache. Children with this disorder not only habitually breathe through the open mouth, but when asleep they snore, a symptom that should always lead to suspicion of these adenoid growths being present.

They often suffer from an impaired state of hearing, owing to occlusion of the Eustachian tubes by the adenoid growths. In certain cases there is inflammation of the tympanic cavity, and the membrane may be perforated, and give exit to a purulent discharge. Moreover, various affections of the fauces are apt to be associated with the presence of pharyngeal adenoid growths. The tonsils may be enlarged, there may be granular pharyngitis, the uvula and the soft palate may be thickened. There may also be catarrh of the anterior parts of the nasal cavities, though in the majority of cases the secretion of the Schneiderian membrane is rather deficient than excessive. The lips may be excoriated by the discharge, and epistaxis or ozæna may be present.

To explore the upper part of the pharynx, the forefinger should be passed between the tongue and the roof of the mouth, and insinuated by the side of the uvula until it glides upwards behind the velum. It is then carried along the posterior edge of the septum of the nose, and turned in various directions until every part of the space has been thoroughly examined. The examination sometimes causes nausea, or pain in the back of the head, and there is often copious bleeding from the growths when they are touched. In the case of adults and of children above nine or ten years old, rhinoscopy is of much assistance in diagnosis. The vegetations can be readily seen in cases of cleft palate, a deformity that appears to be not infrequently associated with their presence.

The systematic examination of school children in Denmark, and also in England, has shown that from 1 to 5 per cent. of them are affected with pharyngeal adenoid growths. The disease is said to be more frequent when the climate is cool and damp. It is seldom seen in persons above eighteen or twenty, and it appears often to be congenital or to date from very early childhood. Out of 107 cases Mr Symonds noted that 90 were under fifteen years of age. It frequently affects several members of the same family, and is sometimes hereditary. Meyer thought that it is more common in boys than in girls, but in Mr Symonds' cases there were 65 female to 58 male patients.* It is sometimes a sequel of measles. As remarked by Wiesener, of Bergen, it often leads to a scrofulous (*i. e.* tubercular) infiltration of the cervical glands.

As a rule, when adult life is reached, adenoid vegetations in the nasopharynx shrink and disappear spontaneously, after having done serious, and sometimes irreparable, damage to the child's development and comfort, both bodily and mental.

Sometimes cauterisation with solid nitrate of silver suffices to destroy them, but they generally have to be scraped off by suitable instruments, of which descriptions may be found in Meyer's several papers, and in the 'Transactions of the International Congress of 1881.' He employed a small oval ring with a sharp though not cutting edge, mounted on a slender stem. The curette designed by Goldstein is now generally preferred, alone or after using Löwenberg's forceps. The operation causes little pain but

* See a lecture reported in the 'Guy's Hospital Gazette,' October 11th and 25th, 1890.

some hæmorrhage, which is easily checked. It has to be repeated only when the vegetations have not been completely got rid of on the first occasion; for if any remains of them are left they are sure to grow again.

Adenoid pharyngeal growths are frequently removed in the out-patient room, without danger and with excellent results. It is necessary in the case of children who have never used the nose for effectual breathing, that they should be taught by definite exercise how to breathe with the mouth shut. The effect of the complete removal of pharyngeal adenoid growths is quickly to restore the natural speech, to change the whole expression of the patient's face, and in many cases to bring back the sense of hearing.

Malignant disease of the tonsils.—Lymphadenoma of the tonsils is sometimes difficult to distinguish from hypertrophy; but occasionally it shows the most conclusive evidence of an infective new growth.

A man, aged forty-eight, under the writer's care in 1888, had enlargement of both tonsils, and of the closed follicles on the back of the tongue. There were also signs of pressure on the left bronchus and of effusion into the left pleura. Blood-stained serum was drawn off, and afterwards three ounces of pus. The temperature was raised while in hospital, but never rose above 102·6°. The diagnosis was of intra-thoracic growth pressing on the left bronchus and causing pleuritic effusion. There was much mental excitement and delirium, and he became excessively anæmic and very emaciated before death. After twelve months' illness he died, and lymphatic tumours were found *post-mortem* in the stomach and intestines, as well as in the thorax. The appearances after death were described and figured by Dr Pitt in the 'Path. Trans.,' xl, p. 80. The tonsil and tongue are preserved in the Guy's Hosp. Museum (No. 479).

Epithelioma (corneous cancer) of the tonsils is occasionally met with (Guy's Museum, Nos. 477, 478). It sometimes destroys life by sudden hæmorrhage, sometimes by suppuration and pyæmia, and sometimes by secondary growths in the skull or in the lungs. Epithelial cancer of the pharynx is less rare than in the fauces, and may lead to constriction like that of the œsophagus. Mr Davies-Colley opened the gullet to relieve a case of this kind in a woman sixty years of age in 1891.

Specimens of sarcoma of the pharynx, and of tuberculous ulceration of the tongue, fauces, and pharynx, are preserved in the Guy's Hospital Museum (Nos. 495-6, 488, and 497-8).

DISEASES OF THE ŒSOPHAGUS.—This part of the alimentary canal is remarkably free from the slighter inflammatory affections which are so common both above and below it. Its thick layer of squamous epithelium seems to protect it from ordinary irritation; and it has neither the rich blood-supply, nor the active secreting functions, nor the abundant lymphatic tissue, which elsewhere in the alimentary tract become occasions of disease.

Chronic *inflammation* of the gullet with thickening of the mucous membrane is sometimes seen either as the result of external pressure, *e. g.* from a thoracic tumour or an aneurysm, or in cases of habitual congestion from chronic valvular disease of the heart. In the former case the thickened and opaque mucous membrane is in external appearance, as in pathology, like the "corns" produced by friction on the pericardium, or the white patches of the tongue called *leucoplakia*. In the latter case extreme venous congestion may be discovered after death, with desquamation of the upper layers of epithelium, a condition comparable to the congestive catarrh of the stomach from disease of the heart.

Occasionally when no source of pressure or irritation can be ascertained, the mucous membrane is found (at times over a considerable space) to be

covered with minute papillæ, which may be large and circumscribed enough to deserve the name of a *papilloma*.

Beside warty growths, traumatic and syphilitic ulcers, and malignant disease, all of which begin in the mucous membrane, we occasionally meet with small, oval, white, firm tumours of the muscular walls (Guy's Hospital Museum, Nos. 556-7). They are quite innocent, and are found to consist of unstriated muscular fibres (*leiomyoma*). Polypi also have occasionally been observed in the œsophagus.

Functional stricture.—The above pathological conditions are without clinical significance; there are also functional affections of the gullet without an anatomical explanation. Of these the most important is what has been called "spasmodic stricture." The patient is usually young, most often a girl at the age between puberty and childbearing, when functional neuroses are most common. It also, however, occurs in male subjects, and one of the most marked and obstinate cases the writer has seen was in a boy of fourteen. In most cases the neurotic or "hysterical" character of the affection is sufficiently evident, and the easy passage of an œsophageal sound, which completes the diagnosis, often effects the cure as well.

Regurgitation.—Another condition which is, at least in its origin, functional, is rejection of food after it has passed the constrictors of the pharynx. This differs from the gastric regurgitation, or, as it may be termed, "rumination," which consists in return of the food from the stomach; for here the food never reaches the gastric cavity, but is detained in the gullet. Œsophageal regurgitation appears to begin rather as a bad habit than a disease; but whether or not there is any structural lesion as its original cause, there is frequently, or perhaps always in confirmed cases, a pouch formed in the lower part of the gullet in which food collects before its regurgitation.

Dilatation of the œsophagus is said to be more frequent in men than in women. It usually involves the whole thickness of the tube; but cases have been described in which the mucous coat alone has protruded between the muscular fibres, so as to form a hernial pouch. A classical case of this curious condition was published in the thirteenth volume of the 'Medico-Chirurgical Transactions' (1849) by Mr Worthington, of Lowestoft. (See also specimens 527, 528, and 529 in the Guy's Museum.)

An œsophageal pouch is most frequently met with, not as a primary lesion, but as the result of a stricture immediately below it. It has occasionally proved to be the result of a mere narrowing of the gullet at its cardiac end. Such a case was described and figured by Dr Fagge in the 'Guy's Hospital Reports' (third series, vol. xvii, p. 414). At last a cancerous growth developed and ended in the patient's death at eighty-four, forty years after the appearance of dysphagia. In the same paper is figured a dilated œsophagus resulting from a simple, non-traumatic stricture of the cardia, which was described by Wilks in the seventeenth volume of the 'Pathological Transactions' (p. 138). Regurgitation and dysphagia had existed all the life of the patient, a healthy farmer, who had once consulted Sir Astley Cooper for œsophageal stricture, and who died at seventy-four of acute pneumonia. Durham discovered two cases of dysphagia and simple stricture, recorded by Sir Everard Home ('Practical Observations on the Treatment of Stricture in the Urethra and in the Œsophagus,' 1821, vol. ii, p. 398). One of these showed, *post mortem*, a fold

of mucous membrane, which narrowed the gullet just opposite the cricoid cartilage.

Dysphagia lusoria, as it has been called, demands a word of notice. The term was first applied by Dr Bayford, of Lewes, to a case (perhaps of spasmodic stricture of the gullet) in which the right subclavian artery arose from the third part of the aorta, and passed to its distribution between the œsophagus and trachea. The existence of this abnormality was probably a mere coincidence; for the much more frequent irregular arrangement of the right subclavian arising from the third part of the arch, and passing *behind* the trachea and œsophagus, between the latter and the vertebræ, is found, *post mortem*, in persons who have never experienced difficulty in swallowing.

Simple, i. e. non-malignant *stricture* of the œsophagus, can in most cases be traced to a *traumatic* origin, most frequently to irritant poisons, such as sulphuric and oxalic acid, phenol, and zinc chloride. Constriction of the gullet by external cicatrices or pressure of diseased vertebræ, abscesses, aneurysms, cancerous mediastinal glands, or possibly a distended pericardium, will of course have the same results as true stricture or contraction of its walls, and will only be distinguishable during life by evidence of the presence of the external compressing cause. In one case, in which Mr Pye Smith, of Sheffield, performed gastrostomy, the patient, an old woman, confessed years after the operation that she had once taken sulphuric acid with suicidal intent.

Stricture is occasionally due to contraction of a *simple ulcer* of the œsophagus, which is, however, a very rare affection compared with the corresponding lesion in the stomach, or even the duodenum. There is no doubt that *syphilitic* ulcers, probably of a tertiary gummatous nature, may give rise to contraction of the œsophagus ('Guy's Hospital Reports,' vol. xvii, ser. 3, 1872, p. 413). Whatever the nature of a stricture, hypertrophy, with dilatation of the muscular walls, may frequently be seen above it.

Malignant stricture of the œsophagus is always primary, and is usually epitheliomatous (the keratoid, or flat-celled kind). Adenoma or glandular cancer has occasionally been observed, but true examples of its encephaloid or scirrhus variety are both extremely rare.

The most frequent position is in the middle of the tube, opposite the bifurcation of the trachea. A less common seat is the upper portion, where cancer spreads so as to be described indifferently as pharyngeal or œsophageal. Lastly, a malignant stricture is occasionally met with at the entrance into the cardiac orifice of the stomach. It is here that its presence is most apt to be overlooked, from the diseased portion being left in the diaphragm when the stomach and œsophagus have both been removed, as Virchow long ago observed. The foregoing statement as to the most frequent seat of cancer is that of Wilks, Rindfleisch, and Klebs. Some text-books, however, follow Rokitansky's original assertion, that the upper part is the most frequent seat and the lowest the rarest. Förster, Moxon, Payne, and Coats say that the commonest seat of cancer of the œsophagus is its lowest third; and this statement is confirmed by the careful analysis of Petri and Zenker. In 58 cases collected by these two writers 4 were in the upper, 14 in the middle, and 24 in the lower third. The remaining 16 cases spread over the middle and adjacent parts also. The last group of cases is the disturb-

ing element which has probably affected the classification of statistics. Of 13 cases collected by Dr Norman Moore, of St Bartholomew's Hospital, the lower third was affected in 7, the middle third in 5, and the upper in 1. It still, however, remains true that the part which corresponds to the bifurcation of the trachea is a frequent seat of œsophageal cancer, and that cancer of the extreme cardiac end is rare.

The malignant growth is sometimes a mere ring like an annular stricture of the colon, but more frequently it forms an ulcerated flat tumour which only partially encircles the tube, and infiltrates and spreads up and down for an inch or more. Hughes Bennett recorded a very rare case of double cancerous stricture ('Princ. and Pract. of Medicine,' p. 453).

The growth of epithelioma of the gullet is slow, and rarely affects more than the neighbouring lymph-glands of the mediastinum; but occasionally secondary nodules are found in the viscera,—least rarely, perhaps, in the liver or the lung. It may produce fatal hæmorrhage, or may perforate the œsophagus by sloughing, involve the vagus nerves, or open anteriorly into the trachea or lung.

Malignant stricture is much more common in men than in women, and is rare before middle life. All the patients under the writer's care have been between fifty and seventy years of age and all but two have been men.

Its early *symptoms* are slight and its progress insidious. Difficulty in swallowing solid food is commonly the first complaint. Pain, though occasionally severe, is often long before it appears, and is sometimes almost absent. Gradually the patient finds it more and more difficult to swallow soft food, and at last even liquids, and increasing emaciation is the result. So latent may the symptoms be that the disease has sometimes been only discovered after death, though the tradition of a diagnosis once made by Sir Astley Cooper shows how the aspect and age of a person suffering from this disease may lead to its recognition by experienced observation, or by a shrewd guess.

In most cases, when a man of middle age, or older, comes complaining of inability to swallow food, he can indicate the exact spot where he feels it stop; and the negative result of examination of the other organs, with his progressive loss of flesh, makes the diagnosis easy. The way in which he eats, taking minute pieces of bread at a time, and fumbling with them before he makes up his mind to try to swallow them, is very suggestive. Soft food, like bread and milk, can be eaten when ordinary diet is rejected, and it is often weeks or months later before difficulty is experienced with liquids. The regurgitation of a little blood with the food is a frequent symptom.

The only possible diagnosis between simple and malignant stricture depends upon the age of the patient and the history of the case.

The aspect of a patient in the advanced stage of the disease is characteristic—the extreme marasmus, without jaundice or cyanosis or dyspnoea, the look of starvation and of long-endured suffering from thirst, the hollow eyes, dilated pupils, sunken cheeks and temples, and, above all, the deeply concave abdomen, feeling empty when examined, and allowing the aorta, the vertebræ, and the kidneys to be plainly discerned by the fingers. The skin is dry and rough, the condition known as *pityriasis tabescentium* . The bowels are confined for days or weeks. The urine is scanty, high-coloured, and often offensive.

The cautious passage of a bougie is the only decisive proof of the nature

or the disease, and also gives a criterion of its position and of the calibre of the tube. Hamburger's methods of diagnosis by auscultation deserve mention (Erlangen, 1871. See also Dr Allbutt's paper, 'Brit. Med. Journ.,' ii, 1875). Of less practical importance is the ingenious attempt to obtain a view of the gullet by Waldenburg; his instrument is figured in the 'Berlin klin. Wochenschrift,' 1870, p. 580.

Event.—At length, "after enduring the tortures of Tantalus, the patient slowly wastes away," as Boerhaave remarked. Death, however, often occurs from intercurrent pneumonia or pleurisy, and sometimes from gangrene of the lung or from hæmorrhage. In one case under the writer's care the primary stricture was latent, and the first symptoms were pain, and afterwards paraplegia, caused by secondary cancer of the vertebræ.

The *duration* of these cases is very variable; it is to be counted by months, and some patients have lived on for two years, or perhaps for longer. Occasionally cases may prove fatal by perforation within a few weeks of the first appearance of dysphagia.

The *treatment* of stricture of the œsophagus is purely mechanical. When free from ulceration the stricture will usually be benefited by the frequent passage of a bougie; in fact, by the same method of dilatation as is adopted for stricture of the urethra. In cases of simple traumatic constriction this is sometimes an effectual cure; but even in cancerous strictures, so long as there is no ulceration, the occasional passage of an olive-shaped bougie frequently affords great relief. The utmost care and gentleness is essential, or fatal perforation may ensue. As Mr Bryant well puts it, when a patient complains of difficulty in passing food onwards down the gullet after the act of swallowing, and of its return into the mouth, the practitioner should first think of thoracic aneurysm, then of cancer, and then of some other kind of ulceration. The dread of such a catastrophe as perforating an aneurysmal sac, or thrusting a bougie into the pleural cavity, will, however, be the best safeguard against its occurrence. No force which an intelligent hand could use will perforate an intact mucous membrane. When ulceration has already taken place, no one would willingly risk the passage of a tube; but when there is no evidence of ulceration the practice is defensible and beneficial.

The *sonde œsophagienne à demeure* is an instrument which is passed through a stricture, and then left in it as a catheter is left in a stricture of the urethra. It was advocated by Mons. Krishaber at the International Medical Congress of 1881 ('Transactions,' vol. ii, p. 392). He advises its passage through one of the nostrils rather than the mouth, and in proof of the tolerance of the instrument relates four cases in which the tube remained continuously *in situ* for 46, 126, 167, and 305 days respectively. Feeding, of course, takes place entirely through the hollow œsophageal sound. A similar plan has been carried out by Mr Symonds at Guy's Hospital with good success.

When obstruction has become complete, life may be preserved for a time by nutrient enemata. For this purpose small quantities of peptonised food without salt or alcohol are best employed; and in many cases raw eggs, beef-tea, and pancreatised milk are well retained and absorbed. But often the rectum rejects the nutriment; after a time this result is almost sure to occur, and even in the most favourable cases the patient is insufficiently nourished. In acute cases of disease, or of injury or operation about the mouth and throat, or even while a gastric ulcer is given time and rest to

heal, the treatment by rectal alimentation is most valuable. But when, as in stricture of the œsophagus, the disease is progressive, it is far better, so soon as occlusion occurs, for the operation of opening the stomach to be performed before the patient's strength and endurance have been exhausted.

This operation of gastrotomy, or *gastrostomy*,* was first performed by Sédillot in 1849, and was introduced into England by Mr Cooper Forster. It has since been amply justified by the long periods of life and comfort which it has afforded to patients who would otherwise have died by one of the most painful deaths—that from thirst. In a case brought before the International Congress in 1881 ('Trans.,' p. 456) the patient survived a year and a half; and in a second case (for malignant stricture), by the same surgeon, life was prolonged for 128 days. For similar cases see a paper by Dr Gross, jun., in the 'American Journal of Medical Science,' for 1884.

The safety of the operation has been increased by the plan introduced by Mr Howse, of first cutting down to the stomach and fastening it by sutures to the abdominal walls, and a day or two afterwards, when adhesions have formed, opening it and introducing the cannula.

* *Gastro-stomie* (*i.e.* making a mouth in the stomach) was the French surgeon's original term. Mr Bryant follows Dr Pooley, of New York, in defining "gastrotomy" as opening the stomach for removal of a foreign body.

DISEASES OF THE STOMACH

“Ex magna cœna
Stomacho fit maxima pœna.”

Regimen Sanitatis Salernitatum, c. 1075.

“Qui Stomachum Regem totius corporis esse
Contendunt niti verâ ratione videntur;
Hujus enim validus firmat tenor omnia membra,
Et contra ejusdem firmanantur cuncta vigore.”

‘*Ventriculi Querelæ et Opprobria*’ of *Bernhard Swalve*, 1664.

Acute catarrhal gastritis—its symptoms, causes, histology, and treatment—Acute paralytic distension—Acute suppurative gastritis.

Atonic dyspepsia and chronic catarrhal gastritis—Symptoms—Anatomy—Causes—Diagnosis and prognosis—Treatment—clinical varieties—drugs and diet—Chronic gastric paresis with dilatation.

Gastralgia—Anorexia—Bulimia—Pica—Flatulence—Eructation and regurgitation—Pyrosis—Vomiting—Hiccough—Hæmatemesis.

GASTRIC ULCER—Anatomy—Pathology—Ætiology—Symptoms and diagnosis—Latency—Event and duration—Treatment—Duodenal ulcer.

CANCER OF THE STOMACH—Carcinoma of the pylorus and of the body of the stomach—Sarcoma—Colloid cancer—Other occasional tumours—Symptoms—the tumour—consequent dilatation—Gastro-colic fistula—Diagnosis—Duration—Treatment, palliative and by operation.

Fibrous induration of the stomach—Gastric concretions.

Two diseases of the stomach are at once common, clinically important, and dependent on a definite anatomical condition—ulceration and cancer.

The few remaining structural lesions are clinically unimportant, while by far the most frequent morbid condition, indigestion, is of uncertain pathology and probably often without morbid anatomy.

Some acute and severe forms of dyspepsia, however, particularly those produced by obvious and direct irritation, are certainly dependent on acute inflammation of the gastric mucous membrane. We will therefore deal with this condition first, and afterwards with by far the most frequent gastric disorder of all, chronic dyspepsia. Under this head we will include not only the cases in which chronic gastric catarrh can be recognised, but others which may depend on deficiency of muscular movements (gastric paresis and dilatation), or deficiency of gastric secretion (apepsia), both of them sometimes demonstrably of nervous origin, sometimes the result of a pyrexial state. Moreover we shall include under the same general clinical account

cases which are perhaps duodenal or hepatic, or possibly pancreatic in pathology rather than strictly gastric.

In health, as everyone knows (or has known) by experience, the process of digestion is unconscious; we ought not to know that we have stomachs. But occasionally, even in healthy persons, digestion is accompanied with unpleasant feelings, varying in degree from a slight sense of weight or discomfort up to the most acute pain.

Beside pain or discomfort mostly referred to the epigastrium, indigestion is frequently accompanied by flatulent distension, and very often by loss of appetite, nausea, or actual vomiting, by constipation, depression of spirits, somnolence, and a sallow, unhealthy pallor of the face.

1. ACUTE DYSPEPSIA.—In healthy persons the stomach sometimes resents a particular meal. Of such cases the following may be taken as examples:—A schoolboy hastily swallows raw and half-ripe apples; within an hour he feels ill, and presently rejects the contents of his stomach; but next morning he is well again. A man eats much more than he requires, or takes more wine than is good for him; he goes to bed with an uneasy feeling at the epigastrium, and in the night vomits the food that he had swallowed, almost unchanged; his discomfort is relieved, and he falls asleep.

A little bile constantly appears in the vomit after the stomach has emptied itself of its accumulated contents; and formerly this fact was regarded as a sufficient ground for calling the attack “bilious.” But we now know that, in consequence of the antiperistaltic movements induced in the duodenum, bile enters the stomach whenever there is much vomiting, and is rejected in its turn.

To attacks like these the name of acute indigestion is applicable; though it is often given to a somewhat different class of cases, which are of longer duration, but are believed to depend upon a catarrhal inflammation of the gastric mucous membrane, so that the terms “acute indigestion” and “acute catarrh of the stomach” are used as almost synonymous.

Symptoms.—In addition to a sense of weight and oppression at the epigastrium, which is common to all forms of dyspepsia, there is local pain; usually a dull aching, but sometimes of a burning, stabbing, or griping character. One patient complains of soreness at the left of the sternum and about the left scapula; another feels more pain in the back than in the abdomen. In some cases the epigastrium is tender, and it often feels tense and full. The patient is thirsty and eager for cold and effervescing drinks; but he has no appetite, and often feels a loathing for all kinds of food. The breath is offensive, the bowels confined, the tongue foul and coated with thick yellowish fur. Nausea and retching are generally present, or eructation of sour gas. Partially digested food, with a very acid taste, is vomited; or a quantity of whitish mucus. Aching in the back and limbs, malaise, and depression of spirits are more or less marked. The skin is generally moist, but in severe cases there may be slight fever, and still more rarely an herpetic eruption on the lips and chin.

An attack of this kind may last from a day or two to a week, perhaps longer; and if injudiciously treated, it may pass into chronic gastritis.

The most obvious *causes* of acute gastric catarrh are indiscretions in diet, either in quantity, quality, or both. Decomposing meat or vegetables, and shell-fish under certain conditions, are apt to give rise to acute catarrh of the stomach. The direct ingestion of irritants like arsenic and antimony,

causes violent inflammation of the stomach, but the action is not only local ; for the same effect is produced by the inhalation of arsenical dust or vapour given off from green wall-papers. Wilson Fox mentions the case of a healthy child who, after sleeping in a room lined with such a paper, was seized with severe vomiting, and brought up blood. In infants improper food frequently causes acute gastric attacks, commonly, but not always, complicated with diarrhœa. In these cases prostration is a marked feature, and death sometimes results.

Exposure to changes of temperature is said to be another cause of gastritis. In England summer heat is generally associated with diarrhœa, while dyspepsia and "a sluggish liver" is apt to follow the east winds of spring.

Epidemic influences are also supposed to act. Some of these cases are due to the influenzal bacteria. Others no doubt depend on common climatic conditions.

Acute catarrh of the stomach accompanies scarlatina, erysipelas, measles, diphtheria, smallpox, puerperal and other fevers. It is almost as constant a complication of chronic gastric congestion from portal obstruction, from mitral disease, or from any cause of dilatation of the right side of the heart.

Anatomy.—Since acute gastric catarrh is very seldom fatal except in infants, one might expect that its morbid anatomy would be unknown. But, half a century ago, the remarkable case of Alexis St Martin occurred. This young Canadian had a fistulous opening into his stomach, as the result of a terrible gunshot wound, so that part of the mucous membrane was permanently exposed to view. The case was carefully investigated by Dr Beaumont, of Pittsburg, in Pennsylvania, and he relates that deep red pimples sometimes appeared, which afterwards became filled with purulent matter ; and at other times irregular circumscribed red patches, small aphthous crusts, and abrasions of the lining membrane, leaving the papillæ bare. These diseased appearances, when considerable, were attended with dryness of the mouth, furring of the tongue, thirst, and acceleration of the pulse, and the secretion of gastric juice was suspended, so that food remained undigested for twenty-four or forty-eight hours or more, although liquids were absorbed as soon as they were swallowed. Mucus was also poured out by the surface of the stomach, and slight hæmorrhages sometimes occurred.

It is true that symptoms were by no means constantly present when the mucous membrane was inflamed ; but the case is of value as showing that the stomach is susceptible of morbid changes which, if they could generally be seen, would arrest attention ; for in the deadhouse such changes can seldom be observed.

After death the lining membrane of the stomach is often acted upon by the gastric juice, so that it becomes softened and pulpy, or may be entirely dissolved over a more or less extensive area, and all the coats of the organ may in this way be perforated, so as to leave a large ragged aperture. As might be expected, this is generally at the back of the stomach, on which the contents rest while the body lies with its face upwards.

In the stomach, as in all other parts, congestion is apt to subside after death. It is true that in chronic disease of the heart the gastric mucous membrane is found intensely reddened, ecchymosed, and lined with tenacious mucus ; moreover great reddening is often seen in the bodies of

those accustomed to drink spirits to excess; but the changes which Beaumont described would no longer be visible at an autopsy.

Other changes, however, are described as characteristic of acute gastric catarrh—a milky opacity of the mucous membrane, which is soft, thick, and lacerable. Under the microscope the secreting cells distend the gastric tubules. Dr Cayley and Dr Fenwick have found in fluids from the stomach tube-casts comparable with those which occur in the urine of Bright's disease. They are figured in the 47th volume of the 'Med.-Chir. Transactions.' Infiltration of leucocytes between the tubules has been also observed, and proves the existence of inflammation of the mucosa.

In addition to these changes, Wilson Fox described an increase in size of the solitary lymph-follicles of the stomach, which appear as small white specks, scattered over its surface, and ulcerated to form little cup-shaped depressions.*

Clinical varieties.—We may in practice recognise acute catarrhal gastritis under the following conditions. First the cases due to irritant *poisoning*, as by arsenic. The possibility of this as a cause must never be forgotten; it may be accidental, suicidal, or homicidal in origin, and it may be the result of a single dose or of gradual absorption suddenly producing its accumulated effect. Itching and redness of the eyes, a silvery tongue, diarrhoea, and pain in addition to the vomiting are the most important symptoms. If no arsenic can be discovered in the vomited matters or fæces, Marsh's or Reinsch's process of identification will probably have already pointed to antimony, should that metal (as in the well-known Balham case) have been the cause of gastritis. Mercury would be almost certain to produce salivation at the same time. Of vegetable irritants, poisonous fungi are the most frequent causes of acute gastritis in adults, and various berries in children. The poisonous symptoms from eating high game, "tinned lobster," or mussels, merge into those of ordinary acute dyspepsia.

Gastritis ab ingestis comes next—dyspepsia from intemperance in eating and in drink, and that of infants fed on milk which does not agree with them.

Then follows the gastric catarrh which accompanies all *febrile* states, but is strictly symptomatic and secondary. Variola is perhaps, of all fevers, that in which the gastric symptoms are most constant and severe, and close to it come scarlatina, pneumonia, and acute gout.

Next come the cases of acute and subacute congestive gastritis which constitute so marked a feature of the morbid anatomy of *cardiac* disease.

Lastly, there is a clinical group of cases in which somewhat severe epigastric pain is accompanied by vomiting, first of the contents of the stomach, then of a quantity of mucus, and lastly, of bile. There is a thickly coated tongue, complete anorexia, considerable thirst, and, as a rule, constipation.

These symptoms occur in elderly rather than in young patients. They

* It is important to note that the cases in which these appearances have been found have not been simply cases of gastric catarrh, for these do not terminate fatally. The observations in question were made in the bodies of those who had died of scarlatina, diphtheria, pneumonia, or some other fever. They undoubtedly prove that these diseases are attended with morbid changes in the stomach, but not, it would seem, that they are to be regarded as so many causes of acute gastric catarrh, in the clinical sense of that term. For, although in the instances in which the morbid changes were found after death, the patient may have been more or less sick and have had a furred tongue, yet it is probable that the catarrh of the stomach was only secondary and did not in any way modify the course of the disease.—C. H. F.

may come in the course of chronic Bright's disease (apart from uræmic vomiting) or other lingering malady; but they are most often seen in combination with bronchitis and myalgia. In such cases we find the heart and the kidneys unaffected and no evidence of more than moderate catarrh of the large bronchial tubes. The temperature is not raised, but the pulse is quick and irritable, and the "muscular rheumatism" severe, particularly in one or other shoulder and in the back and loins; or there is decided pleurodynia without a pleuritic rub or effusion. The patients appear to be more ill than any physical signs discovered account for; and one is anxious lest some primary lesion has been overlooked. Usually, however, with careful nursing, warmth, strict diet, and a dose of calomel, these cases do well.

A somewhat less severe attack of the same kind is often called "bilious," and is associated with a sallow complexion or an icteric tinge in the eyes, nausea and a furred tongue, with pink deposit in the urine and constipation.

Such symptoms are almost always relieved by a blue pill over night and a black draught next morning, or, in some patients, by two or three grains of euonymin with hyoscyamus, and a dose of Epsom salts with bicarbonate of sodium, or the "white mixture" (MM. c. MS.) of the hospitals.

The *diagnosis* of acute gastritis is generally easy. Both Bamberger and Wilson Fox thought that enteric fever in its early stage is the disease most likely to be confounded with it. What is most important is to remember the possibility of poison, and, particularly in children, of the advent of scarlatina, pneumonia, or some other acute disease, of which the gastric disturbance is only a symptom.

In the *treatment* of an attack, the first thing to be attended to is the diet. In mild cases, rest in bed with complete abstinence from everything but warm diluents for twenty-four hours often effects a cure. In severe and protracted cases, nutrient enemata may be used with advantage. If any nourishment is given by the mouth it should be milk in small quantities, diluted with soda-water or lime-water. Sometimes this is less grateful than hot milk and water with a little cinnamon, or barley-water, or rice-water. Persons who cannot take milk may have water-arrowroot, or veal or chicken broth. As the symptoms subside, jelly or light farinaceous puddings may be allowed. Unless there be great prostration the patient should abstain altogether from alcohol; and champagne is not so suitable in this as in other forms of irritability of the stomach. If a stimulant appears necessary, brandy well diluted is the best.

In sucking infants the quality of the milk must be carefully inquired into. Often they are given more than they can digest, and pain and vomiting ensue. Sometimes it is advisable to keep the child from the breast for a few hours, giving it only a little rice-water or very diluted milk at intervals, or adding lime-water. In infants brought up by hand, the substitution of asses' milk for cows' milk is sometimes effectual; but too often one can save the child only by engaging a wet-nurse at once. The "humanised milk" which is made by diluting cows' milk and adding lactose, is often retained, and nourishes the child well. When no milk is tolerated, we depend on veal or chicken broth, gum-water, and diluted white of egg.

Hot fomentations or linseed poultices may be applied to the abdomen, or a hot-water bottle; or spongiopiline may be used, soaked in hot water, squeezed out, and sprinkled with laudanum.

The administration of emetics and purgatives is an important question. Two cases of Sir Thomas Watson's may here be quoted.

One was that of a person who had been taking large quantities of cream with his tea and coffee. After suffering for several days with severe gastric pain and disorder, he threw up a mass of hard curd like a small cream cheese, and he was at once completely relieved. In the other case a similar fit of indigestion terminated in the ejection of a mass of snuff.

It is certain, therefore, that irritating matters may remain for a considerable time in the stomach. But, on the other hand, as Bamberger remarks, one must not trust too much to the fact that the patient continues to experience uneasy sensations, for these may continue long after their cause is removed, just as one goes on imagining that there is a foreign body in the eye long after it has been expelled. Bamberger, indeed, gives a caution against the use of emetics or purgatives, which, he says, have caused catarrh of the stomach much more often than they have cured it. Fox says that an emetic may be administered "when the presence of undigested food is indicated by cramp-like pain, nausea, ineffectual attempts to vomit, and faintness;" but adds that antimony and even mustard are to be avoided, and that ipecacuanha with large draughts of lukewarm water or of infusion of chamomile is the best emetic in these cases. He recommends, however, rather active purgatives; from three to eight grains of calomel followed by a black draught, or by castor oil; or a dose of blue pill and compound colocynth pill, with a seidlitz powder afterwards. Dr Beaumont certainly found in the case of Alexis St Martin, already referred to, that the administration of calomel was followed by subsidence of the morbid appearances in the gastric mucous membrane* (p. 315).

Whatever may be said as to beginning the treatment of acute gastritis with an emetic or with a purge, when once given it should not be repeated, but followed by sedatives and antacids. For severe vomiting, dilute hydrocyanic acid is the best drug. In ordinary cases bismuth is particularly useful; ten grains of the carbonate with as much bicarbonate of soda and a little morphia will often give immediate relief. Seltzer or Vichy water may be prescribed with much advantage, a pint and a half being given daily, and effervescing medicines are also grateful; but the patient should not be allowed to take them in such quantities as to distend the stomach. During convalescence the remedies for chronic dyspepsia become applicable; but the use of the bitter tonics in protracted cases of gastric catarrh is probably harmful.

Acute gastric distension.—As a rare event fatal collapse may occur after symptoms of gastritis have lasted a few days or a week or two. Dr Fagge described two such cases in the 'Guy's Hospital Reports' for 1872-3, under the name of "acute dilatation of the stomach;" but a better title would

* We ought to be very careful in prescribing purgatives in cases of supposed acute gastric catarrh. I can never forget a case which I diagnosed as of this nature, and which proved to be one of acute suppurative peritonitis. A bank clerk felt poorly one day after having eaten some pears in the afternoon. In the night he woke up with epigastric pain and vomiting. A medical man was not sent for for two days, and when he came he gave a mild aperient. This operated, and a day or two later the sickness subsided. There was a little delirium about the third day. The pulse was at no period of the case over 100; the temperature ranged from 100° to 101°. I was asked to see him on the sixth day. He then appeared to be better; the sickness and pain had ceased; he had begun to take food again. The pulse was about 90, of fair volume; the temperature exactly 100°. Except that the countenance was sunken, and that the eyes were surrounded by deep brown rings, there appeared no reason for alarm, and I concluded that the attack had been one of acute indigestion, and that the patient was in a fair way to recover. Within twenty-four hours, however, he died, and it turned out that there was diffused peritonitis, set up by ulceration of the vermiform appendix.—C. H. F.

perhaps be "acute paralytic distension," for the condition appears to be entirely passive.

One case occurred in a man, aged thirty, who had for some time been in the Hospital under Dr Owen Rees, and was supposed to have incipient phthisis. He was seized with persistent vomiting; he passed no urine, and he gradually became collapsed. On examining him on the third day I found that the abdomen was retracted, and that its walls were rigid. There was dulness above the pubes and half way up to the umbilicus. This might have been attributed to distension of the bladder, but a catheter had been passed, and no urine could be obtained. Moreover, a splashing sound was obtained by manipulation of this region or of the iliac fossæ. He died the same afternoon. At the autopsy the stomach was found to fill the whole abdomen, and to contain a large quantity of fluid. But when removed from the body, it shrank back to about its natural size, showing only a number of white striæ on its serous surface, apparently analogous to "lineæ gravidarum."

The other case was a man aged twenty, who had for a fortnight been suffering from abdominal pains and repeated vomiting. For two days the sickness had ceased, but he was worse in all other respects. His countenance was sunken; his eyes glassy and surrounded by deep rings of pigment; his breath nauseously sweet. His abdomen was generally distended, but the right hypochondrium was flat, and passing downwards and to the right above the navel a line could be seen, which indicated the upper border of the stomach. On manipulation of the lower part of the abdomen there was fluctuation and a splashing sound. Dilatation of the stomach was evident, and the long tube of a stomach-pump was passed down the œsophagus. A greenish fluid was ejected through it and by its side with considerable force. The pump was then connected with it, and no less than *seven pints* of the same fluid were removed by its means. The abdomen became deeply hollow while this was being done. The patient said that he felt much relieved, but he died four hours afterwards. The autopsy showed, as we expected, that the stomach had returned to its natural size and form; but there was a sloughing abscess behind the duodenum, communicating with the bowel.

It may reasonably be hoped that in an uncomplicated case like the former one the prompt use of the stomach-tube would afford a chance of saving the patient's life. The cessation of vomiting is perhaps due to a paralytic state of the gastric muscular coat, comparable with that of the bladder in cases of retention of urine. It is not clear what is the origin of the large quantity of fluid which the stomach contains in these cases.

The physical characters which indicate acute paralytic distension of the stomach during life are—(1) A rapidly increasing distension of the abdomen, which is unsymmetrical, the left hypochondrium being full, while the right is comparatively flattened. (2) The presence of a surface-line which descends obliquely from the left hypochondrium towards the umbilicus. It corresponds with the lesser curvature of the stomach, and moves up and down each time the patient breathes. (3) Dulness and fluctuation in the pubic region, with resonance over the front of the abdomen. (4) The production of a splashing sound on manipulation. In one of the above cases, however, the first two of these signs were absent.

Phlegmonous and other rare forms of gastritis.—Another rapidly fatal disease of the stomach is acute diffused suppuration, a still more rare affection than paralytic distension. Bamberger mentioned it in Virchow's series of handbooks (1855), but Rokitansky had previously described it as suppurative inflammation of the submucous connective tissue, and refers to older cases recorded by Munro, Lieutaud and others, which were collected by Albers. Sometimes the process, instead of being diffused, is confined to one or two spots, and may then be defined as submucous abscess of the stomach; such abscesses, or "gastric carbuncles," as Virchow calls them, may burst into the cavity of the viscus.

Ackermann collected thirty cases, mostly puerperal. Of 45 cases collected by Eichhorst, 38 occurred in men and only 7 in women; the youngest

patient was seventeen, the oldest seventy-six. Suppurative gastritis has been compared to phlegmonous erysipelas or "pseudo-erysipelas." It is septic, and Wilks and Moxon mention hepatic abscess as a result. A typical case of this rare disease was recorded by Dr Fagge in the 'Path. Trans.,' vol. xxvi, p. 81.

The rarest form of all the anatomical forms of gastritis is *membranous* or "*croupous*." It is sometimes the result of extension of diphtheria down the œsophagus, as observed by Jenner, but has also been seen in Bright's disease by Wilks, in phthisis by Fox, and apparently as an idiopathic affection by Niemeyer and Delafield. It is sometimes found associated with a similar membranous inflammation of the colon.

Sloughing, or, as German writers call it, "diphtheritic" gastritis, is described by Billard and Bednar as occasionally seen in new-born children.

CHRONIC DYSPEPSIA.—Two forms of chronic dyspepsia have been described; one called "atonic," the other "chronic catarrhal dyspepsia."

A patient suffering from *atonic dyspepsia* complains of a sense of weight and uneasiness after food, which may last for some hours. The seat of these sensations is usually the upper part of the abdomen; but sometimes, particularly by ladies, they are referred to the chest behind the sternum, or to the back between the shoulders. The former is associated with dyspnœa, the latter with dysphagia. There is rarely actual pain, except as the result of flatulence; there is no tenderness of the abdomen, and pressure rather gives relief. Eructations are not uncommon, and often cause an offensive or rancid taste, perhaps due to the presence of butyric acid. The appetite is generally deficient; there may be a distaste for food of all kinds, even though the want of it gives rise to a sense of exhaustion. Thirst is generally absent; indeed, swallowing liquids often seems to aggravate the symptoms. The tongue is broad, pale, and flabby; it is marked at its edges by the teeth, and is not much furred. The bowels are always constipated.

Beside these symptoms the patient complains of languor and weariness after meals. His spirits are depressed. His pulse is soft, slow when he is at rest, and sometimes intermittent with palpitation on exertion. The skin is moist and there is no fever. The complexion is often pallid, sallow, and muddy, but there is seldom marked anæmia or loss of flesh, except in very chronic cases, and even then it is remarkable how well nourished the dyspeptic patient may be. The urine is copious and clear.

The symptoms of *chronic gastric catarrh* are in most respects similar. The patient complains after his meals of weight and discomfort rather than pain; and there is little or no tenderness of the abdomen. Thirst is a frequent symptom, especially in the intervals between meals; and the sense of exhaustion and internal heat is usually relieved by drinking water. The appetite is capricious, the breath is often offensive, and a disagreeable taste in the mouth is complained of, especially on first rising in the morning. The gums are spongy, red, and inclined to bleed. The tongue is thickly covered with white or yellow fur, but is sometimes particularly red and raw-looking at its sides and tip. The mucous membrane of the pharynx is often granular and secretes a tenacious mucus, which is a source of great annoyance to the patient.

There is no marked loss of flesh, so that emaciation or decided anæmia should make one search carefully for evidence of phthisis, gastric ulcer, or

carcinoma. Slight febrile disturbance is not uncommon at night. The urine is generally scanty and deposits lithates, or sometimes oxalates.

In the dyspepsia of habitual drunkards, vomiting of mucus, especially in the morning, is one of the principal symptoms, and it is probable that this kind of vomiting is always an evidence of a catarrhal state of the gastric mucous membrane.*

In severe cases of dyspepsia, when vomiting occurs, the ejecta are intensely sour—not from an over-secretion of acid by the stomach, but from the formation of lactic, butyric, and acetic acids by fermentation of starchy and saccharine food. These acids are often developed with great rapidity, and in such large quantity, that when the patient vomits the throat burns, the teeth are set on edge, and the eyes smart, just as though strong acetic acid had been taken into the mouth. At the same time the sour smell of acids, volatile at a moderate temperature, is diffused through the air. A further evidence that fermentation is the cause of the formation of acid in such cases is the fact that gas is evolved, which has been found to consist of a mixture of carbon dioxide and a volatile hydrocarbon.

A "stomach-cough" is still regarded as a frequent malady, but most cases so called are either an accidental coincidence of two common disorders, or are really cases of the dyspepsia of early phthisis. Cardiac palpitation, for which patients often come, supposing the heart to be diseased, is one of the commonest symptoms of flatulence with an overloaded stomach.

One result of chronic catarrh is that the muscular walls of the stomach lose their power, and allow of gastric dilatation, and this further interferes with digestion by not bringing each part of a meal into contact with the secreting mucous membrane. The food therefore lies as an inert mass in the stomach, and then the fermentative changes just described begin, and acidity from lactic, acetic, or butyric fermentation of carbohydrates causes heartburn and acid eructation. At the same time carbonic acid gas, hydrogen, and phosphuretted or sulphuretted, or, occasionally, carburetted hydrogen are developed and increase the distension. This flatulence is one of the most common and distressing results of dyspepsia by its mechanical distension of the abdomen. The eructations produced are most often of tasteless and odourless carbon dioxide, but sometimes changes in the constituents of the food proteid lead to the evil odour of rotten eggs, and more rarely so much carbonic hydride is evolved that the eructations become inflammable.

Some of the more important of these conditions may be regarded as complications rather than ordinary symptoms of chronic dyspepsia, and will therefore be considered again particularly as to their pathology and treatment in a later part of this section (*infra*, pp. 328, 329).

The morbid anatomy presented by the mucous membrane after death is believed to distinguish atonic dyspepsia from chronic gastric catarrh. In the former the lining of the stomach is thin and transparent; in the latter it is almost always thickened and sometimes indurated, so that it can be stripped off in large pieces, or the submucous tissue may be white and fibrous with increased rather than diminished difficulty in separating the coats. The mucous membrane near the pylorus is often mammillated in cases of chronic gastric catarrh; but this condition may also be found in a healthy stomach, owing to contraction of the muscular fibres round the

* Frerichs showed that starchy substances are sometimes converted in the stomach into a tenacious glutinous material which may closely resemble mucus. A few drops of tincture of iodine at once decide the point.

secreting glands. The most characteristic change in cases of chronic catarrh is ash-grey pigmentation of the gastric mucous membrane. This, when closely examined, is seen to depend upon the presence of numerous minute specks scattered thickly over it. Under the microscope they are found to consist of granules of pigment (doubtless originally derived from hæmatin), which are deposited in the connective tissue between the tubes, or in the epithelial cells.

The gastric glands are affected in both forms of chronic indigestion, catarrhal and atonic dyspepsia. The secreting tubes are found to be shrunken and wasted, and to have undergone fatty degeneration. Their epithelium may have almost entirely disappeared, being represented only by granules and fat globules. Cysts are occasionally found, which are probably the result of distension of parts of the tubes that were constricted off from the rest.*

Fenwick found that when the secreting tubes are atrophied the digestive power of the dead mucous membrane is much less than under normal conditions.

Redness of the mucous membrane has often been mentioned as one of the appearances characteristic of catarrhal gastritis; but, as a rule, there is no redness after death. No doubt the most intense injection of the gastric mucous membrane is often found; but these are cases of chronic catarrh from *disease of the heart*; the stomach is lined with a thick layer of mucus; and after this is washed away, the surface is seen to be of a vivid crimson colour, either uniformly or in spots. Ecchymoses are often present at the same time, and still more frequently they are simulated by small patches, due to arborescent injection of the branches of some minute vessel. When effusion of blood occurs into the submucous tissue the gastric juice sometimes dissolves off the corresponding part of the mucous membrane; and a minute ulcer is the consequence, the floor of which is occupied by a layer of black coagulum. This process is known by the name of *hæmorrhagic erosion*. The redness and congestion in such cases is due not to inflammatory but to passive congestion.

Another form of gastric catarrh in which the stomach is often found intensely reddened is that which results from drink. An unskilled pathologist may easily be led to suspect the presence of an irritant poison in cases of this kind. Dr Fagge once made an autopsy in a case of a young man who had suddenly died in a railway train early in the morning. There was no cause for his death discovered, but the stomach was intensely reddened and ecchymosed. It was clearly ascertained that there had been no foul play, and there appeared to be little doubt that the abuse of stimulants had been the cause of the gastric irritation. As Wilks and Moxon remark, no mere redness and injection of the gastric mucous membrane is enough to prove the presence of an irritant poison. There must either be chemical evidence of its presence or actual ulceration.

Pathology of chronic dyspepsia.—As in most disorders of the human frame, dyspepsia may be due to an external irritant which disturbs the action of the machine, or to some internal weakness or defect which renders

* With reference to the early observations of Habershon, Handfield Jones, Fenwick, and Wilson Fox, it must be said that the fatty or granular degeneration of secreting epithelium is a condition which has often been supposed to exist from want of knowledge of the normal or the *post-mortem* changes of these delicate and changeable structures. All observations made with low powers on the tissues, not perfectly fresh, and without the aid of osmic acid and the other modern histological appliances, need confirmation.

the ordinary physiological stimulus to action irritating and disturbing. Cold or dusty air reaching the air-passages provokes bronchitis or sore throat; but when a chronic catarrhal inflammation is once established, the purest and warmest air becomes an irritant to the over-sensitive organs. So a vigorous digestion will deal with a large and hastily swallowed meal for once, but if the excess is repeated, chronic gastric catarrh is set up, and at length the dyspeptic may be no longer able to digest the most bland and carefully chosen food.

The digestive apparatus being healthy, indigestion may arise from the food put into the stomach being too large in quantity; or imperfectly comminuted by mastication; or imperfectly insalivated; or not cooked enough, or over-hardened by cooking. Also, digestion may be checked by accumulation of its own products in the stomach.

The quality of the food may cause dyspepsia. The late Dr Chambers cited the case of a poor needlewoman who had subsisted for a year on bread, potatoes, and tea, getting sometimes a little bacon but hardly ever other kinds of meat, and who suffered so much from dyspepsia that she dreaded to eat. For such a patient, and for many hypochondriacs, variety and attractiveness of food are essential. In other cases indigestion always follows some particular article of diet, such as fatty matters or soups. Chambers attempted to describe "indigestion of vegetable food," "of albuminoid food," "of fatty food," and "of watery food" separately, but with no great success. He gives an instance of a lady who from childhood had never been able to take roast beef without afterwards having heartburn, and this he attributed to the fat which lies between the muscular bundles in stall-fed beef. With regard to digestion, however, many idiosyncrasies are met with, of which it is not possible to give explanations, but which the physician must not overlook. Strawberries disagree with some persons, eggs with others, melted butter with many. Twice-cooked meats, potatoes, new bread, cauliflower, and jams or candied fruits are some of the most frequent sources of indigestion.

Theoretically the following are all possible or probable causes of indigestion of wholesome food:—want of secretion of hydrochloric acid or of pepsin, or over acidity of the gastric juice, or over secretion of mucus; want of active movements of the stomach walls; fermentation of food, leading to development of carbon dioxide, of acetic or of butyric acids; irritability of the gastric mucous membrane from inflammation or from neurotic hyperæsthesia, leading to pain or to vomiting, or to inhibition of secretion or movement, by reflex influence.

Of these causes, lack of hydrochloric acid is certainly one; when deficient or absent, in so-called atonic dyspepsia, there may be mere want of the necessary acid medium of digestion, or there may, in the presence of lactic and other organic acids, be reaction enough; but these form an imperfect substitute for pepto-hydrochloric acid. Excessive acidity, again, acts as an irritant, and produces gastric catarrh. Want of movement of the muscular walls of the stomach prevents due mixture of the digestive secretion and of the food, and allows it to remain in the stomach, with accumulation of the by-products of digestion, instead of being regularly propelled through the pylorus. Fermentation and the flatulent distension which it produces, aggravate this want of power in the muscular coats of the viscus. Lastly, paralysis of secretion and movement alike may be caused by sudden emotions of anxiety, anger, or distress, so that a meal may lie a dead weight in

a cavity which has become an inert bag without chemical or mechanical influence on its contents.

Faults in diet may be regarded as the most frequent causes of dyspepsia from chronic catarrh of the stomach: want of secretion and want of movements are probably important causes of atonic dyspepsia; and direct inhibition of cerebral origin is also without doubt frequently operative. It is possible that a disposition to dyspepsia is sometimes inherited; and doubtless sedentary occupations, constant pain, prolonged anxiety of mind, or depression of spirits, are predisposing causes of chronic dyspepsia.

Other frequent causes of indigestion are: over-eating, the habit of waiting too long between the meals, *imperfect mastication* of the food (the state of the teeth should always be looked after), taking too much fluid (especially cold water) with the meals, hasty eating, solitary meals, the *abuse of stimulants*, of condiments or of tea, excessive smoking, and taking bodily exertion or making mental efforts while digestion is going on.

Although atonic dyspepsia sometimes attends senile decay, the ordinary chronic dyspepsia which comes on after a meal, and usually goes with flatulence and constipation, is by far most frequent in young adults. It is very rare in children, though they furnish some of the most striking cases of acute gastric catarrh (p. 314). Young men and unmarried girls are extremely liable to it, and it often continues through the first half of adult life. In women it is aggravated by uterine irregularities, and sometimes begins about the menopause; but most men suffer less after fifty, and in old age, notwithstanding the loss of teeth and sedentary habits, it usually disappears; so that, as Sir Michael Foster puts it, the old man who suffered martyrdom from dyspepsia throughout his active life, "now eats with the courage and the success of a boy." These facts seem to show that while indigestible food is certainly the occasion of dyspepsia, it is not its essential cause; and also that the histological changes described above do not exist or are not operative in the majority of cases.

The mental interest and anxiety which belong to active life, appear to spoil digestion by a kind of inhibition, either of the vaso-motor or of the secretory nerves, or possibly of the muscular walls of the stomach; and this quite apart from the irregular meals and hasty eating and over-use of stimulants which often accompany business life. The careless and unharassed periods of life—youth before the toil and anxiety have begun, and age when both have passed—are the periods of unconscious and therefore successful digestion.

The *diagnosis* between atonic dyspepsia and chronic gastric catarrh is unsatisfactory, but, after all, it is of little consequence, for with practical experience one learns to adjust one's remedial measures to the necessities of the case without always attempting to draw distinctions between maladies of which the true pathology is still obscure, and far less important than their clinical characters from the point of view of prevention and treatment.

The diagnosis between chronic dyspepsia and carcinoma of the stomach is much more serious. We must remember that the early symptoms of cancer are those of mere indigestion, and that the possibility of malignant disease must never be overlooked in any dyspeptic case that is protracted or severe. Vomited matters should always be examined chemically as well as microscopically, and we must examine the patient in the recumbent

posture, and with the abdomen exposed to view as well as to manipulation.* (cf. *infra*, p. 359).

The *prognosis* in dyspepsia depends chiefly on whether its causes can be removed. Judicious advice scarcely ever fails to give relief if it is followed; and most young adults are cured, but the disorder is apt to revive, and some cases of long standing seem to be beyond reach of treatment.

Treatment.—It will be convenient to classify the forms of chronic indigestion for purposes of treatment as follows:

(1) The so-called “bilious” dyspepsia of young adults, corresponding to what is called chronic gastric catarrh, with constipation, furred tongue, and frontal headache.

The first prescription for this, perhaps the commonest kind of dyspepsia, is to eat less, to eat slowly, and to take stimulants sparingly during and none between meals.

Next come injunctions to secure as much as possible of open air and exercise, to rise early, and to bathe every morning.

Thirdly, the drugs most useful are alkalies and gentle laxatives. Sir William Roberts recommended a lozenge of nine grains of bicarbonate of soda, with one grain of common salt to promote the flow of saliva. In the more irritative cases bismuth is almost always useful, particularly when there is nausea or vomiting without constipation. A drachm of the *Liquor* of the *Pharmacopœia*, or ten grains of the subnitrate, may be administered three times daily. At the same time if there is pain, small doses of morphia may be given with great advantage.†

Laxatives are almost always needed. A useful dinner pill contains aloes and extract of *nux vomica*, to which a little *ipecacuanha* may often be added with advantage. Another is *euonymin*, with *Pil. Rhei co.* or *Pil. Col. et Hydr.*

Among the natural mineral waters of Great Britain, the most useful in cases of chronic gastric catarrh were said by Wilson Fox to be those of Harrogate, Bath, and Leamington. Trousseau recommended *Plombières*, *Vichy*, and *Bagnères de Bigorre*, in France; while *Carlsbad*, *Marienbad*, *Wiesbaden*, and many others are advised by German writers. The water itself is one chief element of value, apart from its more or less aperient salts.

Where there are less inflammatory symptoms with more constipation and flatulence, a sallow complexion, and yellowish fur on the tongue, sodic carbonate is indicated, together with small doses of blue pill, *euonymin*, or *iridin*, to which *taraxacum* or *ipecacuanha* may be added. A brisk purge once a week is better in these cases than frequent aperients, and nothing is better than *magnesian sulphate* in hot water.

(2) The chronic catarrhal gastritis of drunkards. This is characterised by nausea, thirst, morning diarrhœa, a yellow furred tongue, and the

* I can never forget the case of a gentleman in whom, as soon as his shirt was raised, the existence of obstruction at the pylorus was indicated by the obvious peristaltic movements of a dilated stomach, but who assured me that his abdomen had never before been examined, although he had been under the care of more than one specialist.—C. H. F.

† The following formula is useful: *R. Bismuthi Carb. gr. x, Sodii Bicarb. gr. v, Pulv. Tragac. gr. x, Liq. Morph. At. mx, Aq. Menthæ Pip. ad ʒj.*

In long-standing cases Wilson Fox recommended the oxide of silver (in doses of one grain to two grains), alum (in doses of two to five grains), tannin or decoction of oak bark, and *matico*. A valuable remedy in cases of this kind is *magnesia*; it may be prescribed with three-minim doses of dilute hydrocyanic acid, and equal parts of lime-water and cinnamon-water.—C. H. F.

general indications of intemperance in the eyes, the skin, and the nervous system. In addition to prompt and complete abstinence, it is best treated with free diluents, soda-water, "imperial drink," and any mild bitter infusion, as buchu, that the patient will take. A blue pill twice a week, and alkalies with *nux vomica*, are valuable aids.

(3) Gouty dyspepsia. This also is probably due to gastric catarrh. It is usually benefited by colchicum and alkalies, particularly potash salts and other diuretics, with copious libations of water.

(4) Cases of chronic dyspepsia with constipation and flatulence, often with heartburn and acidity, but without a furred tongue and without nausea, may be called "atonic." They are more common in women than in men, and are often seen in early cases of tuberculosis. Probably the chief cause of the symptoms is want of active movements of the stomach and bowels; this leads to distension with gas, delay of food in the stomach, slow digestion, and slow peristalsis.

The exact pathology of gaseous distension of the stomach is not yet fully made out. Hysterical flatulence probably depends in part on swallowing air; other cases result from chemical decomposition in the stomach and duodenum of sodium carbonate by hydrochloric acid (v. *infra*, p. 332). Sometimes it is probable that there is no abnormal amount of gas; the muscular walls of the containing viscera yield, and the gas naturally present expands as a physical result of the diminished pressure.

In these cases the diet must be somewhat scanty, but regular and varied. It should be dry in quality, soups and broths being apt to cause flatulence; and potatoes, cauliflowers, and even bread, should be replaced by boiled rice, spinach, and toast or biscuits. Meals should be eaten slowly, and no cold water should be drunk with food, but either bitter ale or a little sound wine. Exercise out of doors should be followed by half an hour's rest before a meal, lying down on a bed or couch; for a tired body does not digest well.

The most useful drug in flatulent atonic dyspepsia is *nux vomica*, given after food in ten drops of the tincture with cardamoms and peppermint, or before meals as half a grain of the extract with the compound rhubarb pill.

(5) The dyspepsia of anæmia in general and of phthisis in particular. This also is of the "atonic" kind, and is best treated by the milder preparations of iron with bitter infusion, and sometimes by small doses of arsenic. In cases of chlorosis, iron and aloes are the cure for dyspepsia and anæmia together.

In many cases of atonic dyspepsia, and particularly in the later stages of the affection, the dilute mineral acids are very useful, especially the hydrochloric. In doses of ten or fifteen minims, properly diluted and taken with or after the meals, it prevents the sense of weight and oppression which would otherwise be experienced by the patient, and relieves flatulence arising from fermentation of the food. Trousseau speaks very highly of this remedy, and mentions that he learnt its use by sitting at dinner next to a tourist, who said that he never travelled without the acid, of which he took a few drops after each meal.

Pepsine, again, is often useful in cases of atonic dyspepsia; but Dr Pavy has shown that care is required in obtaining it at the druggist's, since much of what is sold in London is devoid of any active properties. The acid solution of pepsine in glycerine, taken in drachm or two-drachm doses after a meal, is valuable in atonic flatulent dyspepsia.

The stronger preparations of iron often disagree with dyspeptic patients, and the same is the case of quinine. When there is much flatulence, creasote, thymol, or carbolic acid is useful in the form of pills.

Brighton is a good place for persons affected with this form of dyspepsia; also Scarborough, Folkestone, Margate, Eastbourne, Malvern, Tunbridge Wells, and Ilfracombe. A stay in Switzerland at a height of 1500 to 3000 feet is often a sufficient cure.

(6) Children are, as before noticed, comparatively little liable to dyspepsia. So common, however, is the complaint, that even in them cases frequently call for treatment. These depend almost always on one of two causes: either unsuitable food, or general anæmia, in which the stomach shares. The first we see in the discomfort of an infant fed too soon on starchy food, or given material fit only for an older child; and again in the acute gastritis of a schoolboy who has eaten too many apples or tarts, or other "trash." The immediate remedy is an emetic, if the stomach has not unloaded itself, and the subsequent treatment is better choice of food in quality and in amount.

The anæmic or atonic form of dyspepsia in children is seen in cases of rachitic, tuberculous, or other kinds of marasmus, and is best treated by steel wine, fresh air, and sometimes by wine or malt liquor with meals.

(7) Senile dyspepsia, when not due to organic disease of the stomach, is usually atonic, and is benefited by wine or spirits at meals, and by dinner-pills of rhubarb or aloes with capsicum. The quantity of food must generally be reduced.

Diet.—As a rule a dyspeptic should take three meals daily, at one of which freshly cooked meat should be eaten. White fish, mutton, poultry and game (but not hare or rabbit), are most suitable; pork and veal, and salted or preserved meats are usually best avoided; and even beef is to some stomachs too rich for easy digestion. It is lightest when eaten cold and rather underdone. Eggs agree well with some dyspeptic patients, particularly in custards or puddings; but others are unable to take them, particularly when hard-boiled. Potatoes should be taken sparingly, and only if well boiled, floury, and not young; turnips, parsnips, and Jerusalem artichokes—in fact, all solid roots are better avoided; but green vegetables, as spinach and asparagus, and in most cases onions, may be taken in moderation. Peas and beans often cause flatulence, and so do cauliflower and other Cruciferæ. When vegetables are found to disagree, their place may be supplied by rice or macaroni. New bread should never be eaten by persons who are subject to indigestion; it should be stale, or may be replaced by rusks, or toast or biscuits. Light farinaceous puddings generally agree well with dyspeptic patients. Every one is agreed that lobsters and crabs, cheese, nuts, pickles, cucumbers, muffins, melted butter, and sauces are to be strictly forbidden; but even here a concession may sometimes be made to individual experience, *e.g.* of cheese or of pickles in atonic dyspepsia. A large amount of fluid should not be drunk at meals. Cocoa, hot water, or milk and water, may be taken instead of tea or coffee. A moderate quantity of wine (claret, hock, or, in some cases, champagne) may be allowed at meals, but with some patients any wine is apt to cause acidity, and this is probably one reason for the present fashion of limiting the patient to weak brandy (or whisky) and water with his meals. Malt liquors are often found by the patient's experience to cause flatulence, a sense of weight, and more or less constipation; but in certain cases a light,

well-hopped, and not effervescing bitter ale is an excellent stomachic; and some young women can take porter without discomfort who bear no other form of stimulant.

Whatever causes flushing of the face after the meal is bad, as hot soup, hot tea, mustard, pepper, curry, forcemeat, and candied fruits. Food is to be taken slowly, time being allowed for mastication and the due admixture of saliva; and on this account it is advisable that the patient should have his meals in company with other persons.

There are certain effects of gastric disorder which, although often associated with the ordinary symptoms of dyspepsia, yet also occur by themselves, and may be so serious as to be regarded as independent affections.

Gastric paresis.—Dilatation of the stomach has already been mentioned as a rare acute condition. It is far more frequent as a result of chronic gastric catarrh with flatulence. We shall see that it often occurs as the result of pyloric obstruction (infra, p. 360); but without any demonstrable lesion of the pylorus, the stomach is often found immensely distended after death, and the diaphragm pushed up. This condition has more than once led to fatal syncope.

Constant fulness, however, is more often caused by gas than by liquid or by food, and it is the constant fulness which at last overcomes the elasticity and the contractile power of the muscular walls of the stomach. The same result, judging from the analogy of the heart, the colon, and the bladder, is probably helped by chronic catarrh of the subjacent mucous membrane. At all events the two conditions are frequently associated.

Clinically we recognise gastric distension by a greatly increased range of gastric, usually tympanitic, resonance, particularly downward, below the umbilicus and to the left. The position of the stomach is also changed, its long axis becoming more horizontal and the greater curvature coming forwards. At the same time the liver is pushed up and the diaphragm displaced, so as to interfere with the due action of the lungs. On gentle succussion, splashing is heard, which points to a large cavity containing gas and liquid, and this can scarcely be anything but a dilated colon or dilated stomach.

The actual degree of enlargement may be ascertained by Leube's method of introducing a sound into the stomach by the mouth. Another method, practised long ago by Piorry, is to fill the stomach with fluid. Less fallacious than either of them is Frerichs' plan of filling the stomach with gas by means of an alkaline and acid solution: a teaspoonful of bicarbonate of soda, followed by one of tartaric acid. The limit of the tympanitic percussion-sound gives the extent of the stomach more accurately than the dulness caused by a litre of water, and avoids the fallacy of the sound slipping along the greater curvature to the pylorus. There remains, however, the discomfort of the proceeding, and the serious danger of all such manœuvres when there is any possibility of ulcer, cancer, or softening of the stomach walls being present.

The effects of dilatation of the stomach are to delay the food for a long time, to favour fermentation and thus to aggravate the distension. The patient experiences all the distressing sensations of weight and fulness caused by a heavy meal, and often relief is only obtained by vomiting.

The treatment of chronic gastric dilatation consists in avoidance of fermentable food, particularly large quantities of starch and cellulose, in the

use of thymol, creasote, salicylates, sulpho-carbolate of soda, or other antiseptics, and in administration of nux vomica to act on the muscular coats of the viscus. Calomel in small doses is often a valuable adjunct; cardamoms, lavender, Sp. Armoraciæ Comp., and cajeput oil are useful as carminatives.

But the most effectual method is daily emptying the stomach by the use of a flexible tube filled with water, and bent over so as to act as a siphon. This is far better than a stomach pump, and used in suitable cases and with proper care is both safe and effectual. The treatment was first proposed by Professor Kussmaul, of Freiburg (now of Strassburg), in 1868. A long rubber tube filled with water is introduced into the stomach every day. On lowering the longer half, the water flows out and the contents of the stomach follow it. As much of the contents as will come readily should first be withdrawn. Some tepid water is then to be injected and afterwards withdrawn again, and the process should be repeated two or three times until what returns is almost clear. Weak solutions of carbonate of soda, or of permanganate of potass, or even of creasote, may afterwards be injected. Dr Schliep introduced the practice into England in 1872 (Clinical Society's 'Transactions,' vol. vi, p. 41), and it has met with great success. The first passage of the tube is exceedingly disagreeable to the patient; but before long he becomes accustomed to it, and he is even glad to pass it himself, so great is the relief which he experiences from its use. The vomiting often ceases entirely; there is usually great diminution of pain; the appetite improves considerably; the patient becomes more cheerful; he regains much of the flesh and strength that he had lost, and he is no longer troubled with constipation.

Gastralgia.—Perhaps a still more important gastric symptom is severe abdominal pain. This has been already mentioned as one of the symptoms of dyspepsia, particularly in its more acute form; but it may also occur independently. Several names have been applied to pains in the stomach, but not always in the same sense. Cullen employed *cardialgia* for the less severe varieties, which would be called "heartburn" or "acidity," while he described as *gastrodynia* a more violent but also more transient pain, such as would be spoken of as "cramp" or "spasm" of the stomach. Most English writers followed Cullen in the use of these names, but some German text-books have used them with meanings reversed. *Gastralgia* is a term used chiefly by French writers, with a wide range of application. It is probably impossible to define two words separately which have the same original meaning, viz. "Stomach-ache," and a mere difference in severity is not a good criterion between the two; neither term has classical authority.

There is one kind of gastric pain which comes on when the stomach is empty, half an hour or so before meal time, and is quickly relieved by even a small quantity of food. Sir Thomas Watson knew a clergyman who was much harassed by its occurrence daily until he found by accident, after having tried a round of drugs, that eating a small biscuit would at once appease it. A dose of the aromatic spirits of ammonia, or carbonate of magnesia, will sometimes remove the pain in a moment. According to Budd, the gastric pain due to an empty stomach is sometimes accompanied by slowness of the pulse and coldness of the surface; the recumbent posture relieves it, and hydrocyanic acid is the medicine he recommends.

Anstie regarded it as a form of neuralgia, and prescribed five or ten minims of the tincture of *nux vomica* three times a day, or sometimes gave $\frac{1}{80}$ to $\frac{1}{50}$ of a grain of strychnia by subcutaneous injection. One case in which this remedy effected a cure was that of a patient who had attempted suicide on account of the agonising pain he endured.

When gastric pain comes on soon *after* food, the diagnosis requires great care, for pain of this kind is often produced by organic disease. The strongest indication of the presence of ulceration is the fact that the pain begins as soon as the food is taken, and lasts until digestion is completed or until vomiting occurs. But, as we shall see, these characters may be wanting in cases of gastric ulcer, whether simple or malignant; while, on the other hand, severe and protracted pain is sometimes complained of after every meal by some patients who are free from any serious disease.

Abercrombie described another form of gastric pain which only begins from two to four hours after a meal, and lasts for several hours; he thought that its seat was in the duodenum. His treatment consisted in giving two grains of sulphate of iron, with one grain of aloes and five grains of aromatic powder, three times daily. Watson found he could generally remove it by giving an alkali, or letting the patient swallow a cup of warm tea; he supposed it to be due to the continued secretion of gastric juice after the food has passed through the pylorus, and adds that the pain may often be prevented by an alkali in some aromatic water taken after dinner. Trousseau speaks of this "duodenal" pain as being often attended with a sense of sinking at the stomach, a craving appetite, and a great feeling of weakness. Constipation usually accompanies it, but sometimes diarrhœa. Bismuth, bicarbonate of soda, and a few drops of *Liq. Morph. Acet.* in chloroform water are useful in such cases, or an alkaline lozenge may be taken.

Pain in the neighbourhood of the stomach sometimes bears no relation to the times at which the meals are taken, or to the stage which the process of digestion has reached. For this kind of pain—coming on at uncertain intervals in violent paroxysms—Cullen reserved the name of "gastrodynia." It is often accompanied by distension, anxiety, and extreme restlessness. In women hysterical symptoms are frequently present, and the stomach is sometimes filled with enormous quantities of gas.

Gastralgia may recur at irregular intervals for a long period without appreciably affecting the patient's health. It cannot in such cases be due to any active disease; but it may perhaps be the indirect result of long past pathological changes. Bamberger speaks of the cicatrices of gastric ulcers as giving rise to paroxysmal attacks of pain, probably by irritating filaments of nerves embedded in them. Dr Fagge once made an autopsy in the case of a lady who had for years suffered from a pain in the back, which was supposed to be connected with an abscess near the sacrum when she was a child. All the parts in front of the spine—the aorta, the vena cava, and the nerves—were embedded in a dense mass of cicatricial fibrous tissue. In another case the destruction of a hydatid in the liver was followed by severe pain, probably due to pressure on nervous filaments during the contraction of the cyst.

But a person may suffer from gastrodynia for many years, and an autopsy show nothing to account for it. Bamberger gives a case of this kind. It occurred in a powerful man who for nine years had been subject to frequent attacks of the most violent pain in the stomach, lasting for

days, or even weeks, and attended with great prostration and temporary loss of flesh. He died of acute phthisis; and slight dilation was the only morbid change found in the stomach.

Gastralgia has to be distinguished from pain arising in the colon, which may closely resemble it; the diagnosis will be discussed when we deal with colic (*infra*, p. 367).

According to Briquet, the abdominal muscles are often the seat of pain, without any affection of the viscera. He lays stress on the circumstances that superficial tenderness is present, that the left recti and obliqui abdominis are the muscles principally affected, that not only their fleshy parts but also their tendinous attachments are concerned, and that dorsal pain and tenderness in the vertebral groove often exist at the same time. But we shall hereafter see that rigidity of the upper part of the rectus with tenderness is a very common effect of organic disease of the stomach, and there seems to be no reason why it should not also occur when the pain is of functional origin. True myalgia of the abdominal muscles would be recognised by its being increased with movement of the body.

In a case of Dr Fagge's, a pain in the left hypochondrium, which had long resisted other treatment, was again and again removed by quinine and iron, and in that case the pain was probably neuralgic; for there seems no doubt that some cases of severe paroxysmal gastrodynia which have no organic explanation may be at least provisionally regarded as gastric or abdominal neuralgia. These cases may be compared with the "gastric crises" of tabes (*cf. supra*, vol. i, p. 694).

Lastly, pain situated in the epigastrium, over the stomach, may be severe, continuous, and last for a long time; and yet be due to no disorder of the stomach, functional or organic, but to aneurysm of the abdominal aorta or disease of the dorsal vertebræ. Some striking instances of epigastric pain due to the latter cause are related by Hilton.

One was that of a boy who for two months had been complaining of severe pain just above the pit of the stomach, and who used to walk about with his hands placed over that region, and with the body a little inclined forwards. It seemed as though he were suffering from irritation of some of the abdominal organs, and he had been treated on that supposition, but without much benefit. The pain was relieved when the boy lay down. Its seat was not to one side of the body more than to the other. Disease was detected between the sixth and seventh dorsal vertebræ, and pressure on their spines excited the pain in front. He was kept in a recumbent posture for four or five months, and was then completely cured.

Another case was seen by Mr Hilton with Dr Addison. A Westminster boy had pain at the pit of the stomach and occasional vomiting. He was found to have disease between the same two vertebræ; he too was easy when lying in bed. He was made to lie down uninterruptedly for two or three months, and made a good recovery.

Continuous and severe gastrodynia without signs of gastric disorder is often intractable. Watson recommended the application of a mustard poultice to the epigastrium, and the administration of a carminative (such as a few drops of cajeput oil suspended in mucilage) or of sedatives, such as hydrocyanic acid. Kussmaul recommended washing out the stomach with large quantities of water containing carbonic acid gas. Leube extolled the benefit of a galvanic current passed between the epigastrium and the spine. The writer has found most benefit from compound tincture of cardamoms, laudanum, or bismuth with morphia, and externally from a small blister on the pit of the stomach.

Alterations of the appetite are often due to gastric disorder. *Anorexia**

* *Anorexia* (ἀνορεξία), want of desire of food.

may indeed be a symptom of almost any kind of disease. It accompanies the loss of digestive power, whether from pyrexia or from some local cause.

Beaumont found that when Alexis St Martin was feverish, the secretion of the stomach was diminished or suppressed, and food remained undigested for twenty-four or even forty-eight hours; and patients suffering from acute diseases lose desire for food.

The appetite may be improved by the administration before meals of bitters with mineral acids. A pill of capsicum, nux vomica, and gentian is useful for this purpose, but should never be prescribed until any gastric catarrh or portal congestion has been corrected.

Excessive appetite is called *bulimia*.* It is supposed to denote the presence of worms in the intestines, and is a symptom in diabetes. It has already been mentioned as accompanying one of the forms of gastralgia.

Lastly, *pica*† is the name given to a perverted state of the appetite, in which "foreign bodies" are greedily swallowed, or filthy matters, such as horse-dung. It occurs either as a variety of the "longings" of pregnant women, or in hysterical, half-demented girls, or as a symptom of mania.

Fermentation and flatulence.—The contents of the stomach and intestines are liable to certain chemical changes due to the action of ferments on the carbohydrate part of the food. Both acetic and alcoholic fermentation may take place in the stomach, but the products are small in quantity. More common and important are the lactic and the butyric fermentations of starch and sugar. Acids are formed, and carbonic anhydride is set free. Occasionally hydrogen compounds with carbon, phosphorus, or sulphur are produced, and these gases are added to the carbon dioxide and to the nitrogen and oxygen which is accidentally swallowed with food. Sir William Roberts believed that only a small portion of carbonic anhydride is due to fermentation. Besides swallowed air, he found the most important source of gastric flatulence in the decomposition of sodium carbonate swallowed with the saliva by the acid of the gastric juice, and a third in a similar decomposition, in the duodenum with regurgitation of the CO_2 . When the stomach walls are weak and dilate, the gases expand and produce the condition known as *tympanites* or *meteorismus*.

That there may be considerable and even extreme flatulence, independently of any fermentation, is shown by its occurring in cases of starvation, and of intentional abstinence.‡

Flatulence is developed most often about an hour after a meal, but in some severe cases the gaseous distension of the stomach begins immediately, or even after the first mouthful. No doubt in some cases of hysterical flatulence air is swallowed down by involuntary efforts. In confirmed flatulent dyspepsia, the fulness may increase until oppression and dyspnoea, or even orthopnoea come on in violent paroxysms during the night.

It may here be mentioned that, although the normal gastric secretion is to some extent antiseptic, it only hinders or prevents the multiplication and secretion of septic bacteria without destroying them. Many forms of schizomyzetæ have been found in the contents of the stomach, and Dr Mac-

* *Bulimia* (*βουλιμία*), lit. ox-famine, means excessive hunger, a ravenous appetite.

† *Pica* is a translation of *κίσσα*, the jay (*Pica glandaria*), or the magpie (*Pica caudata*), a word applied by the Greek physicians to the indiscriminate craving of morbid appetite.

‡ Jeremy Taylor speaks of the liability to this condition as the result of fasting, and calls it the work of "a windy devil."

fadyean has traced *Staphylococcus aureus* through the stomach from the mouth to the intestines.

According to Dr Sidney Martin's experiments, gastric juice, with its due amount of hydrochloric acid, readily destroys the vibrio of cholera (as was found by Koch in experiments on guinea-pigs); but the bacillus of anthrax less easily, while that of tubercle has, unfortunately, a high power of resistance.

Eructation and regurgitation.—In many gastric disorders gaseous or fluid matters are returned from the stomach through the œsophagus. Generally gas alone arises in eructation or belching, but sometimes a small portion of gastric juice or half-digested food as well, which is instantly carried back into the stomach. This is of course associated with flatulence, and often with heartburn.

When only a few drops are regurgitated, the hot acid taste is unpleasant in the mouth, but there is no true vomiting; a little secretion is accidentally belched up, entangled in the bubbles of gas which form the eructation.

In quite distinct and very rare cases, however, a large part of the food is habitually brought back into the mouth, deliberately remasticated, and swallowed a second time. Some years ago there was a patient of Dr Pavy's in Guy's Hospital who had this habit of ruminating. Copland collected many cases of this kind. In one patient the rumination began in from fifteen minutes to an hour after almost every meal; each bolus of food came up during an act of expiration, with the same taste and flavour as when first swallowed; there was neither nausea nor pain, and he masticated it a second time with pleasure.

Pyrosis.—A somewhat analogous affection is what is called waterbrash, or *pyrosis*.* It is said to be very common in Scotland, owing to abundant consumption of oatmeal, and to be still more common in Norway, Sweden, and Lapland. Cullen, who was very familiar with pyrosis, described it as coming on usually in the morning when the stomach is empty. According to him, it begins with a severe pain at the pit of the stomach, which, after continuing for some time, brings on eructation of a thin watery fluid in considerable quantity. The fluid is sometimes acid, but is often absolutely tasteless. The repetition of eructation seems at length to give relief to the pain, and the attack is then at an end. It is apt to return more or less frequently for a considerable length of time. Cullen says that the complaint occurs chiefly among the lower classes, in women more than in men, and between puberty and middle age rather than at any other period of life. He speaks of it as often unattended with any symptoms of dyspepsia.

In England pyrosis seldom appears as an independent malady, apart from other effects of gastric disorder, but Watson speaks of one remarkable case in which no less than three pints of a thin tasteless liquid were brought up every day.

There is difference of opinion as to the nature of this secretion, and possibly it is not always the same. If the liquid brought up is acid (as the term pyrosis might seem to imply) the case is really one of eructation of gastric juice, as above described. But "waterbrash" often means the ejection of a much larger quantity of clear neutral or alkaline fluid, neither gastric juice, nor mucus, nor food. The late Dr Handfield Jones referred

* Etymologically, pyrosis ($\pi\tilde{\upsilon}\rho$ =fire) should mean the same as heartburn; but in England it is never used in this sense. In Greek, the word means inflammation.

pyrosis to catarrh of the gastric mucous membrane, and compared it to bronchorrhœa; but the liquid is often ejected without effort, and immediately after or even during a meal. Dr Chambers gives the case of a retired surgeon, who often had to leave the room at mealtimes, and would throw off as much as five or six ounces of frothy clear liquid. The contents of the gastric cavity never came up at the same time, although the ejection of the fluid sometimes made him retch. There seems, therefore, to be much probability in Chambers' suggestion that the fluid is really saliva, which trickles down the œsophagus, and, being arrested by spasm of the cardiac orifice, collects there until it gushes back into the mouth. Frerichs, indeed, detected sulpho-cyanide of potassium in the regurgitated liquid.

Sir Wm. Roberts carefully studied this curious condition, and analysed the liquid. He found it to possess diastatic powers, so that at least in his cases there can be no doubt that it was saliva, rapidly secreted, swallowed, and brought up again ('Lectures on Dietetics,' p. 81). He calls this disorder paroxysmal pyrosis with cramp of the stomach.

The preparations of bismuth are very useful in the treatment of pyrosis. Watson recommended opium combined with an astringent, as, for instance, in the pulvis kino compositus.

Vomiting.*—A frequent symptom of the more severe kinds of gastric disorder is *emesis* or *vomitus*. This occurs occasionally in severe cases of dyspepsia, and in almost all cases of organic disease of the stomach. It is also, as we have seen in previous chapters, an important symptom of cerebral disease, and we shall meet with it in the discussion of biliary and renal calculi, and of intestinal obstruction.

The act of vomiting is usually preceded by a peculiar feeling, termed *nausea* (*vauria* literally "ship-sickness"), accompanied by headache, giddiness, faintness, and coldness of the surface, pallor of the lips and face, and a small and feeble pulse. After a pause, salivation and then retching occurs, and is followed by the expulsion of the contents of the stomach. But vomiting is not always preceded by such painful sensations and efforts; for some women are subject for years to occasional sickness, attended with scarcely any discomfort, like that of infants. It is especially apt to occur in the early morning. So far it resembles the vomiting of alcoholic dyspepsia, and of pregnancy.

Sometimes such recurrent vomiting, without pain or other indication of gastric disorder, may at last become alarming, and even dangerous to life. At Guy's Hospital we have had several cases of this kind, and Sir William Gull used to describe them as the vomiting of women with "mad stomachs." Some striking instances were recorded by Dr Chambers. In one of them the affection had been of three years' duration, and it was stated that the food was always returned, unchanged in appearance, within ten minutes after being swallowed. Another patient was said for five years to have hardly ever kept down a whole meal. This kind of sickness is almost confined to the female sex, and occurs chiefly in young women. It is frequently associated with menstrual disorder; in one of the cases just quoted it was attributed to a chill, by which the catamenia had been suppressed for several months. These patients, too, are often hysterical; and sometimes reject the food before there has been time for it to be swallowed. Like other neuroses, this kind of vomiting is sometimes catching. But the most re-

* *Gr.* ἔμεσις.—*Lat.* Vomitus.—*Fr.* Vomissement.—*Germ.* Erbrechen.

markable feature of many of these cases is that, although the vomiting is so constant, yet there is little or no loss of flesh; so that some of the food must clearly be retained by the stomach. In this particular such cases differ from those described under the title *Anorexia nervosa*, but the distinction is only one of degree (cf. vol. i, p. 971).

The vomiting of sea-sickness is certainly cerebral in pathology, and unconnected with gastric catarrh. It begins by salivation (with an alkaline taste). Then the contents of the stomach are ejected (with an acid taste), and then follow those of the duodenum, producing bilious vomiting (with a bitter taste). Giddiness, headache, and fatigue of the ocular muscles accompany the sickness. It is increased by movement, mitigated or prevented by the recumbent attitude and by closing the eyes. It is rapidly cured by landing. It is very rare in young children. It is lost by the patient becoming accustomed to the motion of the ship. All these characters agree with a nervous, cerebral origin, and resemble those of concussion of the brain. The absence of diarrhoea or jaundice and the extreme rapidity of recovery are strong arguments against its being a primary disorder of the stomach, liver, or bowels. The fact that no discomfort or unpleasant food or smells or sights produce sea-sickness when the movement of the vessel is no longer felt; and the fact that movement in a pleasure-boat, in a train or a carriage, particularly with the back to the horses, and in a swing, will produce all the symptoms of sea-sickness, appear to confirm the above conclusion. We may even fix on the semicircular canals, with their branch of the seventh nerve and its cortical centre, as the apparatus primarily involved.

That vomiting is a symptom of organic disease of the brain must never be forgotten; in some cases of cerebral abscess there are few other symptoms than sickness. As Romberg long ago observed, cerebral vomiting is characterised by the absence of nausea and retching, and by its occurring when the head is moved, as in swinging, shaking, or stooping, or in suddenly rising; it also occurs when the patient is erect rather than when he is recumbent. Affections of the cord seldom cause gastric disturbance.

Vomiting, though not a frequent, is an important symptom of incipient phthisis. Dr Fagge used always most carefully to examine the lungs before admitting that habitual vomiting was merely due to functional disturbance of the stomach; and, if there were any other ground for thinking that tuberculous disease was likely to develop itself, would only give a guarded opinion, even when there was no discoverable stethoscopical sign of its presence.

Another malady, of which vomiting is a principal symptom, is Addison's disease of the supra-renal capsules.

In female patients the possibility of pregnancy must never be forgotten, particularly if the vomiting should be only of a few weeks' duration.

The above kinds of sickness are usually attended with constipation. When chronic vomiting and diarrhoea occur together, and intemperance can be excluded as a cause, the presence of Bright's disease should be suspected, and the pulse and heart as well as the urine should be carefully examined, for the quantity of albumen in the urine is often very small, and might easily be overlooked.

Another possible cause for the existence of vomiting and diarrhoea in the same patient is chronic poisoning by small doses of arsenic, antimony, or other irritant poison.

Vomiting as the result of violent coughing, no doubt owing to irradiation

from the respiratory centre, has been referred to under whooping-cough (vol. i, p. 215). That which is secondary to gastric ulcer or cancer of the stomach will be dealt with presently, and that due to intestinal obstruction in a subsequent chapter.

The *treatment* of vomiting depends mainly upon its cause. Sea-sickness can be relieved by the patient lying in his berth, or if he can do it, lying quietly on deck with the eyes closed; sleeping as much as possible, and taking a little tasteless food, such as dry biscuit, toast, or arrowroot from time to time. Brandy and water is usually given, but in most cases without benefit. A little champagne with a crust of stale bread is more often useful. Bromides, chloral, chloralamide, laudanum, and "chlorodyne" are sometimes beneficial by inducing sleep, but more often fail. A great point is to postpone, if not prevent, the act of vomiting as long as possible, by keeping the horizontal posture and taking only the kind and quantity of food which can be retained. Icebags to the head or back, mustard to the abdomen or the feet, and the many drugs recommended as cures, are each found wanting when tried on many patients. The constipation which follows sea-sickness sometimes needs help from drugs, and a blue pill and magnesium sulphate afford it most effectually.

When the diagnosis of primary neurotic "irritability of the stomach" has been reached, its treatment is often highly successful. In many cases the best plan is to give the stomach entire rest for one or two weeks, the patient being fed solely by enemata. Or minute quantities of milk may be administered by the mouth, as in the well-known case related by Dr William Hunter. A boy was brought to him in a state of the most extreme emaciation, who vomited almost everything that he swallowed, in spite of the treatment of three very eminent physicians. Dr Hunter recommended that only a single spoonful of milk should be given at a time; and the boy was never sick afterwards; he gradually became able to take more and more nourishment, and at last recovered entirely. A most graphic and interesting account of the case is given in the sixth volume of William Hunter's 'Medical Observations and Inquiries.'

In some neurotic cases "an enforced administration of more and better food is often the surest as well as the shortest road to deliverance from gastric hyperæsthesia,"* and in the most severe kinds of hysterical vomiting, the treatment introduced by Weir Mitchell of seclusion and forced feeding, will generally prove effectual when other means have failed.

The best treatment of the vomiting of early phthisis is to give soda or bismuth in chloroform water, with a compound rhubarb pill at night.

That of pregnancy is frequently relieved by oxalate of cerium. That due to uræmia will be considered in the chapter on Bright's disease.

When vomiting is due to gastric catarrh, the most valuable of all our drugs is bismuth in powder or suspended in mucilage. The application of a blister to the epigastrium is often highly serviceable. Sedatives may often be prescribed with advantage, but particularly morphia or opium. Occasionally the oxalate of cerium (in doses of two or five grains) succeeds when bismuth and hydrocyanic acid fails; but these two remedies are in most cases the ones we depend on. Hydrocyanic acid relieves pain as well as reflex vomiting; three or four drops may be given in a drachm of Aq. Chloroformi.

* Sir William Roberts, in his admirable "Address on Some Points in Dietetics" ('Brit. Med. Journ.,' Oct. 18th, 1890).

The writer has never seen much benefit from the administration of ice-pills, as they are called in Germany; but a drop of creasote on a small lump of sugar sometimes succeeds in checking obstinate vomiting. Of late years two, three, or four drops of tincture of iodine in a teaspoonful of water has become a favourite remedy in our wards, and it certainly appears sometimes to be efficacious.

In any case every effort should be made after the stomach has been emptied to check further retching, and it is never wise to persist in feeding while the stomach refuses to retain food.

Singultus or hiccough is due to a sudden spasmodic contraction of the diaphragm, repeated at more or less regular intervals, and attended with a clicking sound from the abrupt passage of air through the glottis. Its recurrence can often be stopped by holding the breath. Hiccough is not usually a matter of consequence, and lasts only a few minutes, or at most an hour or two. But it sometimes continues for days together, so as to exhaust the patient, and appear to be the immediate cause of death. Indeed, a persistent hiccough in any chronic disease has always been regarded as a bad omen. It is very difficult to cure.

Dr Edward Liveing had a man past middle age under his care in whom hiccough occurred in paroxysms of twelve hours' duration about twice a week for four years; and he quotes the case of a little girl of twelve, who for nearly three years was subject to fits of violent hiccough, even during sleep; they lasted from ten minutes to an hour, and returned three or four times during a day and night. She was cured after taking turpentine.

Frequently hiccough is due to the presence in the stomach of food which is incapable of being digested, and it is sometimes relieved by an emetic.

No one can make frequent autopsies without observing how often the stomach contains a pint or more of egg and brandy mixture, or beef-tea or half-digested milk, which must include all that had been given by the nurses for some hours before death. In one case of fever, as the end was approaching, the relatives flattered themselves that the patient would recover because he took all his nourishment; but it evidently was not absorbed, for a splashing sound could be constantly produced by manipulating the upper part of the abdomen. Such overzeal probably embarrasses the heart, and hastens or at least disturbs the hour of death.

Hæmatemesis, or vomiting of blood, is with rare exceptions either the effect of acute irritant poisoning, or of gastric ulcer or cancer; or of congestion of the stomach from primary disease of the heart or the liver.

When we are told that blood has been vomited, we must first make sure that it really came from the stomach. Strange as it may appear, there is often considerable difficulty in distinguishing between hæmoptysis and hæmatemesis, particularly when one has to rely upon the statements of the patient, and does not see the blood actually brought up. When blood escapes copiously into the air-passages, it may issue from the mouth in gushes; while some of it may pass back into the pharynx, and there excite retching and vomiting. Indeed, a portion of the blood may run down the œsophagus: as in an autopsy on a child who died of hæmoptysis, where Dr Fagge found an ounce of coagulated blood in the stomach. On the other hand, sudden and profuse hæmatemesis may provoke a paroxysm of cough.

The patient's statements, therefore, are an unsafe basis for distinguishing between vomiting and spitting of blood.

Diagnosis must be founded first upon the appearance of the blood brought up; and secondly, upon the history and other symptoms pointing to the lungs or to the stomach.

The blood of hæmoptysis is usually frothy from admixture of air; it has an alkaline reaction, and is of a bright red or "arterial" hue. That of hæmatemesis is acid and darker coloured, or brown, from being mixed with the gastric juice.

No doubt, when the hæmorrhage is very profuse—and particularly if a large artery has been opened—blood rejected from the stomach may be fluid and of a scarlet colour; but whenever it is retained for any length of time before being vomited it becomes dark, and it often coagulates, and sometimes solid clots are rejected which almost choke the patient. More frequently the blood, whether clotted or not, is acted on by the gastric juice, the acid of which decomposes the hæmoglobin and produces the brown and granular deposits of acid hæmatin. The presence of comparatively small quantities of blood in vomited matters thus gives them the appearance of coffee-grounds.

A great quantity of blood may ooze into the stomach before it excites vomiting; hence hæmatemesis is often preceded by the characteristic symptoms of hæmorrhage—pallor of face, dimness of vision, giddiness, or fainting. Hæmoptysis, on the other hand, occurs suddenly, the patient finding the hot blood in his mouth or feeling a tickling in his throat, or a sensation as of bubbling in the chest, immediately before he begins to cough up the blood.

After hæmatemesis, any blood that may be left in the stomach passes downwards, and is ultimately discharged in an altered condition from the bowels. But when any part of the air-passages is the seat of the hæmorrhage, the blood that is sucked into the smaller bronchial tubes is afterwards got rid of by expectoration, and the sputa remain discoloured for hours, or even for days.

Blood which passes from the stomach into the intestines is found in the stools black or, as it is called, "tarry." Evacuations having this character were supposed to consist of "black bile" by the older writers, who described them as characteristic of a special disease which they called *melæna*. But it has long been known that this condition is one of the results of hæmorrhage into the alimentary canal. Addison taught that when blood having this black colour was discharged from the bowels, the source of the bleeding was always the stomach. An exception to this rule must be made for the duodenum above the entry of the alkaline bile and pancreatic juice; but when the hæmorrhage occurs from the rest of the small or large intestine, the blood is always more or less distinctly red.

The dark appearance which the fæcal evacuations so constantly present when a patient has been taking a preparation of iron or bismuth medicinally must be distinguished from that due to blood. It is of a more slaty hue, and the fæces are as a rule remarkably dry. In a doubtful case one might apply the guaiacum test or that of the spectroscope.*

* The detection of iron in an acid solution of the dark pigment by yellow prussiate of potash would distinguish *melæna* from the effect of bismuth. On one occasion a hysterical or malingering schoolboy, whose case is referred to in the first volume (p. 975), said that he had passed blood from the bowels, and produced very dark motions. There was no evidence of disease, and it was found that the scybala were of natural colour, except on the

It is important to note that hæmorrhage into the stomach may, and often does, occur without any blood being vomited. Thus a case was observed at Guy's Hospital in which a patient (who had once before brought up a large quantity of blood) became blanched, called out that she was dying, and expired in twenty-five minutes, after a convulsive seizure. The stomach was full of clotted blood. Other instances of a similar kind have occurred at the hospital, and one such is mentioned by Watson. Doubtless, therefore, it is a frequent occurrence for small quantities of blood to escape into the gastric cavity without exciting vomiting. Up to a certain point, it is probable that blood undergoes digestion and is absorbed before it is passed down the whole length of the intestine; so that the stools may fail to afford evidence of the hæmorrhage. Still, whenever a patient's symptoms can be attributed to bleeding into the stomach, the fæces should be most carefully examined.

A case in point is given by Chambers. A woman, aged thirty-three, who had suffered from well-marked symptoms of gastric disease, grew rapidly weaker and paler, and her tongue became dry and furred. It was long suspected that she was passing blood, but she constantly denied it. She was made an in-patient, and melæna was then discovered.

On the other hand, blood may be vomited without any hæmorrhage from the vessels of the stomach. Such a mistake may occur in good faith when blood escapes into the back of the mouth or pharynx from epistaxis, and is swallowed by the patient unconsciously, particularly during sleep; the first indication of the fact may be profuse hæmatemesis. But hæmatemesis has sometimes been (so to speak) manufactured, the patients having first secretly drunk the blood which they afterwards vomited in the presence of others. Watson mentions two cases of this kind.

In most cases of hæmatemesis, however, the blood really comes from the blood-vessels of the stomach; and we have now to consider its causes.

Ætiology.—In the first place gastric hæmorrhage may occur in certain general diseases, as a consequence (it is supposed) of changes in the blood itself: for example, scurvy, purpura, malignant smallpox, yellow fever, acute yellow atrophy of the liver, and the grave forms of anæmia—leuchæmia, Hodgkin's disease, and Addison's pernicious anæmia. Again, it may be a symptom of organic disease of the heart, or of the high arterial tension produced by chronic Bright's disease, when the arteries are often morbidly brittle.

Hæmatemesis is a symptom of the two principal organic diseases of the stomach—simple ulcer and cancer. In cases of cancer, however, it is rare for any considerable quantity of blood to be vomited until the disease has reached an advanced stage, or has, at any rate, declared itself by well-marked symptoms. On the other hand, a simple ulcer of the stomach is sometimes latent up to the time when a large hæmorrhage takes place; the patient having either had no symptoms previously, or only slight indications of gastric disorder. Sudden large hæmatemesis from perforation of an artery by a gastric ulcer is not infrequent in young subjects, and is very seldom immediately fatal. In some cases it returns at intervals of a few hours, and the patient succumbs within a week; but more often no fresh bleeding occurs for several weeks or months.

surface. The stools gave no iron reaction, and the colour was found to be due to a silver solution which had been poured into the nightstool from a bottle used in photography.

* It is said that hæmatemesis may be caused by disease of the spleen. But in such cases the splenic enlargement and the hæmatemesis are probably joint results of portal congestion due to hepatic obstruction.

The writer once saw a young man, believed to be the subject of gastric ulcer, who had vomited a large wash-hand-basin full of blood, carefully observed by his medical attendant. He was blanched and faint, with a very weak pulse, and was no doubt in great danger; but he completely recovered from this severe hæmorrhage.

After death it is sometimes impossible to discover the origin of the fatal hæmorrhage. The late Dr Murchison placed on record two cases, in each of which the source of bleeding was a mere pore-like aperture leading directly into a large branch of artery. A preparation of a similar kind is contained in Guy's Hospital Museum (No. 653). It is worthy of note that in these three instances the seat of the ulcer was not the lesser curvature, but the cardiac pouch of the stomach. A small ulcer of this kind might easily be overlooked, and it is possible that such may have been the explanation of some of those cases in which a *post-mortem* examination has failed to reveal the source of the hæmorrhage. In other cases the source of hæmorrhage is a varicose vein in the cardiac region (Museum, No. 606).

Some time ago a carman, aged thirty-two, who was in the hospital for gout and albuminuria, died suddenly from hæmatemesis. The stomach was full of blood, but we could not discover from what part of the stomach it had come. The vessels of the stomach were not found much congested, for the hæmorrhage had doubtless emptied them; and the liver appeared to be quite healthy.—C. H. F.

Vomiting of blood is very frequent in cases of cirrhosis of the liver, and it is often the earliest symptom; but many cases in which ascites has already appeared terminate by sudden hæmatemesis. Other forms of chronic hepatic disease causing portal congestion may have the same effect: adhesive pylephlebitis, perihepatitis, and syphilis; but all these causes are comparatively rare.

Hæmatemesis from rupture of an aneurysm into the stomach is also an infrequent event.

The older writers mention hæmatemesis due to suppression of the menses.* Such vicarious hæmatemesis is, to say the least, exceedingly rare; and probably many supposed cases would not stand modern criticism. We do not even see that in cases of suppressed menses a patient with gastric ulcer is more liable to free hæmorrhage every month.

Another cause for hæmatemesis, independent of visceral disease, is atheroma of the blood-vessels. In one case under the writer's observation it followed profuse epistaxis, and was itself the precursor of cerebral hæmorrhage.

Notwithstanding the many possible occasional causes of hæmatemesis, its occurrence in a person who presents no other well-marked symptoms of disease is, in by far the most cases, the result either of a latent ulcer, or of congestion of the stomach from early cirrhosis of the liver. Can these two conditions be certainly distinguished from one another? Both Dr Murchison's cases, above mentioned, occurred in persons who had been intemperate; one of them was a plethoric woman, aged fifty, the other a soldier aged twenty-eight, whose liver was cirrhotic. It might well have been

* Sir Thomas Watson relates a case of this kind which came within the knowledge of the late Dr Peter Latham. A girl about the age of fourteen became the subject of hæmatemesis, recurring at monthly periods. She married without ever having menstruated, and became pregnant; the hæmatemesis then ceased, and did not return until she had been confined, and had suckled and weaned her infant. Watson also quotes Mr North as having met with two instances in which suppressed menstruation was followed by repeated and at length fatal hæmorrhage. I have searched in vain for the original record of these two cases, so that I cannot tell whether the presence of a gastric ulcer was disproved by an autopsy.—C. H. F.

thought that in both instances the hæmorrhage was due to portal obstruction.

There remain certain unexplained cases of large and repeated hæmatemesis occurring in persons otherwise healthy. The writer has seen more than one case of the kind, in young and temperate men, without the pain and vomiting which usually accompany gastric ulcers.

Treatment.—Small gastric hæmorrhage should not be interfered with. It does no harm, is a valuable symptom for the physician, and often a useful admonition for the patient. Moreover we are not likely to succeed if we try to stop it.

In cases of free hæmorrhage from portal obstruction, Sir Thomas Watson advised five grains of calomel to be given every night and a black draught every morning, till the stools lose their pitchy colour; and he pursued this plan even when the patient had been blanched by previous hæmorrhages, and when the pulse was feeble and irregular. But if the case is one of gastric congestion, the occurrence of hæmorrhage shows that the vessels are on the way to relieve themselves, even if they have not already done so. If the blood comes from ulceration into an artery, the only rational treatment is to keep the circulation and the abdominal viscera as quiet as possible. The best course, therefore, is to wait for a day or two, until we see whether the hæmorrhage returns. Even if the patient should pass several tarry evacuations in succession, this is not a proof that bleeding has occurred more than once, for they may all have arisen from a single hæmorrhage.

When repeated attacks of hæmorrhage occur in a person already blanched by loss of blood, they can scarcely be caused by portal congestion, and acetate of lead should be prescribed in doses of three or four grains, with a quarter of a grain of opium, every two or three hours. Some writers have spoken highly of oil of turpentine, twenty minims every four or six hours. Other valuable styptics are gallic acid and hazeline.*

The patient may suck small pieces of ice, but he should be allowed to take nothing else into his stomach. Starvation is indeed the cardinal point in the treatment of hæmatemesis, nutrient enemata and suppositories being given if support appears necessary. If the hæmorrhage is profuse the patient's head should be kept low, and a bladder of ice applied to the epigastrium.

GASTRIC ULCER.†—We have seen that vomiting, gastric pain, and hæmatemesis may each or all be symptoms of ulcer of the stomach.

Minute hæmorrhagic erosions are often seen in cases of advanced cardiac disease, and we occasionally meet with a large number of small chronic ulcers.

Anatomy.—The lesion now to be described differs altogether from small, shallow, secondary, and always multiple ulcers. It affects only a small part of the surface of the stomach. Most frequently there is only a single ulcer; sometimes there are two, and very rarely more. Rokitansky found the ulcer solitary in sixty-two out of seventy-nine cases, and in twelve of the

* It is doubtful whether sulphuric acid can act as an astringent in cases of diarrhœa, of hæmoptysis, or of profuse sweating; for in medicinal doses it must always be neutralised by the bile and pancreatic juice, or, if absorbed from the stomach, by the soda of the blood. It may be of service when applied directly to the mucous membrane of the stomach; even in cases of hæmatemesis, however, it is rarely prescribed without opium, catechu, or some other astringent, so that it is difficult to judge of its efficacy.

† *Synonyms.*—Ulcus ventriculi—Abercrombie's ulcer—Round ulcer—Simple chronic ulcer of Cruveilhier—Perforating ulcer of Rokitansky—Ulcus pepticum.

rest there were two. When there are more than one, they differ in size and in other characters, which show that they began at different times.

It is remarkable that the seat of a gastric ulcer is, in the great majority of cases, along the lesser curvature of the stomach. Sometimes an ulcer lies across the curvature itself; more often it is situated either in the anterior or posterior wall, but almost always close to that line.* Not infrequently two ulcers are found in the same stomach exactly opposite one another, one on each surface; and since they generally appear to be of different dates, it has been supposed that one of them has been set up as the result of its coming into contact with the other. Occasionally the pylorus is the seat of the ulcer, and exceptionally the greater curvature or cardiac pouch.

An exactly similar ulcer may be found in the first portion of the duodenum (*vide* p. 353).

The acute perforating ulcer is seldom larger than a sixpenny piece. It has a sharply-defined edge, at first entirely free from thickening, and the form of a flattened cone, the base corresponding with the mucous surface of the stomach. It is often described as "punched-out," on account of its regular circular form and the evenness of its margin. Its floor may consist of the muscular coat, or the ulceration may extend through this, and form a narrowing pit, at the bottom of which the peritoneum is visible. Frequently the serous coat in its turn becomes attacked; a minute yellow slough forms; and the detachment of this allows the contents of the stomach to escape into the lesser bag of the peritoneum or into the general abdominal cavity. Not infrequently, instead of eating its way through the coats of the stomach, it erodes an artery, and thus gives rise to hæmatemesis.

A round, single, deep, perforating ulcer like this is very seldom found anywhere but in the stomach. Sir Wm. Flower once recorded an instance in which a small round ulcer developed itself in the œsophagus, and passed straight through into the descending aorta. In the duodenum similar ulcers are extremely rare except in the first two inches, and only isolated cases have been published of similar ulcers in the jejunum, the ileum, and the descending colon.

If a gastric ulcer becomes chronic, it acquires further characters which are still more peculiar to it. Its edge, although still perfectly even and regular, becomes thickened; and, for a little distance beyond, the gastric tunics are matted together. These changes evidently depend on a chronic inflammatory process; and the peritoneum covering the floor of the ulcer also becomes thickened, opaque, and adherent to the neighbouring tissues.

From the locality affected by the chronic gastric ulcer, its floor almost always becomes attached either to the under surface of the left lobe of the liver, if it be in the anterior wall of the stomach, or to the pancreas and the adjacent connective tissue and vessels, if it be on the posterior wall. Thus perforation of the serous cavity is for the time prevented, while the ulcer gradually increases in size. Its growth in different directions is not always uniform, and thus it often loses its circular shape, and becomes oval or irregular in form. This last result is frequently due rather to the fact that when two ulcers are present they often come into contact as they grow larger, and finally coalesce. As before mentioned, two are often found just

* According to Brinton, ulcers are found on the posterior surface of the stomach eight times as often as on its anterior surface. But our *post-mortem* records at Guy's Hospital by no means bear out this statement.—C. H. F.

opposite to one another, one on each side of the lesser curvature ; and these, when they run together, give rise to a single ulcer of dumbbell shape. The size to which an ulcer of the stomach attains is sometimes very considerable ; the 'Pathological Transactions' contain a record of one which measured five and a half by three inches. While thus spreading in circumference, gastric ulcers also increase in depth. The peritoneum is gradually worn through where it is adherent, and the surface of the pancreas or of the liver comes to form part of the wall of the stomach. This at first takes place only at a small spot, but the area of adhesion and that of destruction gradually becomes more extensive. Thus a great part of the pancreas may in time become exposed in the floor of the ulcer, covered only by a thin film of connective tissue, through which its lobulated character can be plainly recognised.

Occasionally a gastric ulcer will form adhesions with the transverse colon and perforate, so as to form a gastro-colic fistula.

In comparatively rare cases the floor of an ulcer in the anterior wall of the stomach becomes adherent, not to the liver, but to the abdominal walls, and at last perforates them, so that a gastro-cutaneous fistula is formed. Murchison collected twenty-five cases of this kind ('Med.-Chir. Trans.,' vol. xli), of which, however, only twelve were instances of gastric ulcer ; six of them were cases of cancer, and in seven the penetration of the walls of the stomach was due to wounds or injuries of the corresponding part of the surface of the abdomen.* A gastro-cutaneous fistula may remain open for several years ; but it sometimes closes of its own accord.

In the only case which has come under the writer's care, in a lady aged fifty-five, the nature of the epigastric ulcer was proved by its discharging a clear liquid of strong acid reaction. Although firm adhesions had taken place, death ensued as the result of profuse hæmatemesis, probably from a second ulcer. It was remarkable that two of this patient's children subsequently died of gastric ulcer at about the age of twenty.

This process of adhesion of the floor of the ulcer to different parts is by no means unattended with risk. In the first place the protective process of adhesion may at any time fail to keep pace with the spread of the ulceration, and perforation into the peritoneal cavity may take place ; or the adhesions may be broken through by some muscular effort made by the patient, when the same result follows. Perforation is more apt to occur when the ulcer is in the anterior than in the posterior wall of the stomach ; indeed, in the former position ulcers seldom attain any considerable size.

In other cases danger arises from the penetration of blood-vessels. We have seen that the recent "punched-out" ulcer often erodes an artery of some size ; but in the chronic cases now described it is no uncommon thing to see a large artery, or even more than one, with its coats abruptly cut across, lying in the floor of the ulcer, and plugged with a little cylinder of clot that can be pushed out with little difficulty. In other cases, in which death has been directly due to hæmorrhage, the vessel is patent. The artery is sometimes a branch of the coronary artery of the stomach, or the trunk of that vessel, or a pancreatic branch of the splenic artery. Even the trunk of the splenic artery itself is sometimes penetrated by a gastric ulcer.

* Of these last, the most remarkable of all is perhaps one recorded by Murchison himself of a woman who for three years kept a penny pressed into the sore left by a seton until an opening into the stomach was formed.

Happily, another change to which a gastric ulcer is liable is cicatrization. This occurs not infrequently. Indeed, one seldom sees a large ulcer which has not healed over in some parts of its surface. Brinton speaks of cases in which the whole extent of the ulcer has been found cicatrised, with the single exception of a point in the centre occupied by an eroded artery, hæmorrhage from which had caused death. But in most instances, when a gastric ulcer heals, the patient has good health afterwards. Should he die from some other disease, the cicatrix varies in appearance according as the coats of the stomach were more or less deeply and widely destroyed; it may show merely a little thickening of the submucous tissue, or it may form a hard, puckered mass, with radiating processes.

When an ulcer is seated at the pylorus, its cicatrization may cause narrowing and obstruction to the passage of food. The result is that the stomach becomes dilated and hypertrophied, exactly as when the bladder dilates behind a stricture.

Again, the cicatrix of a large ulcer occupying the middle of the stomach may constrict it, and so cause what is termed an hour-glass contraction.

Pathology and origin.—The credit of having originally described this disease is commonly assigned to Cruveilhier, who published his account of it in 1830; or to Rokitansky, whose work appeared in 1839. But Abercrombie had previously pointed out all its distinctive characters in 1828, when he not only described its anatomy and symptoms with cases in proof of his statements, but clearly showed its course, and the events of hæmorrhage and of perforation ('Diseases of Stomach,' 3rd ed., pp. 18-40). No doubt the three accounts were each based on independent observations.

Rokitansky suggested that a gastric ulcer begins as a hæmorrhagic erosion, but one objection to this view is that hæmorrhagic erosions are multiple, and frequently met with in cases of portal obstruction, and yet nothing like the single round ulcer with its special locality is met with in long-continued congestion of the gastric mucous membrane, either from hepatic cirrhosis, or from cardiac disease. Virchow, in 1853, adopted this hypothesis, and attributed the destruction of the coats of the stomach to the corrosive action of the gastric juice. This, he said, cannot dissolve the mucous membrane so long as the circulation is maintained, for the alkaline blood will neutralise the acid as it penetrates the tissues. He therefore supposed that the starting-point of the affection was some morbid change in the blood-vessels of that part of the stomach, whether obliteration of an artery or obstruction of a vein. He also traced the conical form of the ulcer to the distribution of the tuft of vessels arising from a single arterial rootlet. A further argument he found in the fact that when perforation occurs, the aperture in the serous coat is always to be found away from the centre of the ulcer; this he attributed to the fact that the apex of the vascular cone is likewise excentric, being always directed towards whichever is the nearer of the two curvatures of the stomach, along which the main arterial trunks run. Panum brought experimental support to Virchow's hypothesis by injecting globules of wax into the branches of the abdominal aorta in dogs. He found that when they made their way into the arteries of the stomach the mucous membrane presented ulcers which resembled an early stage of idiopathic gastric ulcer. Dr Pavy and others since have found that ligature of a vein tended to the formation of a thrombosis and a local slough.

In its early stage, gastric ulcer is, as Virchow pointed out, like ulcers

which may occur in other parts of the alimentary canal. But a chronic, well-developed ulcer of the stomach presents characters seen nowhere else, with the single exception of the first part of the duodenum—which also is exposed to the gastric juice. The frequent contact of an acid secretion with the surface of an ulcer may not only retard its cicatrisation, but also set up chronic inflammation in its edge and floor, and thus give it special characters. A further argument in favour of this view is the fact that truncated blood-vessels are often exposed in the floor of a gastric ulcer, whereas in all other parts of the body the walls of arteries show a remarkable power of resisting ulceration.

It seems, therefore, probable that the digestive action of the gastric juice is really the cause of the distinctive characters of the completed round ulcer of the stomach, which may therefore be rightly called “peptic;” but its origin is still doubtful.

So far, although bacterial necrosis has been often observed, its relation to the ulcer remains uncertain. Dr Martin compares gastric ulcer to the perforating duodenal ulcer of guinea-pigs, and ascribes the comparative rarity of ulcers in the cardiac region to the abundant secretion of gastric juice in that part of the stomach.

The fact that the great majority of ulcers occupy the lesser curvature of the stomach, or its close neighbourhood, may, as Gull suggested, be in some way dependent on the fact that this part is more fixed than the rest of the organ.

Sex.—All writers are agreed that the symptoms of gastric ulcer occur much more often in females than in males; according to Brinton and Wilson Fox, two or even three times as often.

Of 100 cases which recovered (*viz.* 93 collected from our clinical records, and 7 others added from notes of private cases to make up the 100), there were 67 in women and 33 in men.

Of 171 cases lately observed in hospital practice by Dr Sidney Martin, 144 occurred in women and only 27 in men.

In von Ziemssen’s *post-mortem* cases the proportion was 35 to 15; but in 101 fatal cases extracted from the *post-mortem* records of Guy’s Hospital for the second edition of this work (1888), there were 59 in men and only 42 in women.

Age.—It is commonly believed that gastric ulcer is a disease of young women, and rare in either sex before puberty or after forty. This, however, was not the opinion of the late Dr Brinton, who maintained that it occurred at all ages, from sixteen to sixty and upwards. The following table, drawn up from the clinical and the *post-mortem* records of Guy’s Hospital in 1888, may be of service in determining this point. Ninety-three of the cases are those of patients who left the hospital more or less benefited; the other 101 are those of patients who died, and in whom the diagnosis of gastric ulcer was verified by an autopsy.

<i>Clinical Cases.</i>							<i>Fatal Cases.</i>						
Age.		Male.		Female.		Totals.		Male.		Female.		Totals.	
Under 9	.	0	+	0	=	0	.	2	+	2	=	4	
10 to 19	.	2	+	9	=	11	.	3	+	4	=	7	
23 „ 29	.	6	+	24	=	30	.	7	+	5	=	12	
30 „ 39	.	9	+	14	=	23	.	7	+	8	=	15	
40 „ 49	.	12	+	10	=	22	.	15	+	7	=	22	
50 „ 59	.	4	+	3	=	7	.	15	+	15	=	30	
60 „ 66	.				=	0	.	9	+	0	=	9	
70 and 72	.				=	0	.	1	+	1	=	2	
		—		—		—		—		—		—	
		33	+	60	=	93	.	59	+	42	=	101	

The striking difference in the two sets of cases seems to show that the prevalent belief that gastric ulcer is a disease of young women must have influenced the diagnosis during life. Of the writer's own cases the slight majority have been in men. He once saw it in a patient of seventy-eight, who recovered after suffering from hæmatemesis and other symptoms referable only to gastric ulcer, and lived for eight years afterwards.

Nevertheless the statistics published by Dr ~~Walsh~~, show that in Baltimore the predominance of female over male cases holds anatomically as well as clinically. Probably women are more able to lie up and submit to necessary treatment than men, and therefore have a less frequently fatal result. See Dr Saundby's remarks ('Brit. Med. Journ.,' 1900, vol. ii).

In Dr Martin's 171 cases, 70 out of 144 in women occurred between twenty and thirty, and 102 between twenty and forty; whereas in men the most cases (10 out of 27) occurred between forty and fifty. A similar difference in distribution as to age between the two sexes appears in the table above given.

There is one class of cases remarkably frequent in girls at or soon after the age of puberty, namely, those cases in which the ulcer remains latent until fatal perforation occurs. Dr Buzzard has recorded an instance of this kind in a girl nine years old. But it would be a mistake to suppose that the risk of perforation is limited to such cases, and ulcers—recognised by the characteristic symptoms of the disease—destroy the patient's life in this way at all ages and in both sexes.

It is impossible not to question whether the different events of gastric ulceration do not point to an essential pathological difference. The contrast is a striking one between the acute, hæmorrhagic, single ulcer perforating rapidly and apparently with little check from treatment, and the chronic, shallow, extensive and often multiple ulceration, which lasts for months and years with much distress but little danger, marked by aching pain and sickness, but by only slight and occasional hæmorrhage; which apparently heals, and then relapses, and seldom or never kills.

Nevertheless it appears probable that these are only extreme examples which are connected by a continuous chain of cases marked with more or less severity. Until we know more of the true origin of gastric ulcers, it seems best not to attempt to distinguish more curiously than does nature.

What is also worthy of note is the exact likeness of duodenal to gastric ulcer, and the absence of any similar lesion in the rest of the alimentary canal, with almost complete absence of enteric, tuberculous, and dysenteric ulceration in the stomach.

Symptoms.—The most significant, and generally the earliest symptom of gastric ulcer is *pain*. This may be of every degree of intensity, from a mere feeling of weight or tightness in the epigastrium, up to the most severe sensations of burning or gnawing or boring, attended with sickening depression. According to Brinton, it is rarely or never described as stabbing or cutting. In the great majority of cases it comes on from two to ten minutes after taking food, continues an hour or two, and subsides when digestion is accomplished. If vomiting occurs, this almost always brings relief. Sometimes the pain does not begin until half an hour after a meal. It is increased by indigestible or hot food, and particularly by tea. In very rare cases it comes on when the stomach is empty, and is relieved by food or hot water.

The pain of gastric ulcer is not always intermittent. It may be con-

tinuous, lasting for days or weeks together. These are generally cases in which the ulcer is of long standing.

The seat of the pain is most frequently the epigastrium, a little below the ensiform cartilage; but sometimes it is felt behind the cartilage, and sometimes two or three inches lower down. Occasionally it is outside the median line, in one or other hypochondriac region; but is still limited to a small area, rarely more than two inches in diameter, and sometimes less than half that size.

Scarcely less important than the pain already described is a pain in the back, to which Cruveilhier first drew attention as a symptom of gastric ulcer. This is of a gnawing character, and is generally referred to a single spot between the eighth or ninth dorsal vertebra and the first or second lumbar.

Brinton believed that variations in the seat of the pain in different cases could be traced to differences in the position of the ulcer. He collected some twenty-five cases in which, the pain having been referred to one or other hypochondrium, the ulcer was afterwards found to occupy the corresponding part of the stomach. He also ascertained that in some cases the position which the patient chose to obtain the greatest ease from pain was a guide to the seat of the ulcer, the prone position indicating that this was on the posterior, and the supine that it was on the anterior wall of the stomach; whereas when the patient found relief by lying on the right side, the ulcer was at the cardiac end, and when on the left side it was at the pylorus. In most cases, whatever the seat of the ulcer, the recumbent posture gave ease.

Pressure on the spot to which the pain is referred almost always aggravates it, and in many cases there is extreme tenderness, so that contact with the clothes, or the gentlest touch of the physician's hand, cannot be endured. Sometimes pressure on the epigastrium increases the pain in the back; but a very few instances have been recorded in which pressure has given relief to the pain.

The second symptom of gastric ulcer is *vomiting*. In the most marked cases this does not occur for some weeks after the patient has begun to suffer. It takes place when the paroxysm of pain induced by food has reached a certain height; there is seldom violent retching, and as soon as the stomach is emptied the patient is free from discomfort. It is only in rare cases that sickness occurs independently of food.

The third symptom is *hæmatemesis*—it was present in four fifths of Lebert's 252 cases observed at Breslau, and in 144 of Dr Martin's 171. Whenever the presence of gastric ulcer is suspected, the vomit should be carefully and repeatedly examined for the presence of blood.

Specimens of vomit selected for examination should contain as little as possible of food. If this precaution be taken, a liquid which had seemed clear will often throw down a sediment containing blood-corpuscles.

In other cases the amount of blood effused into the stomach is larger. Being altered by the gastric juice, it gives to the vomited matters a brown colour, like "coffee-grounds," due to the presence of acid hæmatin.*

It must not be supposed that traces of blood in the vomited matters, or even the occurrence of obvious hæmatemesis, is a proof that an artery

* The administration of a preparation of iron may give a blackish colour to the contents of the stomach, if the patient should about the same time take tea (Brinton).

has been exposed in the floor of the ulcer; the blood often comes from the minute vessels which supply its surface.

The appearance of altered blood in the stools in the form of melæna is no less important a diagnostic sign than hæmatemesis itself (cf. p. 338).

Other symptoms of gastric ulcer are less characteristic:—as flatulence, pyrosis, and constipation. The appetite is generally defective, but in some cases excessive and ravenous, particularly when there is habitual vomiting of all that is taken; but although there is great desire for food, the patient is afraid to eat because of the pain which follows every meal.

In young women amenorrhœa is frequent. Brinton observed that when pain is worse with each period (a frequent occurrence), this usually occurs before rather than during the flow. In older women menstruation often goes on regularly, although they may suffer from gastric ulcer for several years; but if there should be anæmia from profuse hæmatemesis the catamenia may be suppressed for a time.

In long-standing cases there is often extreme wasting; lines are worn in the patient's face by the constant pain and the deprivation of food; and so peculiar is the physiognomy, that Brinton was often able to recognise the disease at a glance in a crowded out-patient room.

When a gastro-cutaneous fistula has developed itself the patient usually ceases to vomit. Food is apt to escape from the orifice, so that the patient has to wear a plug of lint or gutta-percha unless the mucous membrane protrudes, and forms a kind of natural valve. The general health, however, often improves.

Diagnosis.—The characteristic symptoms of gastric ulcer, then, are the occurrence, soon after the ingestion of food, of a pain peculiar in kind and seat, which is relieved by vomiting, or subsides when digestion is completed, and the presence of blood in vomited matters or as melæna in the fæces.

When these symptoms are present in a person under forty years of age, the recognition of gastric ulcer is not difficult; but often they are absent or obscured, and in older persons may sometimes be due to cancerous ulcer of the stomach, or even to cirrhosis of the liver. In young women the symptoms of gastric ulcer are often simulated by anæmia from other causes, and by phthisis.

The gastric juice obtained from the stomach in cases of simple ulcer is stated by Ewald, Reigel, and other observers to be constantly, or almost constantly, more acid than in health. The chemical diagnosis which between ulcer and cancer of the stomach rests on this basis will be mentioned under the latter head (p. 363).

Gastric ulcer has been mistaken for chronic poisoning. The symptoms of the fatal illness of the Duchess of Orleans in 1670 were commonly attributed to foul play, until Littré in a masterly clinical and historical commentary on the case showed conclusively that she died from perforation of a gastric ulcer.

Gastric ulcer is not infrequently altogether *latent* until it erodes a large artery or perforates into the cavity of the peritoneum. When a patient in apparent health is suddenly attacked with an illness which proves fatal in a few hours, an ulcer may be found which presents no sign of inflammatory reaction in its walls; and there appears no reason why all the coats of the stomach should not be destroyed in a few days. But often when perforation occurs in persons apparently in good health, the ulcer is found with smooth rounded edges, and must have existed for some time. Moreover it not in-

frequently happens that an ulcer is found accidentally in the stomach of a person who has had no gastric symptoms, and has died of some other disease.

More often ulcer of the stomach is overlooked, because the symptoms of gastric disorder have been so slight that the case has been regarded as one of mere dyspepsia. The absence of vomiting is particularly apt to lead to this mistake. Brinton mentions one case in which there was no vomiting for four years during which an ulcer remained active; and he speaks of other cases in which it was represented by slight nausea only, or was limited to a single attack, or occurred only at the very close of the disease. No absolute rule can be laid down for the diagnosis of gastric ulcer in cases of this kind.

Events.—A gastric ulcer may have several different terminations. Cicatrisation sometimes occurs, and the patient regains his former state of health. Several years ago the state of the stomach was noted with great care in a large number of autopsies at Prague, and both cicatrices and unhealed ulcers were found very frequently, in the proportion of 147 of the former to 156 of the latter. Scars in the stomach have sometimes been discovered in the *post-mortem* theatre at Guy's Hospital, but far more rarely than at Prague.

Few would endorse Cruveilhier's statement that "simple ulcer of the stomach tends essentially to a cure;" but many cases are on record of symptoms followed by spontaneous cicatrisation. One is that of the anatomist Bécларd, who suffered from pain in the stomach and vomiting, from which he gradually recovered; when he died, many years afterwards, the scar of an ulcer was found in the lesser curvature of the stomach.

The healing of a gastric ulcer is not infrequently only partial. Cicatrisation may take place on one side, while in the opposite direction it goes on spreading. The stomach may acquire an "hour-glass contraction," or the pylorus may become narrowed, so that symptoms of obstruction may arise, in addition to those of the ulcer; or the sore may heal for a time and afterwards again break out. Probably this is one cause of the remarkable fact that there may be a complete intermission of all the symptoms of gastric ulcer for many months, after which they return. In other cases, however, an apparent intermission is really due to the fact that, after the first ulcer has finally healed, a fresh one develops. Lastly, it appears probable that the inclusion of nervous filaments in a cicatrix is sometimes the cause of the continuance of pain after the subsidence of the other symptoms, indeed after the cure of the disease.

The *duration* of this disease is often exceedingly protracted. Cases have been recorded in which symptoms were present uninterruptedly for twenty or thirty years.

The proportion of cases of gastric ulcer which recover under suitable treatment is certainly large; but in 100 cases in our wards fourteen deaths occurred while in the hospital.

There are several different ways in which gastric ulcer may prove fatal. Sometimes the patient dies by gradual exhaustion. This, however, seems to be rare; it is recorded only three times at Guy's Hospital out of thirty-four cases in which the immediate cause of death is noted. Still, Brinton saw three or four cases of this kind within a few months.

In eleven of our thirty-four cases death was traceable directly to hæmorrhage, and in seventeen to perforation. Other results of gastric ulcer are

much more rare. In some cases the destruction of the gastric coats is followed, not by general peritonitis, but by a circumscribed abscess, which generally occupies the left hypochondrium, and less frequently the right. This subphrenic abscess may in its turn perforate the diaphragm and set up fatal pleurisy. In other cases, after perforation, an abscess is formed in the posterior mediastinum. Still more rarely, the ulcer sets up pylephlebitis and abscesses in the liver.

It is an interesting question whether cancer ever develops itself secondarily in the floor or edge of a simple ulcer of the stomach. Trousseau speaks of the two diseases as antagonistic, but Brinton is disposed to think that the one may pass into the other. We have had at Guy's Hospital more than one case in which the stomach presented part of what was apparently a simple ulcer, of which the remainder had been replaced by a malignant growth. If this view is correct, the occurrence is probably more frequent than might appear from the absence of direct observations of it; for in many cases the extensive development of cancer would doubtless obliterate all traces of the previous ulcer.

Dr L. E. Shaw reports that, of fifty autopsies, in six there was found a cicatrix surrounded by or near the cancer which was believed to be of older date than the latter, but on comparing the clinical histories of these cases no confirmation of the previous existence of a simple ulcer could be found.

Treatment.—In cases of ulcer of the stomach our first object is to relieve the pain, vomiting, and hæmatemesis; our second is to promote the healing of the ulcer. Bismuth, or the kino powder with opium, is often effectual in arresting hæmorrhage from the stomach. Brinton expressed a strong opinion against the use of the oxide or nitrate of silver in this disease; but although it is no doubt impossible that these medicines can act upon the surface of the ulcer as lunar caustic does upon a sore to which it is directly applied, it is certain that they sometimes give relief to the pain. Opium, beyond its influence in relieving pain and sickness, seems to have a favourable influence on the ulcer, as it certainly has in cases of chronic ulcer of the leg.

The use of purgatives requires much caution in cases of ulcer of the stomach, and there is no doubt that they should as far as possible be avoided. But Brinton more than once noticed a definite and repeated coincidence between the occurrence of a paroxysm of pain and vomiting and an accumulation of fæces in the colon; and for such cases he recommends the use of castor-oil. Of the fact there is no doubt, but probably enemata are a safer treatment.

A blister often relieves the symptoms of gastric ulcer, and one applied to the back has been found to relieve the pain in that region. Brinton disapproved of the use of leeches, but Wilson Fox believed that the application of two or three sometimes gives marked relief.

If the cicatrisation of gastric ulcers is prevented by the action of the gastric juice, the rational treatment is evidently to keep the stomach empty for a time, supporting the patient by enemata or nutrient suppositories. In cases of obstruction of the œsophagus life may be maintained in this way for at least two or three weeks, a period which is probably long enough to enable a gastric ulcer to take on a healing action, even if it is not sufficient for its complete cicatrisation. Whenever the diagnosis is clear, and the patient can be induced to submit to this method of treatment, it would be reasonable to carry it out for a certain period at once. However slight

the symptoms may be, one never can tell how near the peritoneum or some large artery may be to the floor of the ulcer; and every week's delay will add something to its size.

Moreover, in other cases, the very urgency of the symptoms affords an argument for giving complete rest to the stomach, and feeding the patient by nutritive injections. Vomiting often resists the action of every reputed remedy. It is also a most dangerous symptom of gastric ulcer, on account of the risk of perforation from rupture of the protective adhesions. Certainly, therefore, no food should be swallowed while obstinate vomiting continues.

In the well-known case of Dr William Hunter, before quoted (p. 336), vomiting that had been uncontrollable was checked by the limitation of the food to milk, given a spoonful at a time. A similar plan was suggested by Cruveilhier, for cases of gastric ulcer. Sometimes the milk is better borne when it has previously been boiled, or when it is mixed with lime-water or peptonised. In some cases a little cream, mixed with thin arrowroot, is better than anything else. As convalescence advances, ground rice may be substituted for the arrowroot, and afterwards biscuit powder. Sugar was specially objected to by Cruveilhier, and subsequent writers have endorsed his opinion; it seems to produce flatulence.

Some persons are unable to digest milk, and even peptonised milk. Animal broths must then be given in its place; but many patients suppose themselves to have digestive idiosyncrasies who are afterwards found to do just as well as others upon the most rigidly restricted diet.

During convalescence from ulcer of stomach, the most extreme care should be exercised as the patient gradually extends his range of diet. The quantity of food taken at one time must be such as will not distend the stomach; all hot food or drink must be avoided, and alcoholic stimulants are best forbidden.

Scarcely less important than rigid diet in the treatment of gastric ulcer is strict confinement to bed. Among drugs bismuth, opium, morphia, and hydrocyanic acid are the best adjuvants. In addition to these drugs, Dr Frazer, of Edinburgh, has recommended bichromate of potash ('Lancet,' April, 1894). Nux vomica and rhubarb are valuable when the more severe symptoms have passed off, and a course of steel with aloes and myrrh is useful in completing the cure.

It must not be forgotten that pressure upon the epigastrium may do harm. A woman should not be allowed to wear her stays, nor a shoemaker to use his last. Care must be taken, in manipulating the abdomen, to use only very gentle pressure; and all violent exercise and sudden efforts must be carefully avoided.

Lastly, it is possible that a healed or nearly healed ulcer may relapse. Convalescence must therefore be slow, and extreme care in diet should be persisted in for several months after subsidence of symptoms.

In cases of perforation of a gastric ulcer, the only formerly possible treatment was by starvation and opium. This was merely palliative in object, although, as in similar cases of peritonitis following perforation in the course of enteric fever or typhlitis, the result was occasionally and beyond expectation successful.

With the justly increased boldness of modern surgery, it is now our duty in cases of acute peritonitis from perforation of a gastric ulcer, as in cases of a similar catastrophe in the course of typhlitis, gall-stone, or even typhoid

ulceration, to consider the operation of abdominal section, washing out the serous cavity, and closing the ulcer. Theoretically this is the only rational and effectual treatment; but practically, as in other abdominal diseases, the diagnosis is in many cases much more obscure and the practice in most cases much more difficult than one would suppose. A patient seen for the first time in a condition of collapse with abdominal pain and tympanites, often accompanied by vomiting and constipation, may be suffering from perforation of the stomach, the intestine, or the gall-bladder, from rupture of an extra-uterine pregnancy, from intestinal obstruction, or from acute peritonitis having its origin in the pelvic organs or in some other source of infection. In the absence of a trustworthy history, or still worse with inaccurate and misleading history, it is sometimes impossible to decide between the possible causes of peritonitis, including even such traumatic causes as the unskilful performance of an operation to bring about abortion. If we decide (as is in most cases the less dangerous alternative) to operate, the state of the patient is often such that to do so with any chance of success we must revive the pulse by subcutaneous injections of brandy, ether, or strychnia, and restore the temperature by hot-water bottles and bandages. Even if our knowledge or information about the symptoms enables us to decide with some confidence on gastric perforation, and if the patient's condition allows of immediate operation, the difficulty of finding the ulcer is often great, particularly with artificial light. In the last case in which the writer advised operation, on introducing a single finger into the incision, he felt the ulcer at once in the front wall of the stomach near the lesser curvature; but when it is situated in the posterior wall or in the cardiac pouch, the search for it is often most difficult and tedious.

Nevertheless numerous successful operations have been performed, and although the unsuccessful ones are probably more numerous, there is no doubt that when the stomach was not full of food at the time of rupture, when the surgeon is called on to operate within twelve hours, and when the patient's condition is favourable, not only is this treatment justified, but it is the only one that can be justified; for it affords a reasonable hope of saving life—and at the same time removing a painful and sometimes dangerous disease.

When perforation has taken place several days and the patient is still alive, the case is probably one of subphrenic abscess, and the best treatment will probably be to feed the patient by the rectum, to relieve pain by opium, and when the condition allows it, to open and drain the cavity which has been formed by adhesions; and finally, to excise and sew up the ulcer at leisure.

Operation for the cure of an unperforated gastric ulcer has been advocated and performed; but the prospects of recovery by medical treatment are too good for the still formidable operation of laparotomy to be justified, at least in the majority of cases.

Duodenal ulcer.—When an ulcer is seated in the first part of the *duodenum*, the pain is said to come on between half an hour and two or three hours after meals, or even later still: vomiting is not very common, but (according to Krause, who has published a monograph on this disease) hæmorrhage has occurred in one third of all recorded cases. Krause collected fifty-eight cases of duodenal ulcer in men and only six in women—a curious reversal of what is generally believed to be the rule for gastric

ulcer. Perry and Shaw found 48 cases in men to 16 in women (besides five cases of ulcer after burns, which is also more common in males). The pathology, anatomy, and effects of gastric and duodenal ulcer are so nearly identical that, clinically, they may be considered as one and the same. The dangers also are the same, hæmorrhage and perforation.

Several cases are recorded in the 'Pathological Transactions,' the earliest by Murchison (1857, vol. ix, p. 197), who refers to Dr Budd's account of the lesion in his work on 'Diseases of the Stomach;' others by Dr Norman Moore and Dr F. C. Turner in 1880, 1882, and 1884. The best account of this disease and of other affections of the duodenum will be found in an article by Drs Perry and Shaw in the 'Guy's Hosp. Rep.' for 1893. The following case, under the writer's care in 1893, proved fatal by perforation.

A man of thirty-three, previously healthy, began a fortnight before his death to suffer from pains in the chest and abdomen, and vomited on three occasions. On the fifth day of his illness (November, 1893) he was suddenly seized with severe abdominal pain and continued vomiting. When first seen there was tympanitis, constipation, and all the symptoms of acute peritonitis. After consultation with a surgeon an exploratory operation was thought unjustified. He was relieved by opium, and was fed by the rectum till he died, a week after being seen.

After death circumscribed suppurative peritonitis was found shut off by adhesions between the liver above, the stomach and duodenum behind, and the transverse colon below; it communicated with the duodenum close to the pylorus by a round punched-out ulcer. There was a second shallow and apparently healing ulcer close up. An abscess had formed, distinct from the larger cavity above described.

Ulceration of the duodenum in patients dying from the effects of burns was first noticed by Curling (1842). Wilks found it to be an occasional, almost a rare event, and Perry and Shaw recorded only five cases out of 149 autopsies; but Holmes found it in 15 out of 125. The pathogenesis is uncertain.

CANCER OF THE STOMACH.—This disease is not only fatal but frequent, and there is reason to fear is becoming more so. Of all forms of cancer in men, that of the stomach is probably the most common, more so than cancer of the rectum; and of all forms of cancer in women, that of the stomach is probably also the most common, more so than cancer of the uterus or of the breast. At Guy's Hospital there were 79 cases of cancer of the stomach in 5990 autopsies, or more than 1·3 per cent. Brinton believed it to be less common than gastric ulcer; but the records at Guy's Hospital during twenty years show that there were rather more than twice as many fatal cases of cancer as of ulcer of the stomach.

Anatomy.—Three local varieties of this disease have been described, as it affects the cardiac orifice, the body of the stomach, or the pylorus.

As regards the first of these, Dr Fagge believed that most of the cases recorded as examples of cancer affecting the cardia were really cancer of the end of the œsophagus, extending into the adjacent part of the stomach. The 'Pathological Transactions' seem not to contain a single example of cancer beginning in the stomach at its œsophageal end; nor does the museum of Guy's Hospital show any specimen in which the lower end of the œsophagus is not also affected. This conclusion is, however, disputed by Perry and Shaw (Guy's Hosp. Rep., 1891, vol. xlviii, p. 146), who justly remark that a spheroidal celled cancer is far more likely to have begun in the stomach than in the œsophagus (*supra*, p. 309).

Cancer of the body of the stomach and cancer of the pylorus have each distinguishing characters. The latter is the more uniform and more frequent affection of the two. Of fifty cases at Guy's Hospital, collected by Dr Shaw in 1889, thirty-six occupied the pylorus, three the cardia, and eleven the body of the stomach, both orifices being free.

As Wilks and Moxon state: If a line be drawn from an inch to the left of the œsophagus to a point on the lower border of the stomach four inches from the pylorus, the part to the left of this line will be found very rarely to suffer from cancer.

The *pylorus*, when affected with cancer, becomes greatly thickened, so that it forms a rounded swelling, often somewhat lobulated, which is almost always sharply defined towards the duodenum, while it passes gradually into the wall of the stomach, or extends for some distance along its lesser curvature. The disease generally involves the whole circumference of the orifice, which is consequently much narrowed. It grasps the finger tightly, or may be too narrow to admit it; but cases are very rare in which a large catheter cannot be passed into the duodenum. The mucous surface may either be smooth or present nodular excrescences, and sometimes distinct villous growths. Most frequently it is more or less extensively ulcerated, and sometimes greatly thickened.

On longitudinal section, the several coats of the stomach are still plainly to be recognised. The thickest part of the mass corresponds with the sub-mucous connective tissue, and generally makes up two thirds of the whole. Next comes the muscular layer, which is likewise thickened, and presents pinkish-grey translucent lines, alternating with opaque bands of cancerous tissue. The subserous tissue is also thickened, though to a less extent, and is generally infiltrated with the new growth. The peritoneal surface is often unaffected, but it may be adherent to the adjacent parts, or present more or less numerous cancerous nodules. The mucous surface is sooner or later destroyed and replaced by a fungous bleeding surface, like that of malignant ulceration of the breast, the tongue or the colon.

Cancer of the *body of the stomach* is much more variable in its characters. In many cases it begins along the lesser curvature, and then it may either remain limited to that part, or spread to one or both surfaces. In one case of this kind only a narrow border along the greater curvature was left untouched by the invading growth. Occasionally it forms a broad ring completely surrounding the middle of the stomach, and giving it an hour-glass shape.

A cancer of the stomach is very liable to become adherent to the under surface of the liver, and extend into the gland, until a large cavity may be produced, in which food (such as grape skins) may lodge. Or the diseased portion of the stomach may become fixed to the surface of the abdomen; in one instance the anterior part of the abdominal wall had altogether disappeared, being fused in a mass of cancer two or three inches thick. A perforation is exceptional, and a gastro-cutaneous fistula almost unknown.

The growth may become continuous with a mass of diseased glands near the pancreas, and at last with that structure itself. Or the first portion of the duodenum may be drawn into adhesion with the back of the diseased pylorus; and sometimes an ulcerated opening forms between them, behind the proper orifice. This depends on the fact, pointed out by Luschka, that the normal direction of the first part of the duodenum is from before

backwards. Lastly, the diseased part of the stomach may become adherent to the colon, and a fistulous communication may form between them (p. 361).

Histology.—Malignant disease of the stomach is with few exceptions true carcinoma. In ten years at Guy's Hospital (1880-1889) there were reported from the deadhouse forty-five cases of alveolar carcinoma, one of colloid, and three of sarcoma of the stomach.

A case of primary *sarcoma* of the stomach, was recorded by Dr Wilks in the tenth volume of the 'Pathological Transactions' p. 146). In this instance, in another cited by Virchow, and also in a third case of Dr Wickham Legg's (*ibid.*, vol. xxiii), the patient was a young girl; and in the first both the ovaries were affected with sarcoma. In a fourth case of Dr Cayley's in a man aged fifty-seven (*ibid.*, vol. xx, p. 170), there were large nodules of the growth projecting into the cavity of the stomach, and extensively ulcerated.

Several cases of pyloric sarcoma have been observed at Guy's Hospital. One was a woman aged forty-seven; the submucous tissue of the pylorus was three quarters of an inch thick, and Moxon described it as thick but flabby, of a milk-white colour, yielding a clear fluid when scraped, consisting mainly of a well-developed fibrous tissue, but also containing some delicate spindle-cells with very large tails. In another case, which occurred in a man aged sixty-six, the pylorus was the seat of a new growth of yellowish look and of firm consistence, which proved to be a round-celled sarcoma. A third case was that of a man aged sixty-seven. The pylorus, through which the finger could readily be passed, presented a large ulcer seven inches in circumference, the base and sides of which were formed by a homogeneous pinkish-white substance, which yielded no juice, and consisted of round and oval cells and spindle-cells, embedded in an intercellular substance containing mucin. In the museum of Guy's Hospital there are two cases of sarcoma involving the pylorus, beside others of the body of the stomach, one of them melanotic (Nos. 717—721).

Cases of pyloric tumour have been described as not scirrhus, but due to a local thickening of the submucous tissue, with hypertrophy of the muscular coat. The growth is dry and without juice, consisting mainly of fibrous tissue. The absence of secondary cancerous nodules is not by itself conclusive; for, out of forty-one cases of true carcinoma of the pylorus in succession at Guy's Hospital, there were at least five in which no cancer existed elsewhere in the body, while in one case, which would otherwise have been regarded as simple fibrous thickening or hypertrophy, there were secondary nodules in the liver. Some cases of fibrous pylorus were found by Mr Bland-Sutton to be really innocent adenomata, and there is no doubt that though rare, this is an occasionally observed lesion. Other cases of non-cancerous stricture of the pylorus are congenital (No. 666).

As Waldeyer first showed, cancer always begins in an overgrowth of the glands in the mucous membrane. These become elongated and dip down into the adjacent connective tissue; when they have reached it they proliferate actively, and so give rise to a cancerous nodule, which spreads out horizontally and is only connected with the glandular layer at its starting-point.

If the fibrous stroma be abundant and the alveoli small, the growth has a tough fibrous appearance, and yields but very little juice. If the stroma be scanty and the alveoli large, the growth is soft and of a milk-white colour, and yields much juice when scraped. But between the former

("scirrhus") and the latter ("encephaloid or medullary") all gradations exist, and in some cases it may be difficult to say under which head the disease should be placed. As a rule, however, carcinoma of the stomach belongs rather to *scirrhus*; according to Brinton, three cases out of four, and among cases affecting the pylorus more than half. As already stated, the tumour grows towards the serous surface between the bundles of the hypertrophied muscular coat. On the other hand, as Moxon pointed out, medullary carcinoma often destroys the muscular coat over a considerable area; so that the whole thickness of the wall is converted into a uniform mass of disease. Sometimes branching processes sprout from the mucous membrane, which are each made up of a central blood-vessel, clothed with thick layers of well-formed cells. These have been termed "villous cancer." Moxon described a case of this kind, in which the floor of the growth was formed by a large mass of soft carcinoma, growing directly into the substance of the liver. Non-malignant villous growths may also be found in the stomach.

In both the soft (medullary) and hard (scirrhus) kinds of cancer, the epithelial cells in the alveoli are, as a rule, *spheroidal*. But frequently a cancerous growth in the stomach presents the characters of a *cylinder-epithelioma*. In sixteen cases microscopically examined at Guy's Hospital, there were five of this cylindrical-celled carcinoma, and one in which, while most of the alveoli contained spheroidal cells, a few contained cylindrical ones.

The growth appears to be always spheroidal or cylindrical, never squamous carcinoma. On this and other points see the valuable article by Drs. Perry and Shaw in the 'Guy's Hospital Reports' for 1893 (vol. xlviii, p. 116), and the excellent monograph on the subject by Osler and McCrae (1900).

The writer once met with a remarkable case of malignant lymphadenoma of the stomach. The patient, a man of forty-eight, was admitted into Guy's Hospital in 1888 with clinical symptoms of an intra-thoracic growth pressing on the left bronchus and causing pleural effusion. After death, besides multiple tumours of the mediastinal lymph-glands, similar lymphatic growths were found in the jejunum, the cæcal appendix and the submucous tissue of the stomach. The last consisted of numerous tumours, some flat, some more diffused, and some prominent and polyp-like (see 'Path. Soc. Trans.,' vol. xl, p. 80; Museum Catalogue, No. 716).

Cancerous growths in the stomach, as in other parts, are liable to undergo fatty degeneration. Sometimes scarcely a trace of active growth is discernible, although the patient had died of an extension of the disease to other parts.

Another kind of new growth to which the stomach is particularly subject is that known as *colloid cancer*. Sometimes the whole thickness of the organ is infiltrated with a jelly-like material, with no appearance of ordinary carcinoma. But more frequently, while some parts of the growth have the colloid character, others have those of spheroidal cancer; and under the microscope it is not uncommon to find more or less colloid change where it was not observed by the naked eye. In such cases it is found that the alveoli are no longer filled with the characteristic epithelial cells, but contain a structureless translucent substance. As this increases, the cells gradually disappear, the alveoli become spherical, the septa between them break down; and thus large translucent globules are formed which, under the microscope, appear almost structureless.

The colloid structure is like that of a myxoma, and a colloid carcinoma may be compared to a myxosarcoma.

Colloid cancer is less apt to infect distant parts than other carcinomata ; but it often spreads over the peritoneal surface, and produces masses of enormous size. It is a remarkable fact that these secondary nodules of colloid cancer reproduce, not the supposed original structure of the primary growth, but its later characters. Thus the lungs may be found studded with translucent gelatinous nodules of typical colloid character.

This was the case in a patient of the writer's in whom a large colloid cancer of the stomach, which produced scarcely any symptoms during life, led to the deposit of innumerable, glistening, transparent nodules, varying from a pin's-head to a pea in size, which filled both lungs and were certainly recent. Such cases make one doubt whether the colloid structure is entirely degenerative.

Whatever its histological character, a cancer of the stomach is almost always ulcerated ; and often a deep sore is formed with hard, raised, ragged edges and a sloughing base. This may lead to erosion of blood-vessels and fatal hæmorrhage. It is possible that the digestive action of the gastric juice may help to detach large masses of the cancer.

Occasionally cancer infiltrates the walls of the stomach and causes great thickening, induration and contraction ('Guy's Hos. Rep.' xlviii, p. 147).

Anatomical results.—When the pylorus is the seat of cancer, the obstruction causes enormous dilatation of the stomach. It will sometimes hold six or seven (instead of about two) pints, and may fill the whole abdomen, while its greater curvature sweeps round just above the pubes. Its walls are usually thickened by hypertrophy of the muscular coat, but sometimes they are exceedingly thin.

In some cases, however, cancer of the pylorus, like cancer of the rectum, destroys more than it obstructs ; or vomiting is so frequent that no accumulation in the stomach takes place ; or the patient's appetite is so bad that scarcely any food is swallowed ; while in other cases the extension of cancer along the lesser curvature tethers the stomach and prevents its dilatation.

When the lesser curvature is the seat of the disease, the cardiac and pyloric orifices may be approximated by the contraction of the growth ; and the anterior and posterior walls of the stomach may be so flattened against one another that scarcely any cavity is left.

It is very rare for a cancerous ulcer of the stomach to perforate. The thickening of the coats, the extensive adhesions, and the comparatively early termination of the case from other causes than the local ulcer, may probably account for the fact. The following case is, however, a proof that it may occur :

A woman of fifty-six was admitted in May Ward under the writer's care in November, 1894, with symptoms of acute probably perforating peritonitis. There had been vomiting and hæmatemesis, but these symptoms had subsided before admission. There was tympanites and also ascites. She died very soon, and after death nineteen pints of serum were found in the abdomen with recent lymph. An oval orifice was found in the lesser curvature near the pylorus, and on opening the stomach it was found to be surrounded by a thick margin of cancer. There were secondary deposits to attest its nature.

Ætiology.—Of the causes of cancer of the stomach very little is known. It occurs chiefly in persons over forty years of age, is most common between

fifty and seventy, and rare under thirty. But about 12 cases are on record of its occurrence under twenty, and half that number under ten.* In patients between twenty and thirty the disease, as observed by Mathieu, and confirmed by Osler and McCrea, runs a very rapid course. Of 46 cases at Guy's Hospital, collected by Dr Fagge, 11 patients only were under the age of forty; of the remaining 35, there were 16 between forty-one and fifty, 11 between fifty-one and sixty, and 8 between sixty-one and seventy. In 47 additional cases, observed by the writer, the youngest patient was thirty-one, the oldest 77, and the average age was somewhat over fifty—numbers which correspond generally with those given by Brinton.

Among 124 of our cases, 82 occurred in men, and 42 in women. This also accords with Brinton's figures, who collected 440 cases in men and 344 in women; and Welch, in over 2000 cases, found that 1233 occurred in men and 981 in women. During the period 1872 to 1898, there were 2387 fatal cases at Hamburg in men, and 1850 in women (Reiche in 'Deut. Med. Wochenschr.,' 1900, cited by Osler).

Hereditary predisposition is said to be well marked in some cases, and the case of the Napoleon family is cited in proof of the fact. In 46 patients of Guy's Hospital whose family history was recorded with sufficient detail, Dr. Shaw found that cancer had been present in one of the sisters twice, and in one of the parents of the patient five times; in four of these seven cases the cancer was gastric in locality. The hereditary cases have been estimated as from 12 to 1 per cent. of the total, more than mere chance would produce.

As we have seen, cancer may develop in a gastric ulcer (p. 350). Some writers admit the influence of irritant poison, of local injury, of chronic dyspepsia, and of depressing emotions in producing cancer of the stomach, as of other organs, but on insufficient evidence. Negatively, cancer of the stomach is quite unconnected with tubercle, with syphilis, or with drink; but neither do any of these diseases, nor chronic gastritis or ulceration protect from cancer.

Symptoms.—At first the symptoms of cancer are most indefinite: the patient begins to complain of discomfort after his meals, he is troubled with acid eructations. His tongue remains clean, yet he has no appetite, and finds that he is losing flesh. The uneasiness at the pit of the stomach passes into pain; a dull aching usually, but sometimes of a severe burning or lancinating character. It is generally more or less increased by meals, but it is by no means limited to the periods at which the stomach contains food. Occasionally moderate pyrexia is present, but it does not appear to have any diagnostic import. Anæmia is constant and leucocytosis frequent.

Vomiting is the next symptom. When the seat of the disease is the middle of the stomach, vomiting may come on soon after meals; when it is the pylorus, the food is usually retained for three or four hours, *i. e.* until it should be passed on into the duodenum. The matters rejected consist at first of partially digested food or mucus, but soon these are streaked with altered blood, which is of a brown or black colour; or they may contain sufficient blood to resemble coffee-grounds. Occasionally the vomit is horribly foetid, probably from sloughing of the growth in the stomach. Constipation and flatulence are common symptoms. The aspect of the patient is altered; he acquires a pale, sallow, or earthy complexion.

* One in a child 8 years old is recorded by Ashby and Wright. Some of these few may possibly have been really hypertrophy of the pylorus (p. 355), but this one was definitely ascertained to be a columnar-celled carcinoma.

and becomes depressed, irritable, and morose. But the most characteristic symptom is progressive loss of flesh.

In some instances most of these symptoms are absent. Cancer of the lower end of the œsophagus extending into the adjacent part of the stomach is often latent, and most of the cases in which gastric carcinoma has run its course without producing any marked symptoms have been cancer of the cardia. But Watson relates a similar case, in which the disease occupied the greater curvature.

A gentleman, between forty and fifty years of age, was on his way home from Scotland (where he had been deerstalking and shooting grouse), when he was seized one night in a London hotel with a deadly faintness, rapid breathing, and severe pain referred to the sternum. He had before been gradually losing flesh and strength, but the only definite symptoms of which he had complained were sour eructations, loss of appetite, and repugnance to solid food. Sir Thomas Watson could detect no physical sign of disease. The next night the patient had a similar paroxysm and died. The greater curvature of the stomach presented throughout its whole extent a mass of scirrhus, while the cardiac and the pyloric orifices were free.

In a patient under the writer's care in Guy's Hospital with colloid cancer of the stomach, there was scarcely any vomiting, and no complaint of local pain during the three months before his death. The diagnosis made during life was cancer of the peritoneum, and this was present, but it was secondary to that of the stomach.

Physical signs.—Examination of the abdomen, in cases of dyspepsia with loss of flesh in a patient above fifty, should never be omitted. The patient must lie down in an easy posture, with the shoulders low and the knees bent, and he should breathe deeply. After the abdomen is exposed, its shape must be observed, and particularly whether there is any fulness of the epigastrium or the reverse. Sometimes a tumour may be seen through the parietes, but most commonly it is to be detected only by manipulation. Beside palpation of the stomach, inspection with a favourable light should never be omitted; the rise and fall of deep respiration may show the movements of a tumour as plainly as the most skilful handling. The abdominal muscles are often very rigid, particularly the upper parts of the recti, and careless handling may throw them into contraction, so that nothing can be felt. If the patient's attention can be concentrated on his breathing, or can be diverted by conversation, his abdominal muscles will often relax. The palm of the hand (which must never be cold) should be gently laid on the abdomen, and allowed to rise and fall as the patient breathes; gradually slight pressure is made, and increased until the abdomen has been thoroughly explored. During all this time the palm of the hand, as well as the fingers, should be kept evenly applied to the surface, and all sudden movements of the fingers which might excite contraction in the abdomen muscles must be avoided.

The position and form of the tumour produced by cancer of the stomach are determined by the seat of the growth. If it occupy the middle of the organ, the epigastric region, a little to the left, will contain any mass that can be felt on manipulation. In one case two nodular ridges could be clearly made out, corresponding one with each curvature; while between them, and further back, lay an irregular mass, which seemed to occupy the posterior wall. In other cases a more or less rounded prominent mass is felt, which is the thickened anterior surface of the stomach. Dr Cayley has related a case in the 'Pathological Transactions' (1869) in which the left hypochondrium contained a firm but slightly moveable tumour which

reached below the umbilicus, and was supposed to be the spleen, but it proved to be sarcoma of the stomach.

When the pylorus is the seat of the cancer, the tumour is usually definite, and in some cases its character can be made out almost as plainly during life as in the deadhouse. It forms a rounded mass, perfectly circumscribed on all sides except towards the left, where it can sometimes be felt to pass gradually into the wall of the stomach; it may vary in size from that of a walnut to that of a billiard ball. Its seat is usually a little above and to the right of the umbilicus; considerably lower than the position of the normal pylorus, which lies so completely under cover of the liver as to be inaccessible to palpation.* When the lesser omentum is thickened and involved in the growth, or when the pylorus is retained in its normal position by adhesions, no tumour can be discovered.

Sometimes it descends much lower, as low as the right iliac fossa, or even the pelvis. In one case a tumour in the left hypochondrium, of which the exact situation varied at different times, according as the stomach was more or less distended, proved to be the pylorus, which had been dragged over to the left side, and was firmly adherent to the parietes and to the edge of the liver. A scirrhus pylorus usually moves slightly downwards when the patient draws a deep breath, being pushed down by the diaphragm; but in some cases the movement is rather apparent than real, depending on the expansion of the ribs carrying the abdominal walls upwards over the tumour.

The tumour caused by cancer of the pylorus often receives an impulse from the abdominal aorta, which may stimulate a coeliac aneurysm. In most cases it is moveable, and occasionally very much so.

It varies in the ease with which it can be felt, and may even disappear for days together if overridden by a distended colon.

To percussion it should yield a dull note, but when it is of small size this is masked by the resonance of the adjacent intestine.

Another important sign is visible peristalsis of the stomach, the wave passing from the left to the right.

Among physical signs may be mentioned the results of examination of the contents of the stomach, not only when vomited, but when obtained by the stomach-tube three hours after a test-breakfast. One of the most important points is deficiency of the normal hydrochloric acid, also the presence of lactic acid, both of which conditions are common in cases of carcinoma.

Gastric dilatation.—The result of obstruction of the pylorus by cancer is to permanently dilate the stomach. The greater curvature descends lower than the umbilicus. It may even reach to the pubes, and the epigastric and left hypochondriac regions are then deeply hollowed, while the lower part of the abdomen is protuberant. This is common enough in persons whose small intestines are distended with flatus, if their abdominal walls are also loose and flaccid; but what is conclusive is detection of the peristaltic movements of its thickened walls. If the surface of the abdomen be attentively watched, a wave of contraction may often be seen to start from the left hypochondrium, descend below the umbilicus, and pass on to the right side, and then a little upwards towards the cartilages of the

* The fact that a cancerous pylorus is often felt in the umbilical region was recognised by Brinton and attributed to the use of staves; but the same thing is observed in men, and in men who do not wear a tight belt. It probably results from the traction exerted upon the lesser omentum by the weight of the tumour.

right ribs. Or a rounded protuberance, as large as an orange, may rise up on the left side and travel round to the right, in the same way as the wave.*

Another indication of enlargement of the stomach is the production of a splashing sound by manipulation of the lower part of the abdomen, like the Hippocratic succussion sound in character and origin (vol. i, p. 1113).

If we pass an œsophageal tube down into the stomach, its end may sometimes be felt through the abdominal walls. According to Leube it may, in health, reach as low as the umbilicus; but if it descends below that level the stomach must be dilated.

In order to map out the extent of dilatation and the form of the stomach, we may use the ingenious plan introduced by Frerichs of filling it with carbon dioxide. The patient takes first a teaspoonful of tartaric acid in solution, and then rather more than half as much bicarbonate of soda in water; carbonic acid gas is at once set free, and the limits of the stomach are seen. If the greater curvature reach below the umbilicus, the stomach is either enlarged or displaced. This plan is safer than pumping in gas, or than introducing a sound and measuring the distance to which it can be passed; but even distension with gas must be used with caution.

In ordinary cases of cancer of the pylorus the patient vomits about three or four hours after each meal, when digestion is completed, and when the food ought to be passing into the duodenum; but when the stomach is dilated vomiting is much delayed, and when it does occur the patient may bring up several pints of fluid at a time. In one instance vomiting never occurred except at night, and sometimes the stomach rejects its contents only at intervals of some days.

The matter thus vomited consists of a thin, highly acid liquid, of a dirty grey, brownish, or greenish colour, which, on standing, becomes covered with a thick, frothy, yeast-like scum, while it deposits a more or less abundant sediment. In the scum, as well as in the liquid, numerous oval spores and beaded threads of the yeast plant (*Torula cerevisiæ*) are often found; and in still greater numbers the *Sarcina ventriculi* (cf. vol. i, p. 19). They are divided by cross-lines into smaller rectangles, some into four, others into sixteen, and some into sixty-four, according to their size. They thus resemble packages tied across again and again by cords; and Goodsir, who in 1842 was the first to observe them, gave them their appropriate name (*sarcina*, woolpack). Chambers found them after death in stringy mucus adherent to the interior of the stomach.

Contraction of the stomach is a much less common result of cancer than dilatation. As the latter is the result of pyloric obstruction, so the former follows extensive disease of the body of the organ. The gastric capacity is much diminished, and vomiting takes place soon after food, and in small quantity.†

Gastro-colic fistula is a rare and remarkable result of cancer of the stomach; the condition was well described by Murchison in the 'Edinburgh Medical Journal' for 1857. In almost all cases there is stercoraceous vomiting; according to Sir William Gairdner, the only exception is when there is at the same time narrowing of the pylorus, so that the stomach is constantly kept overloaded with its proper contents. The patient's breath often has a foul odour, and he is liable to eructations of

* I have more than once observed distinct antiperistaltic movements (from right to left) in a hypertrophied stomach.—C. H. F.

† See several cases reported by Osler and McCrae (loc. cit., pp. 83—91).

intolerable factor, or a horrible taste in his mouth. The fistula sometimes allows matters to pass the other way, from the stomach into the colon; but this appears to be much less frequent, for there are only seven out of the twenty-three cases collected by Murchison in which undigested matters were recognised in the fæces. This last condition is termed *lientery*.* It must not be supposed to be of itself a proof that a fistula exists. The attempt has been made to increase its significance by giving food coloured with cochineal to patients who pass undigested matters from the bowels, and by observing what length of time elapses before the colouring matter appears in the evacuations. In a case of Schönlein's this occurred only at the end of twelve hours, in the last of seven evacuations that took place during that period. He inferred that the case was not one of gastro-colic fistula; and (with less reason) that the lientery was due to widening of the pylorus. In patients who have an opening between the stomach and colon the appetite is generally very bad, but in one instance there was craving for food. Pain is not invariably present. Indeed, the formation of the fistula sometimes leads to the relief of pain that had before existed.

Sometimes a gastric cancer opens into the third part of the duodenum. In a case recorded by Dr Fagge this gave rise to no symptoms.

The most rare and remarkable complication of cancer of the stomach is adhesion of the new growth to the anterior abdominal walls, and infiltration of both muscles and skin, so that, without perforation, the disease reaches the surface and presents the form of *squirrhe en cuirasse* (vol. i, p. 92).

In the latter stages of gastric carcinoma, either with or without the presence of a tumour, the liver is often found to be enlarged and painful. Jaundice may appear from secondary hepatic carcinoma, or from pressure on the common bile-duct; ascites, from extension of the cancerous growth to the peritoneum, or from compression of the portal vein; œdema of one or both of the lower limbs, from thrombosis of the corresponding femoral or external iliac vein or veins.

It is a remarkable fact that towards the fatal termination of the disease its symptoms often subside, particularly the pain and vomiting; but emaciation goes steadily on. In some cases the patient lies for several days before his death with cold extremities, and a scarcely perceptible pulse, but suffering no pain.

Diagnosis.—The early detection of cancer of the stomach is very difficult. When gastric pain comes on in a patient over forty years of age, with loss of strength and of flesh, the possibility of cancer must always be borne in mind. When vomiting and hæmatemesis are added, the diagnosis lies between cancer, ulcer, and cirrhosis of the liver. When a tumour can be felt the diagnosis is usually complete.

When a tumour cannot be felt, the most important sign of cancer of the pylorus is passive dilatation of the stomach. This may be recognised by the altered resonance, by the form of the abdomen, by visible peristalsis, and by splashing on succussion.

* The term *λειεντερία* (*leĩos*, *levis*, smooth; *έντερον*, intestine) was applied in Greek medicine to cases of diarrhœa in which food or drink, as soon as taken, seems "to run through the body" without being digested. In ordinary cases the stimulus of food in the stomach provokes peristalsis, but the stools consist of mucus or other intestinal contents.—'Επί διαρρόϊη, δυσεντερία, ἐπὶ δυσεντερίῃ λειεντερίῃ ἐπιγίνεται (Hipp., 'Aphor.,' vii, 76).—Intestinorum levitas, quâ continere nihil possunt, et quidquid assumptum est imperfectum protinus reddunt (Cels., lib. iv, cap. xvi).—Levitas intestinorum, Græce λειεντερία, est velox exitus eorum quæ comeduntur atque bibuntur, quæ talia dejiiciuntur qualia fuerunt devorata (Stephani, 'Vocab. med. expos.,' 1564).

Between gastric ulcer and cancer the diagnosis is often easy. In young adults, malignant disease scarcely ever occurs in the stomach except as a sarcoma, attended with ascites, but with slight gastric symptoms.

In cases of ulcer, the pain and sickness are much closer to the time at which food is taken than in those of cancer. Vomiting of blood in considerable quantity is much more apt to occur, and that at an early stage of the disease, whereas coffee-ground vomiting and occasional scanty hæmatemesis is more frequent in cases of cancer. We may say of any doubtful case in which well-marked symptoms have existed eighteen months or more, that it can scarcely be one of malignant disease.

Cases of simple ulcer affecting the pylorus have been recorded in which this part has been so thickened and indurated as to simulate a scirrhus mass; and when an ulcer occurs at this part of the stomach, it tends, when it heals, to narrow the orifice, and so cause dilatation. Congenital stricture of the pylorus is only a possibility in young subjects.

The diagnostic value of a pyloric tumour is very great, even when there are no other symptoms of gastric disease. A pulsating tumour in the epigastrium has often been mistaken for an aneurysm or a mass of fæces in the transverse colon, but has proved to be cancer of the pylorus. On the other hand, it must not be forgotten that in many cases of cancer of the stomach, and in some of cancer of the pylorus, no tumour can at any time be discovered.

Free hydrochloric acid is almost absent from the fluid removed by a siphon in cases of cancer of the stomach. But it may be absent in cases of chronic gastric catarrh and even in persons presumably healthy.

The presence of lactic acid is usual in cases of cancer, but this is less constant than the absence of hydrochloric acid.*

Prognosis.—The duration of cancer of the stomach cannot be stated with precision, because we have no means of fixing the date of its commencement. But it seldom fails to destroy life within a year or eighteen months from the appearance of well-marked symptoms.

Brinton estimated this period at an average of twelve and a half months; Niemeyer, at from five to fifteen months; Martin, at eighteen months. Wilson Fox quotes as the most rapid case one recorded by Valleix, in which death occurred in four months. But at Guy's Hospital four cases have occurred, in which the duration of the symptoms was stated at four weeks, five weeks, nine weeks, and three months. The longest case known to Fox was one in which the patient lived three and a half years after the first symptoms; the longest of Osler's, two and a half years. Napoleon had paroxysms of severe pain for nine years before his death in St Helena, but there is no reason to regard them as symptoms of the cancer of which he died. They had all the characters of acute gastralgia, and he continued stout and well for long after their occurrence. If indigestion never preceded cancer of the stomach, it would prove that the one malady protected from the other.

* See the seventh number of the 'Berliner klinische Wochenschrift' for 1887, and Nos. 6 and 7 of the same periodical for 1888; also a judicious paper on modern methods of diagnosis in diseases of the stomach, by Dr F. C. Shattuck, of Boston ('Trans. Assoc. Amer. Phys.,' May, 1890), and a more recent abstract of the present state of the question, by Mr Shufflebotham, in the 'Guy's Hospital Gazette' for November 6th, 1897 (p. 515).

For details of the various colour-tests used for determining the amount of free hydrochloric acid in the contents of the stomach, of the same combined with albuminous foodstuffs, and of organic acids (lactic, acetic, or butyric), see Gamgee's 'Physiological Chemistry' (vol. ii, p. 498) and Martin's 'Diseases of the Stomach' (pp. 124—137).

Treatment.—With regard to the medical treatment of cancer of the stomach there is, unfortunately, little to be said. The patient's diet should be only limited by pain, and the plan of treatment by abstinence recommended for cases of simple gastric ulcer is quite inapplicable here. Alcoholic stimulants may be allowed, and even prescribed.

Medicines are only required for the relief of symptoms, and those remedies available in the treatment of gastric pain, of vomiting, and of hæmorrhage have already been fully discussed (*v.* pp. 331, 336, 341).

When chronic stricture of the pylorus leads to great dilatation of the stomach with its resultant discomfort, it is justifiable to employ the siphon in the way described above (p. 329). The counter-indications are hæmatemesis and evidence of affection of the body as well as the pyloric end of the stomach. In suitable cases the writer has seen remarkable benefit from this treatment.

In favourable conditions surgical treatment is now frequently successful. The possibility of the operation was proved by Heidenhain in the course of his experiments on the gastric and pyloric secretion in dogs. Pylorectomy was first practised on a human being by Billroth, and afterwards by Péan in Paris, by Senn and other American surgeons. In this country Maylard performed excision of the pylorus on a patient of Dr Coats at Glasgow ('*Brit. Med. Journ.*,' July 24th, 1886), and Sir Wm. Stokes in 1890 (*ibid.*, vol. i, p. 997). Many other surgeons have followed Billroth's lead, with a few cases of immediate death, a considerable number of temporary recovery, and a very few of final cure.

Other surgeons following Prof. Loreta (1882), of Milan, have opened the stomach and forcibly dilated the pylorus with the fingers.

When excision of the growth is impossible, the obstruction may be relieved by making a communication between the stomach and the first part of the jejunum (gastro-enterostomy). This "short-circuiting" has been followed by success. Mr A. P. Gould published a case of "jejunostomy" for pyloric cancer in the '*Lancet*' (December 12th, 1885), and Mr. Golding-Bird one in the '*Clinical Transactions*' for 1886, (vol. xix, p. 70). What is needed in this as in other abdominal operations is greater power of early diagnosis. An explanatory operation without a definite object is always hazardous. But at present we must either run the risk of a grave operation that is needless, or often delay until an operation has lost its best chances of success. The reader is referred to a valuable series of papers on gastric operations by Mr Barker, of University College, in the '*Clinical Journal*' for 1898 (April 20th, p. 457), and to Mr Mayo Robson's Hunterian Lectures in 1900 ('*Brit. Med. Journ.*,' vol. i, p. 562).

Secondary cancer of the stomach, as of all epithelial surfaces, is excessively rare.

Innocent growths are also very seldom met with—tubular adenoma, or liomyoma, or innocent villous growth. The writer met with one case of fatal lymphadenoma of the stomach (Cat. Guy's Hosp. Mus., No. 716).

Gastric induration.—A remarkable condition of the stomach is one in which its walls are uniformly thickened. In the most striking cases the cavity is greatly reduced in size. But there may be the same thickened walls with a dilated or normal stomach. This condition has been called

“fibroid induration” or “cirrhosis” of the stomach.* We shall see hereafter that it is occasionally the starting-point of a general chronic peritonitis. The coats of the organ may be from half an inch to an inch and a half thick, and only capable of containing four or five ounces of fluid. The mucous membrane is thrown into permanent rugæ. The muscular coat is hypertrophied, and its interstitial connective much thickened; while the serous tunic loses its natural glistening aspect, and looks of a dull dead white.

The symptoms of this affection are exceedingly obscure. A tumour may be discoverable, and this may be more or less resonant on percussion. It seems at present probable that most cases of this kind are really diffused cancer of the stomach. Fibrous thickening of the coats of the stomach may however be an exaggeration of the thick, pigmented, almost warty condition (*état mamelonné*) of chronic gastritis. It may affect the whole organ, but is more often confined to the pyloric region, and occasionally forms what may be termed a non-malignant fibrous tumour of the pylorus. When it extends to the whole of the stomach, to use Dr Brinton’s comparisons, it retains its form like a large artery or an india-rubber bottle.

Gastric concretions.—Brief mention must be made of certain rare cases in which immense masses of hair and string, matted together and moulded to the shape of the stomach, have been found in its cavity, and in that of the upper part of the intestine. Sir William Gull brought a case of this kind before the notice of the Clinical Society in 1871, and another was related at a meeting of the Pathological Society by Mr Pollock. In the former case the mass, when dried, weighed five and three quarter ounces; it was composed of string, thread, cotton wool, and hair of three colours, that of the patient herself (a woman aged thirty-two) and of her children. She had never been noticed to eat hair; but the person from whom Mr Pollock’s specimen was taken, and who was a delicate girl aged eighteen, had been observed to put hairs into her mouth when only three or four years of age. In that case, a projecting tumour, the size of a large orange, was felt in the epigastric region during life; it was apparently solid and slightly moveable. A tumour was also felt in a third case, referred to by Sir William Gull. It occurred in a woman aged thirty, who for fifteen years had indulged in the habit of eating her hair, and who had suffered all the time from pain in the stomach, but had worked as a servant until six years before her death. In that case the mass weighed thirty ounces.

In a case of the kind, Mr Knowsley Thornton removed from the stomach of a girl of eighteen by abdominal section a mass of hair weighing two pounds, and the patient made a good recovery. He refers to a similar case successfully operated on by Dr Schöborn, of Königsberg, and quotes several instances only discovered after death. In one of these the mass of hair when removed from the stomach was found to weigh four pounds and seven ounces. In lunatics a similar condition is not infrequent. A fatal termination appears generally to occur sooner or later from perforation of the stomach, with consequent acute peritonitis.

* Chronic interstitial gastritis, fibroid infiltration, and cirrhosis or sclerosis of the stomach are other terms used. Brinton proposed the unfortunate term “*Linitis* (from the Homeric *λίνον*, *rete ex lino factum*),” or plastic linitis. Dr Sidney Martin calls it cirrhosis ventriculi, with Brinton’s term as a synonym. He figures a section of the thickened walls, and remarks that two specimens which were put up as examples of this disease turned out, on microscopical examination, to be scirrhus cancer.

FUNCTIONAL AND INFLAMMATORY DISEASES OF THE INTESTINES.

O dura messorum ilia—HOR. *Epod.* iii.

COLIC—onset—causes—diagnosis—prognosis and treatment—Lead-colic—history of its recognition—modes of infection—diagnostic characters—the “blue line”—pathology and treatment—other effects of lead.

CONSTIPATION—Causes and pathology—effects—treatment—general, and of certain clinical varieties.

DIARRHŒA—Acute and epidemic form—symptoms—treatment—chronic forms—Tuberculous enteritis—Diarrhœa from lardaceous and malignant disease—Psilosis—Enteritis.

Acute catarrhal colitis—Ulcerative colitis—Enteritis—Inflammation of the small intestines—Membranous colitis—Dilatation of the colon—Intestinal casts.

DYSENTERY—History—Symptoms—Course—Anatomy—Catarrhal and diphtheritic forms—Sporadic and epidemic dysentery—Ætiology—Diagnosis and event—Prophylaxis—Treatment of acute and of chronic dysentery.

THE organic diseases of the Intestines include those of other mucous membranes, catarrhal inflammation, and cancer, together with effects of infective microbes causing the specific ulceration of Enteric fever, Tubercle and Dysentery. Except in the duodenum there is nothing like the peculiar ulcer of the stomach.

But the most characteristic and frequent of intestinal disorders are those which depend on the physiological process of propulsion of the contents of the bowels through their long and tortuous passage. The peristalsis which accomplishes this task in health is often languid and inefficient, and thus a state of more or less obstinate constipation ensues; or peristalsis is too active, and the colon hurries on the contents of the small intestine without allowing time for their consolidation by absorption of water, and thus the opposite condition of diarrhœa with too frequent liquid motions is the result. Again, if the obstruction is considerable and continued, and the peristalsis active, the efforts of the muscular coat to overcome it are so strong, that they occasion a pain like that known in the limbs as cramp, like that of parturition, and like that of strangury.

We will first consider this colic pain, and then constipation and diarrhœa in order.

COLIC.*—This name has been universally applied to a severe form of abdominal pain or “belly-ache” of a twisting, dragging, or wringing character, generally referred to the umbilicus or to the epigastrium, but moving from one point to another. It comes on in paroxysms; and when these are severe, the patient rolls about, or lies on his stomach with his hands clasped together beneath him, or leans the weight of his body across the back of a chair. During the intervals the patient is easy, and there is no tenderness on pressure. In exceptional cases, however, pressure increases the pain, especially when there is much flatulence.

An attack of colic may be combined with nausea and vomiting; but the skin is cool, the temperature normal, and the pulse often slower than natural.

In a case of severe abdominal pain, admitted into Guy’s Hospital one night, the fact that the temperature was two or three degrees higher than normal led to some doubt as to its real nature; yet next morning the patient was well, and a review of the symptoms seemed to prove that the attack had been one of colic.

In another case of colic, which I watched for some hours with not a little uneasiness, the skin was covered with a profuse cold sweat, and the pulse was much quickened. The expression was anxious, but there was not the peculiar sunken look of the features which belongs to the more dangerous forms of abdominal disease.—C. H. F.

Colic appears to depend on a spasmodic contraction of some part of the large intestine. If there is accumulation of gas in adjacent parts of the bowel, the attacks of spasm are attended with rumbling noises, “borborygmi,” which are audible to the patient and those about him. In such cases there may be partial distension of the abdomen; but in most cases of colic it is hollow, the muscular walls are hard, and the rectus feels drawn up into knots.

It is evident that colic is neither inflammatory nor neuralgic. It is local affection of the muscles—a kind of intestinal cramp.

A *cause* of colic which must never be forgotten is plumbism (cf. vol. i, p. 597), and it is equally important to remember that colic is a constant symptom of intestinal obstruction. When not the result of peristalsis contending against paralysis or mechanical obstruction, colic is due to the presence of indigestible food, unripe fruit, or tainted meat, sausages, or game. Ices may produce the same effect, or mushrooms or hard potatoes, carrots or turnips. Even wholesome food may excite colic by being swallowed too quickly, without due mastication and insalivation. Most purgative medicines give rise to intestinal pains, which are true colic, and due to strong peristalsis.

Colic may be associated with diarrhœa, as in irritant poisoning; but in most cases of obstinate constipation the attack does not terminate until the bowels act freely. In such cases, hard scybalous masses in the colon can often be plainly felt through the abdominal walls.

The *diagnosis* of colic always needs caution. For some of the most dangerous forms of inflammation to which the abdominal viscera are liable may for the first few hours present symptoms of colic, and a mistake may be fatal. If we define colic as a painful spasm of the bowels, it is a symptom of strangulation. But it is more convenient to limit the term to those cases in which the pain is, so far as our knowledge carries us, the substantive complaint—as we do with such terms as epilepsy, neuralgia or dyspepsia. The rule will then be that no case should be set down and treated as one of

* *Synonyms*.—*Passio colica*—Colum (Pliny)—*Enteralgia*—The gripes.—*Fr.* La colique.

colic, until we are sure that no organic disease is present. The most important characters are a retracted, hard, knotted state of the abdomen, the fact that pressure relieves the pain, and the absence of pyrexia and other indications of general disturbance. Sometimes important light is thrown on a case by the patient telling us that similar attacks have before passed off in a few hours; or that he has not long before eaten something which by previous experience may cause severe griping pain.

Colic has also to be distinguished from other functional disorders. One is gastric pain with distended stomach which has already been described (p. 330). The epigastrium is prominent in both affections, but they can generally be discriminated by gentle percussion; the note being, as a rule, more tympanitic, *i. e.* less prolonged and higher pitched, over the colon than over the stomach. Moreover, the pain has seldom exactly the same position in colic and in gastrodynia; in the former it often extends into the right hypochondrium or downwards into the left iliac fossa in the direction of the sigmoid flexure, whereas in the latter it is absent from these regions. In doubtful cases inflation of the stomach with carbonic acid gas, as described above (p. 328), would decide the question.

Treatment.—An attack of colic always ends in the recovery of the patient, and that within a few hours, or a day or two at most. It is therefore not of the greatest importance to adopt active treatment, particularly at first; and since some of the drugs which would shorten this complaint would do the greatest harm if there were really obstruction of the bowels or peritonitis, one cannot be too cautious in any doubtful case.

Colic, in fact, is the only exception to the rule which Wilks used to lay down, that whenever a pain in the abdomen is so severe as to cause the patient to send for his doctor, this of itself makes the administration of a purgative unjustifiable. The rule is of great value, and should be kept constantly before one's mind; but there are a few cases of manifest colic and nothing more, in which it ought to be infringed. The patient may clearly owe his attack to something which has disagreed with him; he may have suffered in the same way before, and have quickly got well after taking a purgative; his abdomen may be retracted and hard, the pain may be relieved by pressure, and it may be entirely paroxysmal, with complete intermissions. In such a case one is fully justified in giving him at once an ounce of castor-oil with twenty drops of laudanum, and in directing that half as much should be taken again at the end of three hours if the bowels should not have acted. Enemata of turpentine or assafoetida or oil of rue may be prescribed if there is accumulation of gas in the intestine.

Meanwhile the abdomen should be shampooed, or rubbed with a stimulating embrocation, or an india-rubber bottle filled with hot water may be laid across it, or a hot bran-bag or a large linseed poultice. A hot hip-bath sometimes gives comfort. The passage of flatus at once relieves the pain, and a free discharge from the bowels ends the paroxysm.

LEAD-COLIC.—Of all the causes of colic the most remarkable is absorption of lead into the blood.

Long before this fact was ascertained, the complaint itself was well known as an endemic colic in certain parts of England and of the Continent. It prevailed in Poitou, and was hence called *Colica pictonum*; in Devonshire, so that within the five years ending in 1767 two hundred and eighty-five cases were admitted into the Devon and Exeter Hospital; and

in the West Indies, where it received the appropriate name of the "dry belly-ache." It was formerly attributed to some local beverage; in France to wine, in Devonshire to cider, in Jamaica to rum. In the first half of the eighteenth century Huxham endeavoured to refer it to the "tartar" (*i. e.* the organic salts) contained in all these liquors.

The discovery that this form of colic is due to the action of lead was made by Sir George Baker, whose paper on the subject, read at the College of Physicians in 1767, is still quoted as a masterpiece of inductive reasoning. He showed first that in the counties of Hereford, Gloucester, and Worcester persons who drank cider did not suffer from colic; secondly, that Devonshire cider alone contained lead. Next he traced the contamination of the cider made in Devonshire to the facts that lead was used in the construction of the cider-presses, and that leaden weights were sometimes put into the casks to prevent its turning sour. Not long afterwards it was shown that preparations of lead were added to the wines made in Poitou with the same object of neutralising acidity, and that in the West Indies the stills in which rum was made had leaden worms.

An important link in the chain of evidence was the fact that both in Poitou and in Devonshire a peculiar form of paralysis affecting the upper limbs was commonly associated with the colic; for this also was traced to the poison of lead.

The recognition of the cause of the complaint soon led to the disappearance of endemic lead-colic from drinking cider, wine, or spirits; but as a sporadic disease it is still often met with.

Colic as a disease incident to certain trades was recognised by Bernardo Ramazzini, Professor of Medicine at Pavia, in his work '*De morbis artificum*,' published in the year 1700. Painters and plumbers suffer most from lead-colic. It also occurs in glassmakers, enamellers, potters (from the glaze), shotmakers, printers, and type-founders. It is not so common as it formerly was; and if the workmen were more careful, it is probable that only those employed in making white-lead, or sugar of lead, would be attacked with lead-colic.

Lead poisoning has been sometimes known to arise from use of snuff with which the red oxide, or yellow chromate, had been mixed, or which had been fastened up in lead-foil.

In white-lead factories colic is mainly due to diffusion of the plumbic carbonate as a powder throughout the workshops, so that, besides being inhaled in respiration, it collects on the workmen's hands, and is carried into the mouth with the food. It is probable that lead is never absorbed directly through the skin.

As an example of the production of colic by lead contained in food, Watson quotes the case of the troops at a station in Ceylon in 1832. More than seven tenths of those who made up the force were attacked, and the cause was found to be the presence of lead in some coarse sugar which had been distributed among the soldiers from one particular estate.

An epidemic which affected no less than 80 per cent. of the bakers and confectioners in Philadelphia in 1887, was traced by Dr D. D. Stewart of that city to their use of chrome yellow as a pigment. They got the lead, we may suppose, from their fingers, but their customers from the poisoned cakes and sweetmeats. In many of these cases there was no wrist-drop; but eclampsia, with the characteristic blue line on the gums. (See an interesting historical, as well as original, paper on the blue line, by Dr

Stewart, in the 'Trans.' of the College of Physicians of Philadelphia, 3rd series, vol. xix, 1897, p. 75.)

The most widely spread cause of plumbism is the presence of the poison in drinking-water. A well-known instance is that of the family of King Louis Philippe when living in exile at Claremont. The amount of lead in the water which they drank was seven tenths of a grain per gallon.

It is well ascertained that water containing carbonic acid and certain salts of lime has less action on metallic lead than water from which carbonates are absent or present only in small amount. Hence the distilled water sometimes used for drinking purposes on board ship is particularly liable to be impregnated with the metal; and even zinc or pewter vessels may contain enough lead to make distilled water which has stood in them injurious to health.

An inquiry held at Sheffield in 1889 showed that the water supplied to the town was capable (owing to a very slight acid reaction, and to the absence of carbonates and carbonic acid gas) of dissolving lead out of the pipes and taps through which it flowed; and numerous cases of colic, constipation, anæmia, and wrist-drop were observed in persons who showed by the characteristic blue line on the gums that they were the subjects of poisoning by lead.

There are marked individual differences in susceptibility to the influence of the poison. Watson mentions persons in whom the colic was caused by their sleeping for a night or two in a freshly painted room; and contrasts such cases with that of a painter, whose first attack occurred when he had followed his occupation for nineteen years.

How lead causes colic we do not know. It is deposited in the tissues; but the late Dr George Wilson, of Edinburgh, found less in the intestines than in several of the other organs. In the case of a woman under the writer's care, who died from plumbic eclampsia, Dr Stevenson discovered 3·5 grains of lead in the liver, which weighed 45 oz.; ·465 grain in the spleen, weighing 4 oz.; ·246 grain in a deeply pigmented part of the colon, weighing 6 oz.; ·054 grain in the heart, weighing 10 oz.; and none at all in 8 oz. of cerebral matter.

Diagnosis.—Ordinary colic and the affection caused by lead do not differ in their symptoms. The real nature of the latter is revealed by a peculiar discoloration of the gums, which is decisive of the presence of lead in the body. This was noticed by Dr. Burton of St. Thomas's Hospital, in 1840 ('Med.-Chir. Trans.' vol. xxxiii). Tanqueril had, however, in his work on Plumbism, published in 1834, described the same blue line and determined its nature and significance. The characters of this line were accurately noticed by Gull. It consists of a single row of black dots, corresponding with the vascular papillæ of the gums, and deposited in their tissue, not between them and the teeth. When complete it has a wavy course, easily detected by a lens if not by the naked eye. (See Dr. Fagge's paper in the 'Med.-Chir. Trans.' for 1876).

The late Sir John Tomes long ago proved that it was caused by the "tartar," or deposit on the teeth of calcareous salts from the saliva, or rather by animal matters which penetrate into the pores of the tartar, and of which the decomposition sets free sulphuretted hydrogen. He showed that where there is a gap between the teeth, so that tartar is absent, no "blue line" is formed. Further evidence of the same fact is afforded by cases in which

persons who keep their teeth very clean have failed to present the line, although they were undoubtedly affected by lead. In many cases the line is exceedingly partial. There may be only two or three black dots on one or more of the processes of gum projecting up between the teeth, and a lens may be necessary to be sure of them. The lead, with which the sulphuretted hydrogen combines, no doubt comes directly from the circulating blood of the vascular papillæ.

Dr Fagge had once an opportunity of examining microscopically the gums of a person who had died while affected by lead-poisoning; and he found that the colour was due to the presence of a multitude of minute granules. Some of these were aggregated together within small blood-vessels, the ramifications of which were mapped out by their presence; others were arranged in double lines which probably correspond with the exterior of other vessels. Thus it seems that the "blue line" is really due to an *excretion* of lead from the blood; and this accounts for the undoubted fact that when iodide of potassium is given to a patient suffering from the poisonous action of lead, but in whom the line happens to be ill-developed or absent, the appearance in question often becomes well marked within a few days, just as the lead can then be found in the urine, although none was being excreted previously. We have had several instances in which a blue line has thus been brought out by iodide of potassium while the patient was in the hospital for symptoms due to lead poisoning; and the late Dr. Frank Smith, of Sheffield, long ago made the same observation.

It was at one time supposed that other metals, such as copper or bismuth, might be capable of producing similar appearances. But there is no evidence that this is the case.

When the salts of lead are given medicinally in considerable doses the line often makes its appearance very quickly. Dr Burton met with instances in which it was developed within two days—one within twenty-four hours—the quantity of acetate of lead taken by each patient having then been only from fifteen to twenty-four grains. When the blood is richly impregnated with lead, and when the teeth are so neglected that plenty of sulphuretted hydrogen is provided, the line may go far beyond what has just been described. The spaces between the dots may be filled up by a uniform black discoloration, which spreads over the gum for some distance from the teeth. The inner aspect of the lips may also present a similar staining; in one patient at Guy's Hospital this was half an inch broad.

The blue line can be seen after death as easily as during the patient's life, especially round the molar teeth.

The following case may perhaps be found instructive.

The writer was asked to see a lady with obscure symptoms, and noticed that there was a blue line on the gums. This was explained by the family physician as due to shot which had been carelessly left in a decanter after it was cleaned. The sherry taken from this decanter had caused the lead-line. The symptoms, however, were none of them referable to plumbism, and it seemed likely that a considerable amount of sherry must have been taken to convey the lead. And on questioning the friends, the fact of intemperance, which had been carefully concealed, was admitted, and the symptoms were explained.

At the present day lead-colic is seldom or never fatal, at least in England; but formerly patients seem not unfrequently to have died of it. In such cases, and in those in which during an attack death occurs from some other cause, the alimentary canal is said to present no morbid change of

importance. Several *post-mortem* examinations were made by Andral and also by Mérat ('*Traité de la Colique métallique, vulgairement colique de Poitou*,' 1810). The former found the intestines free from inflammation, and neither dilated nor contracted; the latter, however, observed the colon to be contracted, and he also noticed the same thing in rabbits poisoned by lead. It is true that contraction of the large intestine is not very uncommon in persons who have died from various causes, but if constantly found in cases of plumbism it would be significant.

Prophylaxis.—The directions given to workmen whose occupations bring them into contact with lead are chiefly that they should pay great attention to personal cleanliness, that they should prevent as much as possible the poison from entering their air-passages, and above all that they should not swallow any particles with their food. They should have an outer suit of linen clothing, worn only while they are at work, and washed at least once a week. They should never take their meals in the workroom. When there is much dust, masks or respirators would probably be useful, but the men can seldom be induced to wear them.

Many years ago Liebig recommended the habitual use of "sulphuric acid lemonade"—a liquid containing a small quantity of sulphuric acid sweetened by sugar, which it was supposed would render any compounds of lead that might enter the stomach innocuous, by converting them into an insoluble sulphate; and Sir Thomas Watson stated that in some works at Birmingham the addition of the acid in question to the treacle-beer which the men drank caused the disappearance of colic. It had before prevailed to a distressing extent; afterwards not a single case occurred for fifteen months.

In the case of the water supplied to Sheffield, referred to above (p. 370), it was believed, after consultation with chemists and engineers, that the most easy, harmless, and effectual remedy was to add chalk to the water in the great "dams" or reservoirs on the surrounding moors from which the town is supplied.

Treatment.—Colic arising from lead must be treated immediately in the same way as any other form of the complaint. Sometimes there is considerable difficulty in bringing about an action of the bowels, so that two or three successive doses of castor oil with laudanum may be required, and it may even be necessary to add one or two drops of croton oil; but when once a free evacuation has occurred all the symptoms generally disappear. The patient should, however, take a course of iodide of potassium. The iodide of lead is, of course, insoluble; but the alkaline iodides possess the power of forming a soluble compound with the lead deposited in the tissues. This is absorbed again into the blood and then excreted by the kidneys, as was long ago established by the observations of Nicholson and Parkes. In a marked case at Guy's Hospital, the urine had contained no lead before the patient began to take the iodide, whereas the presence of the metal was afterwards detected without difficulty. Probably it is because the lead is apt to remain in the body all through an attack of colic, that the complaint sometimes relapses after it has been cured, when the subsequent course of elimination has been neglected.

Other effects of plumbism beside colic are a peculiar form of atrophic palsy chiefly affecting the extensors of the forearm, atrophy of the cerebral cortex, epilepsy or mania, anæmia, myalgia, menorrhagia, abortion, chronic atrophic nephritis, and saturnine gout. Some of these have been already

described (vol. i, pp. 519, 845), and others will be noticed in the chapters on Bright's disease and anæmia.

CONSTIPATION.*—This, by far the most frequent disorder of the bowels, has already been mentioned as a symptom of most fevers, occasionally even of enteric, of cerebral disease, of dyspepsia, and of colic; it is also the most obvious effect of mechanical obstruction of the bowels. But constipation is still more often met with as a primary complaint.

Pathology.—There are great differences in the frequency with which healthy persons go to stool. In some an action of the bowels occurs only at intervals of two or three days, yet they suffer no inconvenience. This is not a condition which calls for medical interference. But the natural impulse for relief is often resisted for several successive days, until it becomes weakened by repression. This may occur in schoolboys merely because they are careless, or unwilling to face the outside air, and in young women because they are too modest to be seen going to the closet. Thus the periodicity, which is so important in most of the bodily functions, is lost.

Apart from such cases as those last mentioned, in which at first the will is alone concerned, the exciting cause of constipation is either that the peristaltic action of the bowel is too slow or deficient in force, or that the fæces are too dry and hard, so that they do not readily pass down the transverse descending and sigmoid colon into the rectum. In other words, there is either paresis of the muscular coat of the intestines, or deficiency in the secretion of succus entericus. Both functions are under the influence of the intestinal nerves; but not so directly as the vascular supply of the bowels. Clinically, obstinate constipation is not only one of the most constant conditions in those suffering from mania, melancholy, and hysteria; but it is equally characteristic of organic cerebral disease, of tuberculous meningitis, and tumour of the brain, and also of the form of anæmia known as chlorosis.

The seat of constipation may be said to be in the colon. If sluggishness of peristalsis and of secretion affected the whole intestinal tract, there would be accumulation in its upper portion. Moreover we never find the small intestines loaded with stagnant contents except when they are distended by a mechanical obstruction. The accumulation in functional cases is chiefly in the sigmoid flexure. The rectum is sometimes found full of hard scybala, but this is usually in a late stage of chronic and obstinate constipation. The cæcum is often distended with liquid fæces, in cases of organic stricture lower down. Scybala† are found occasionally in the transverse colon, frequently in the descending and sigmoid portion, or in the rectum.

Of the two possible physiological causes of constipation, deficiency of secretion and deficiency of peristalsis, it is probable that the latter is the most frequent and important. Paralytic secretion in the small intestine is abundant, not deficient (p. 377, foot-note †), and the mucus secreted by the colon or rectum is often increased by the irritation of scybala.

Among the occasions of constipation may be mentioned habitual restriction to an animal diet, so that there is not the bulk of food necessary to excite healthy peristalsis.

* *Synonyms.*—Obstipatio—Alvus adstricta.—*Fr.* Constipation.—*Germ.* Hartleibigkeit.

† The Greek σκύβαλον, a word of uncertain derivation, is applied to refuse, offscourings, and dung; but in the plural is confined by modern usage to hard, dry, and separate pellets of fæcal matter.

Sedentary habits also help in preventing the bowels from acting properly. Active exercise is essential to muscular and glandular activity, and none are more frequently constipated than those who are bedridden, or prevented from walking by obesity or lameness.

When the fæces accumulate so as to cause a mechanical blocking of the colon, mere constipation passes into a more serious condition—that of obstruction of the bowels—which will be described separately.

The rectum in health is generally empty until a short time before the call for defæcation, and when a fæcal mass, even of small size, has entered this part of the bowel, it ought at once to excite sensations which lead to its expulsion. But in chronic cases of inaction of the bowels the rectum may be found blocked up by hard scybala.

Sometimes the passage into the rectum of round pellets which had been moulded in the sacculi of the colon excites a great desire to go to stool, and violent straining before they are got rid of.

When the rectum loses its natural sensitiveness, and becomes obstructed by hard, dry scybala, the lining membrane may become irritated and pour out mucus, which, mixed with liquid fæces, may pass down by the side of the retained masses, so that a condition of diarrhoea may be closely simulated.

Dr Bright was once summoned into the country, in consultation with an eminent surgeon and a general practitioner, to see a lady who had been in vain treated with astringents, for looseness of the bowels. He asked to see the evacuations, whereupon a single hard pellet of fæcal matter was shown to him, and it was at once clear that a purge alone would give relief.

Habitual constipation has a marked influence on the general health and spirits of the patient. The tongue is furred, the breath foul, and there is an unpleasant taste in the mouth. The temper becomes irritable and melancholy, and the countenance depressed and sallow.

Constipation is less common than diarrhoea in infants and young children; it is very frequent in old age, partly from the diminished reflex activity, and partly from the more sedentary habits of that period of life. The latter cause is most likely the chief cause of constipation being more common in women than in men; but another reason is in the prevalence of chlorosis in young women, for a sluggish state of the bowels is almost constant in this form of anæmia. The bowels are more apt to be confined in winter and in spring, with the wind in the east, than in summer and autumn, and in the colder regions rather than in the tropics.

We have seen that lead has a powerful paralytic influence on the colon, and insoluble carbonates of bismuth and calcium have a similar effect, but probably by checking secretion rather than peristalsis, and drugs containing tannin probably act in the same way.

Dry food tends to constipation, fruit and salads to relaxation of the bowels. Red wine is believed to have the former, and white wine the latter effect. Tea is thought to be binding and coffee relaxing, as is also hot broth and beef tea. Many persons find that beer produces constipation, along with dyspeptic symptoms which are commonly called bilious. Intemperance in liquor leads to diarrhoea, but only in the later stages when there is irritation from mucous colitis. Tobacco-smoking, by almost universal experience, antagonises constipation.

It is remarkable how much more general is a confined than a relaxed condition of the bowels throughout the world; not only among the sedentary classes, among women or old men, but in the backwoods of Canada,

in Australia, New Zealand, and the Cape, among shepherds and ploughmen, soldiers and sailors, men who ride and men who row, among ancient Romans and modern Turks, at every stage of civilisation and with every variety of food and of habits.*

Treatment.—It is no wonder that those who suffer from the discomforts of constipation are ready to take purgatives almost every day and in increasing doses. One sees patients who for years have never had a natural action of the bowels. Most quack medicines consist of aloes, senna, gamboge, or purgative salts. Each time that they are taken they cause free evacuations, but they make the intestines less capable than before of responding to natural stimulus.

A similar though not so rapid effect is produced by the habit, more common in France than here, of frequent or daily use of an enema of water, at first with the chill off, afterwards quite cold; or by using suppositories of cacao-butter, or small injections of glycerine.†

Our object should be to use as little purgative medicine as possible. Diet will do much. Many persons eat too soft and too concentrated food; less meat and made dishes, more green vegetables, salads and fruit, will often set them right. Mastication is better performed, secretion more active, and the increased amount of undigested woody fibres, spiral vessels and seeds is a healthy stimulus to peristalsis. A baked apple taken every evening is an excellent prescription in habitual constipation; and brown bread is better than white for the same reason.

Exercise out of doors is essential and, next to riding on horseback, bicycling appears to be the most useful. A glass of cold water while dressing of a morning is often effectual; but in certain cases a spoonful of olive oil acts better. In obstinate cases kneading the abdomen from right to left is a valuable mode of treatment.

A most important point is that the patient should seek relief at a regular hour each day, and allow the necessary time for the bowels to act. If the *habit* of daily action can be acquired half the battle is won.

The drug which Trousseau chiefly relied on in cases of habitual constipation was the extract of belladonna.

When we are obliged to use pills the best are the compound rhubarb, the colocynth and henbane, or the aloes and myrrh, and it is often important to give sulphate of iron along with laxatives. A combination of nux vomica, ipecacuanha, aloes and steel, with henbane or a drop or two of carminative oil is justly esteemed.

* See the graphic account by the late Capt. Burnaby of his ride through Asia Minor, and the advertisements of purgative pills which fill every newspaper at home and abroad and disfigure the face of the earth.

† The following plan of treatment was recommended by Dr Spender, of Bath, in the 'Medical Times and Gazette' for 1870. It consists in the regular administration of a pill containing from one to three grains of sulphate of iron, and about a grain of the watery extract of aloes, or the compound extract of colocynth, or the compound rhubarb pill. At first the patient should take three pills a day, one after each meal. He should be told that for two or even three days he is not to expect a motion, but that when the bowels have once acted they will be moved more frequently. And now comes the point of importance—that whenever there is a loose evacuation he should instantly decrease the number of pills which he takes. Nothing approaching to a purgative effect should ever be permitted. Very soon two pills a day are sufficient; and a fortnight later a single one perhaps produces the desired effect. Within another month he is able to do with a pill once or twice a week. The cases which Dr Spender reports are very striking, and after repeatedly putting this plan in practice I can confirm his testimony to its value.—C. H. F.

No plan, however, is always and in all cases the best, and one must adapt one's treatment more or less to the patient.

(a) In children constipation is seldom obstinate, and is usually the result of over-eating; they are, as a rule, more inclined to looseness of the bowels.

Costiveness in infants often depends on too much starchy food being given, and is best treated by adding animal broths to their diet. Lactose, maltose, and manna are each useful in such cases. Castor oil should only be used on occasion, not as an habitual aperient. Friction of the abdomen is useful, particularly when constipation is combined with colicky pains. A small piece of soap is a harmless and useful substitute for an enema.

With older children, constipation is often the mere result of carelessness, or of over-eating, and needs no drugs for its cure. Figs, prunes, and baked apples, oatmeal porridge, and green vegetables usually suffice. As an occasional laxative, castor oil is the safest and most effectual. In the less frequent chronic cases, the combination of rhubarb and magnesia (or rhubarb and soda) with an aromatic, known as Gregory's powder, the Pulv. Scammonii Comp., or the Pulv. Glycyrrhizæ Comp., are each useful, and better than saline laxatives. Cascara sagrada is also a harmless domestic medicine.

(b) When constipation is associated with anæmia and amenorrhœa in young women, we must always add steel to laxatives. Griffiths' mixture and Blaud's pills consist of sulphate of iron and carbonate of potash; pilulæ Rufi (pil. aloës c. myrrhâ) should be combined with these, or decoction of aloes may be given separately.

(c) In the most numerous cases of all, those of habitual constipation in adults of both sexes, the disorder not infrequently disappears of itself during a holiday. Sedentary occupations favour it, and exercise is often a cure; yet patented purgative pills have a great sale among American cow-boys and Australian herdsmen, who live in the open air and are always in the saddle.

The first indication is to avoid habitual medicine if possible. Exercise and diet should be tried first. Oatmeal porridge at breakfast, treacle or honey, brown bread, chocolate, and fruit—particularly figs, stewed prunes, and baked apples—are all valuable. A larger proportion should be taken of vegetable to animal food, and of liquids to solids; a tumbler of water (cold or hot) while dressing, and a baked apple for supper, is an excellent prescription for habitual costiveness. With many persons a pipe or cigarette after breakfast seems to act as an efficient stimulus to peristalsis, perhaps only by directing the thoughts and favouring regular periodicity.

When drugs are necessary, small doses of belladonna at night, and nuxvomica and aloes or rhubarb before dinner, form the best laxative pills; while aperient salines may be added to the early draught of water.

Some persons are certainly better if they take a purge once a week, once a fortnight, or (as was formerly stipulated in the indentures of London apprentices) once a month. For this purpose two compound rhubarb pills, with or without two or three grains of blue pill, may be taken overnight, or a calomel and colocynth pill may be resorted to, followed by a seidlitz powder or other saline draught the next morning.

(d) In patients who are subject to gout, and in those who habitually live too freely, a blue pill followed by a black draught is still the best occasional remedy; and in chronic cases of gouty dyspepsia with constipa-

tion, a well-diluted warm saline laxative before breakfast is often the best treatment, taken for weeks or months together. Rochelle salts, Carlsbad salts, Friedrichshall or Hungarian bitter waters (Hunyadi-Janos), are best adapted for this purpose. Or patients may journey to any of the springs which furnish laxative waters, to Epsom, Cheltenham, or Bohemia. Pure magnesian sulphate is perhaps the most efficient and least unpleasant saline aperient.

(e) The constipation of women at the climacteric period of life is generally best treated by a combination of sulphate of iron, sulphate of magnesia, and aromatic sulphuric acid, attention to diet, and substitution of cocoa or coffee for tea.

(f) Lastly, the atonic costive state of the bowels which may be called senile constipation is perhaps the only one in which the habitual use of clysters is advisable. Of drugs, belladonna is in these cases the most valuable; nux vomica comes next, and may often be combined with a small quantity of aloes as a daily pill. Many cases of what looked like malignant disease of the rectum or sigmoid flexure have been cured by the persevering use of unmedicated enemata and belladonna. Of this we had in 1887 a striking instance in Philip Ward, where a case of most obstinate constipation with tympanites and visible peristalsis was thus treated, with the best result; and the writer has seen the same result in many cases before and since.

DIARRHŒA.*—A frequent result of intestinal disorder is *diarrhœa*—the discharge of the contents of the bowel in a fluid condition, and with excessive frequency.

This depends upon one or both of two causes; an increase in the peristaltic movements of the bowels, or an increase in their secretion. It is often difficult to distinguish between these two conditions; and no doubt, in many cases, both are in action simultaneously.†

As with constipation, so with diarrhœa, the chief seat of the disorder is in the large intestine.

Subacute and acute diarrhœa.—The exciting cause of an attack of diarrhœa is sometimes mental; it attacks the boy who is in dread of being punished, or the man who is about to be examined or to lecture. A similar condition is common in young soldiers under fire. In infants it is apt to be set up by amylaceous food, which cannot be digested, because the secretions which convert starch into sugar are not formed during the first few months after birth. Certain kinds of food, high game, melons, and too much over-ripe fruit in general, are well-known causes of diarrhœa.

A frequent cause of diarrhœa, particularly occurring in the morning, is intemperance; so much so that a daily loose motion before breakfast should always suggest this possibility.

Another form of diarrhœa is that produced by chill. In some persons exactly the same exposure which they know will be followed by a "feverish cold" in the winter, will cause abdominal pain with furred tongue, thirst, and slight pyrexia, and the bowels will be freely open for a day or two.

* Διάρροια, i. e. a running through: διάρροιας ἕμα ἀκράτου ἐπιπιπτούσης, οἱ πολλοὶ ὕστερον δι' αὐτὴν ἀσθενεῖα ἀπεφθείροντο—of the Plague of Athens, Thuc., ii, 49.—*Alvus soluta*—Looseness of the guts.—*Fr.* Diarrhée.—*Germ.* Durchfall.

† See a report to the British Association by Sir Lauder Brunton and the writer, on the nervous mechanism of intestinal secretion and movement (1874-5-6); also an article by Starling and Bayliss in the 'Journ. of Physiology' (1898, vol. xxiii, p. 9), and one by Dr Edkins in Schäfer's 'Text-book of Physiology' (vol. i, p. 554).

The same effect may be produced by an east wind without a definite chill.

Diarrhœa is frequently observed in the course of acute specific fevers, independent of enteric or other kinds of specific ulceration; in variola and scarlatina, when it is always of unfavourable import; in the infective peritonitis of the puerperal state, and as a chief symptom of the gastric or abdominal form of influenza (vol. i, p. 226). So-called uræmic diarrhœa probably depends on the presence of catarrhal or ulcerative colitis (infra p. 384-5).

Impure drinking-water containing suspended vegetable matters, or contaminated with sewage, or with dissolved sewer gases, is certainly capable of causing diarrhœa.

In September, 1859, at Salford Gaol, 266 out of 466 prisoners were attacked with the complaint within four days, whereas none of the officers nor any members of their families suffered. The water which the prisoners drank was at once examined, and found to have a yellowish colour and an unpleasant taste. The cause of this was that the overflow pipe from the cistern led directly into a sewer, and conveyed a most foul stench to the cistern, which was covered in closely with boards. The water supplied to the officers, on the other hand, was clear and pleasant, although it came from the same source, and was merely stored in a separate cistern.—C. H. F.

*Epidemic infantile diarrhœa.**—It has long been known that in late summer and early autumn diarrhœa is very apt to prevail epidemically, particularly in London and other large towns. In Dr Greenhow's report to the Privy College in 1860, he stated that where it prevailed most severely, some local cause could always be traced in the air or the water. The outbreak at Salford, above mentioned, he regarded as a crucial instance, proving that such conditions are really the cause of epidemic diarrhœa.

Subsequent inquiries, however, have not borne out this explanation of epidemic diarrhœa. There is no reason to suppose that "bad smells" produce diarrhœa in themselves; they are rather valuable evidence of the possibility of contamination of food or water. The fatality of epidemic, as of sporadic, diarrhœa is almost wholly confined to children under five years, and most of the cases occur in those under two years of age but above six months.

Dr Crane, of Leicester, investigated the conditions under which 283 children had been placed who died from this cause in the summer of 1873, and found that a large majority lived in houses not in bad sanitary condition, and that 107 were wholly suckled, 98 partially suckled, and 78 fed by the bottle alone. Impurity of drinking-water clearly was not the cause of the disease in the 107.

The late Sir George Buchanan, while not denying that summer diarrhœa has associations with filth, suspected that it is really due to a specific poison. He maintained that whatever may be the heat of the weather before July, it does not cause epidemic diarrhœa. Yet the connection of the disease with autumnal heat is certain, for the mortality from this cause is much greater in hot than in cool seasons.

The late Dr Ballard ascertained that summer diarrhœa in children is associated with a rise of temperature to 56° Fahr., at a depth of four feet from the surface of the soil ('Report to Local Government Board, 1888'); and Dr Dawson Williams has adduced many facts which point to a high minimum temperature as an important element in the causation of the disease.†

* *Synonyms.*—Gastro-enteritis, muco-enteritis, epidemic enteritis, intestinal or gastro-intestinal catarrh in young children.

† See Dr Newsholme's Milroy Lectures ('Lancet,' May 9th and 16th, 1895), and Address on Epidemic Diarrhœa ('Public Health,' Dec., 1899).

Specific diarrhœa.—The human intestine is the seat of innumerable microbes of numerous species and varieties. It would appear almost hopeless to identify those which are specific and pathogenic; but this has been done, as we have seen in the case of the vibrio of cholera, the bacillus of enteric fever, and the *Bacillus coli communis*. A minute bacillus, isolated and identified by Gaertner, has been detected in cases of epidemic diarrhœa. In this country Dr Herbert Durham has traced to this *Bacillus enteritidis* (*B. sporogenes*) epidemics in lunatic asylums in more than one instance ('Brit. Med. Journ.,' Sept. 3rd, 1898).

The diarrhœa due to the causes hitherto mentioned is an acute disorder; one that may run its course in a few hours, and that does not last more than a week or ten days.

In all cases of acute diarrhœa the possibility of irritant poisoning should be borne in mind, whether accidental, suicidal, or criminal. And not less anxiously should one look for the signs of Enteric fever.

Chronic diarrhœa is scarcely ever a primary disorder. When it continues beyond a fortnight or three weeks, tuberculous disease should be suspected in children, and one of the other kinds of secondary diarrhœa mentioned below in adults.

The *anatomical changes* discovered when acute diarrhœa proves fatal are very slight. As in the case of other mucous membranes, vascular injection disappears after death, and softening, on which French pathologists formerly laid stress, is probably a cadaveric change.*

After diarrhœa has run a more chronic course, slaty or black patches may be seen after death in the mucous surface of the colon, or black dots and rings corresponding with the solitary follicles. Its coats may be thickened, and it may be lined with a viscid opaque mucus. More frequently, however, one is unable to detect any definite change in the intestine, although the diarrhœa may have been present for a long time before death. Particularly in infants, with whom diarrhœa is a frequent cause of death, one seldom finds any pathological change.

Symptoms.—The discharges of diarrhœa consist of fluid fæcal matter like that which the small intestine normally contains. It has been so rapidly hurried down by the colon that little or no absorption has taken place. It may be of a bright yellow colour, or more or less brown. In infants the evacuations of diarrhœa are often green, like spinach, a result not due to the administration of calomel, but to change of the bilirubin into biliverdin. In adults the evacuations look sometimes like almost pure bile, but the later diarrhœal discharges are pale and watery, like "rice water stools" of cholera. Under the microscope crystals of triple phosphate can often be detected in the matters voided from the bowels in all forms of diarrhœa. Mucus is sometimes found in considerable quantity, but pus is not present unless there is ulceration. Blood also can seldom or never be detected, even when advanced tuberculous ulcers exist.

Acute diarrhœa is generally accompanied by some degree of colic, by thirst and want of appetite, and by slight fulness of the abdomen. In chronic diarrhœa these symptoms are absent; the abdominal walls are sunken and retracted. In infants prolapse of the rectum is apt to occur, and the anus becomes sore and excoriated.

Mild cases, in adults, cause little disturbance of the general health;

* Sometimes after death from severe and acute diarrhœa a *pseudo-diphtheritic* state, especially marked in the valvulæ conniventes, may be found throughout a large part of the jejunum.—C. H. F.

but in infants diarrhœa may quickly weaken the patient and produce coldness of the surface, dark pigmentation and hollowness round the eyes, and depression of the fontanelles. This last is a serious symptom, and must always be borne in remembrance; it often gives a warning of danger at a comparatively early period, when the child would otherwise seem to have but little the matter with it, and when neither the pulse nor the respiration is accelerated. As already mentioned, in infants the disease is very apt to terminate fatally. Death may also occur in old people, with symptoms of collapse.

Cases in which the evacuations are profuse and watery are called "choleraic diarrhœa" or often "English cholera," and these names appear in the returns of the Registrar-General every autumn; but they are probably misapplied, since even severe cases of this kind are distinct from Asiatic or epidemic cholera (vol. i, p. 287).

Treatment.—In the slighter forms of diarrhœa it is sufficient to see that the patient is *warm*, particularly his belly and his feet; to make him lie down and keep perfectly still, and to give him arrowroot. Broths, and particularly beef-tea, are contra-indicated. A flannel binder or a hot bran-bag is often comforting.

Next to warmth rest is essential; a patient, who has been tormented by griping and frequent loose evacuations while going about, finds relief and comfort by lying in bed. The food should be restricted to rice or arrowroot, milk and lime-water, dry toast or biscuits, with a little brandy and water if there is faintness.

In many cases diarrhœa is the result of irritating food, and then a dose of castor oil is the appropriate remedy, just as an emetic may be the best cure for vomiting.

In acute diarrhœa it is sometimes desirable to begin with a dose of castor oil with a little opium; or, what is perhaps better, a scruple of Gregory's powder, or some other preparation of rhubarb. But, as a rule, the contents of the bowels are being swept away by the diarrhœa itself, and the best medicine is a carminative with a little alkali. A popular formula consists of 10 or 15 grains of carbonate of soda, twenty minims of aromatic spirits of ammonia, and an ounce of peppermint water; this may be repeated every two or three hours. Another valuable remedy is the subnitrate of bismuth in powders or lozenges, or the aromatic chalk powder.

It is not advisable to prescribe opium, or morphia, in acute diarrhœa, at least until other remedies have had a fair trial. Nor should astringents be given at the commencement of the attack; but in chronic diarrhœa they are often most valuable. Hæmatoxylum, krameria, kino, catechu, hazeline; the extract of Indian baël; the compound chalk powder and the carbonate of bismuth; alum, sulphate of copper with opium—each of these may be used with good result in suitable cases. Many physicians employ sulphuric acid as an astringent; but the few drops prescribed must be neutralised in the duodenum, and since they are always used with some preparation of tannin or of opium, no experience can be pleaded in their favour.

Sometimes each astringent seems to lose its effect after the patient has been taking it for a few days, and one is often obliged to prescribe many in turn. In such cases Dover's powder or some other form of opium is very serviceable; and it may be continued for several months without affecting the patient injuriously.

In the diarrhœa of infants and young children, Dr Eustace Smith says

that he has been disappointed in such antiseptic remedies as thymol, sulpho-carbolates, phenol, and creasote; and has found nothing so useful as grey powder or small doses of calomel.

In these patients rest in bed, warmth to the abdomen and hot water with milk or whey or barley water are indicated, and brandy is called for in severe cases. Early treatment is of the utmost importance. In some cases washing out the rectum with warm water has proved of great value.

A method of treating diarrhœa, which often succeeds in infants, consists in giving no food whatever except raw meat, finely grated into a pulp and mixed with powdered sugar or currant jelly to make it palatable. Trouseau used to prescribe this as "conservé de Damas." He relates the case of a young lady who had suffered from intractable diarrhœa for six months, and who was quickly cured by raw meat.

The prophylaxis against epidemic diarrhœa depends on giving children only boiled milk, or "humanised" milk, on avoiding stale or tainted meat and fish and bruised or unripe fruit, and on cleanliness and free ventilation in houses during hot and dry seasons.

Chronic tuberculous diarrhœa.—The earliest stage to be recognised is when opaque yellowish spots are seen beneath the mucous membrane. They doubtless are the result of caseation of the tissue of solitary follicles, or of the follicles of a Peyer's patch, the result of inflammatory changes in the lymphatic tissue, set up by the tubercle bacillus. Next, the mucous membrane covering the little yellow spots breaks through, and a small circular ulcer is formed. This soon acquires a smooth rounded edge, which is indurated, so that to the finger it feels almost like a rim of leather. The increase in size of the ulcer is chiefly in a direction transverse to the axis of the bowel. Thus its form becomes elliptical; or roughly oblong, and it may become so broad as to encircle the bowel. Its floor is formed by the muscular coat, which is thickened by inflammatory products, and may still have caseous nodules adherent to its surface. The subserous tissue and serous membrane become thickened and opaque; and these changes, with the presence of an injected zone of blood-vessels round the ulcer, enable its seat to be clearly recognised on the outer surface of the intestine. A more important character still is the presence, on the serous membrane, of miliary tubercles in clusters, or forming long ridges, which correspond with the sheaths of lymphatic vessels, or (according to Rindfleisch) with the smaller arteries.

Tuberculous ulcers are more common in the lower part of the ileum than in any other part of the intestine; they are often very numerous, and just above the ileo-cæcal valve form extensive patches of irregular shapes.

Occasionally they are confined to the upper part of the ileum, or even to the jejunum; and Perry and Shaw record eleven cases of tuberculous ulcers in the duodenum ('Guy's Hosp. Rep.,' vol. 1, p. 185).

They are frequently seen in the cæcum or the colon, with, or occasionally without, ulceration in the ileum. Tuberculous ulcers of the intestine are probably never seen in the *post-mortem* room without the lungs being likewise the seat of active tuberculous disease; and their clinical importance is generally subordinate to that of the pulmonary phthisis, to which the patient succumbs. They often accompany diarrhœa; but in many cases this and all other symptoms are wanting. Sometimes, however, before any symptom or auscultatory sign of phthisis is discoverable, the patient may

suffer for a long period from diarrhœa, due to a tuberculous affection of the intestine. This is most often seen in children.

It is said that tuberculous ulcers sometimes heal, and that their cicatrices may produce stricture of the bowel. Acute peritonitis has in a few recorded instances been caused by ulcers of this kind perforating. More commonly the affected coil of intestine becomes adherent to a neighbouring coil, and an opening forms between them; in this way a series of communications between one part of the bowel and another may be formed.

Lardaceous disease.—A once frequent cause of chronic diarrhœa was the presence of lardaceous change in the intestinal mucous membrane. This seems never to occur without other organs being affected. It may be caused either by syphilis or by protracted suppuration, of which one instance is that which accompanies chronic pulmonary phthisis. The peculiar change begins in the walls of the minute arteries, and spreads from them into the tissues around. To the naked eye the mucous membrane presents an appearance which one can more easily recognise than describe; Moxon compared it to wet wash-leather. Iodine stains it the colour of dark walnut wood. Peyer's patches are generally less affected than the rest of the mucous membrane.

Malignant diarrhœa.—Another cause of chronic diarrhœa—comparatively a rare one—is the development of a new growth in the intestinal walls. A carcinomatous ulcer sometimes gives rise to this symptom, instead of narrowing the bowel and causing obstruction; and it is clinically important to remember that cancer of the colon or rectum may produce, not obstruction, but diarrhœa, or obstruction with diarrhœa.

Syphilitic ulceration may also produce severe and prolonged diarrhœa, with or without stricture of the rectum.

Cancer of the small intestine is one of the rarest diseases. In a case reported by Dr W. H. Ransom in the 'Lancet' (November, 1890) the symptoms during life were those of chronic intractable diarrhœa.

More frequently diarrhœa is set up by a form of lympho-sarcoma, the distinctive characters of which were pointed out by Moxon. It may invade a large extent of the intestine, and completely surround it at various points, but always with the effect of making it wider than natural. It constitutes a white, soft, medullary growth, and has little or no tendency to ulcerate. A marked instance of this affection occurred in a child who died under Dr Fagge's care in the Evelina Hospital; the growth everywhere seemed to have entered the coats of the intestine along the line of its attachment to the mesentery.

*Psilosis.**—A remarkable form of diarrhœa has been described under this title; but it is known in the East, where it is endemic, by the term "Sprue." The disease is tropical in distribution, and is most often met with in China, in Ceylon, and in Further India, but has been often confounded with other forms of tropical diarrhœa. Dr Thin in a communication to the Roy. Med. and Chir. Soc. June, 1891 (vol. lxxv, p. 285),† describes and figures a re-

* *Ψίλωσις*, from *ψίλος*, bare, stripped, bald, smooth. *Synonyms.*—*Aphthæ tropicæ*—*Apepsie coloniale atrophique*—White flux—Sprue. The term "sprue" was given to the disease by Dutch physicians in Batavia. It is in use in the north of England and Scotland, as in Holland, for "sore mouth" or "aphtha."

† See also his papers in the 'Practitioner' (1883 and 1887), and the 'Brit. Med. Journ.,' June, 1890.

markable condition of the tongue, palate, and fauces, which accompanies the diarrhœa so constantly that he calls it *Psilosis linguæ et intestini*. The mucous membrane is raw, with prominent, bright red papillæ. The denudation of epithelium here is found also *post mortem* in the intestinal mucosa, which is thin and smooth, with atrophy of the secreting tubules. The stools are pale and frothy, with abundance of bacteria. There is flatulent dyspepsia, dryness of the skin, anæmia, and wasting. The disease runs a chronic and obstinate course, and if not checked, ends fatally. Beside the changes mentioned in the mouth and intestines, there is no constant anatomical lesion found after death.

Dr Fagge once made a *post-mortem* examination in the case of a gentleman who had come home from China with what is termed "white flux," or "sprue,"—constant diarrhœa with discharge of matters devoid of bile. At the time of his death the complaint had lasted some years, and the only morbid appearance discovered was extreme thinning of the intestinal tunics.

The treatment recommended by Dr Thin, Dr Manson, and others who are familiar with the disease, is a strict milk diet; or, if this fails, removal of the patient to another climate.

ENTERITIS.—This term is sometimes applied to inflammation of the bowels generally, sometimes to inflammation of the small intestine as distinguished from *colitis*, or inflammation of the large intestine. French writers use the terms *gastro-entérite* and *muco-entérite* to denote catarrhal gastro-enteritis or enteritis; but the terms are scarcely needed, for acute gastritis or gastro-enteritis is better described clinically as acute dyspepsia or acute diarrhœa, and chronic enteritis as chronic diarrhœa.

Severe enteritis occurs as a complication of mechanical obstruction of the small intestine, as in invagination and strangulation and volvulus, and will be referred to in the following chapter. The affection in cholera is probably not true inflammation.

Ulcerative enteritis has already been described when due to typhoid fever and to tubercle. Otherwise the small intestines are very little liable to serious primary diseases, and the jejunum particularly is remarkably exempt. Colic, constipation, and diarrhœa affect the large more than the small intestine. In dysentery the lower part of the ileum is sometimes affected, but only by the disease spreading from the rectum and colon.

Ulceration of the bowels as a complication of chronic Bright's disease may affect the small or large intestine, or both. It is described below as a form of colitis.

Acute catarrhal colitis.—There is a somewhat rare primary affection of the bowels, which is inflammatory in its character and acute in its course, yet distinct from ordinary acute diarrhœa on the one hand, and from dysentery or any form of ulcerative enteritis on the other.

A previously healthy man, thirty-four years old, after exposure to cold and fatigue during severe winter weather was attacked with shivering and diarrhœa, accompanied by severe abdominal pain and tenesmus. The temperature rose to 103° F., and there was complete anorexia, scanty high-coloured urine, a thickly coated tongue, pains in the back and limbs, and slight febrile delirium at night. The symptoms were more severe than those of acute catarrhal dyspepsia (p. 314), and vomiting and nausea were absent. There was no source of irritation from food or other ingesta, nor from arsenical poisoning. More-

over, after the bowels had been emptied there remained tenesmus, with passage of abundant, clear, colourless, and inodorous mucus, entirely free from any trace of faecal matter or bile, of blood, or of pus. In rather less than a week acute symptoms had subsided, but a somewhat tedious convalescence, with great muscular weakness, followed.

Such cases in a less marked degree are not very uncommon both in children and adults. Dr Hale White quotes three in 'Allbutt's System,' vol. iii, p. 940. They often appear to be due to direct chill, and though seated in the colon, correspond pathologically to acute gastric catarrh. Warmth, fomentations, diluents, and opiates seem to be the treatment indicated.

Secondary catarrhal colitis is a frequent complication of Bright's disease, and of enteric fever. A case of the former association occurred under the writer in 1888, in a man aged 59. He passed large quantities of mucus, and, *post mortem*, catarrh without ulceration of the colon was found.

Follicular enteritis has been described as a separate affection, on account of the solitary follicles of the colon being primarily affected.

Ulcerative colitis.—A more common disorder, chiefly met with among women and children, is ulceration of the colon and rectum, running a sub-acute or chronic course, and resembling dysentery in the passage of blood and purulent or muco-purulent matter with the stools, in the tenesmus and most other symptoms. But it does not appear to depend on any of the causes of true dysentery, and does not occur epidemically. Accordingly Dr Eustace Smith and Dr Goodhart admit as dysentery in children only chronic cases which have been acquired in the tropics. It is almost the only disease in which unmixed pus is passed without an abscess having opened into the gut.

Ulcerative colitis is a serious and not infrequent complication of Bright's disease, like the catarrhal form with which it is closely allied in symptoms and pathology. The writer has seen both kinds, most often in chronic cases, with little dropsy, hard arteries, hypertrophied heart, and contracted kidneys; but ulceration also occurs in cases of tubal nephritis with a large white kidney. A case was recorded by Dr Bright himself in the 'Guy's Hosp. Reports' for 1838. Dr Dickenson drew attention to this condition in the 'Croonian Lectures' of 1876, and again in the 'Med.-Chir. Trans.' for 1894 (p. 161 of his 'Occasional Papers').

It affects the caput cæcum coli and the rest of the colon, and also the lower part of the ileum. The twenty cases recorded by Dr Dickenson occurred in men and women, and at almost all ages, from a case in a boy of 14 to one in a man of 66. In all but three there was hypertrophy of the left ventricle, and in six cases there was albuminuric retinitis or retinal hæmorrhage. In twenty cases of ulcerative colitis taken from the Guy's Hospital Museum, thirteen occurred in men and seven in women. The ages varied from 9 years to 38. Six of these cases occurred in the course of Bright's disease, two preceded by pneumonia, one complicated by purpura; two followed pyæmia; one was probably tropical dysentery; five occurred in patients slowly dying from paraplegia, from cancer, from cystitis, or hip disease; and the remaining three were "idiopathic." Peritonitis is a frequent result, sometimes from perforation of the ulcer and extravasation, but more often with no local cause. But pleura, pericardium, and peritoneum are all disposed to inflame on the least provocation, when it occurs in a case of Bright's disease.

The symptoms of ulcerative colitis are often most obscure. Pain is

constant, and local tenderness nearly so; there is diarrhoea with passage of blood, mucus, and pus, and sometimes tympanites. Fever, vomiting, and furred tongue are, as a rule, absent. There is almost always anæmia with loss of flesh.

In a man of forty-two, under the writer's care in John Ward (November, 1895), who had repeatedly suffered from similar attacks, the above symptoms were present with enormous distension of the colon, which was at last punctured when the breathing became dangerously impeded, with decided relief. The previous attacks were supposed to be typhilitis, but he was sent into hospital for "enteric fever with typical typhoid stools and enlarged spleen." There was, however, no exanthem, no delirium, and the course of temperature was not that of enteric. The diarrhoea was not profuse, and no blood or pus was ever passed. After three weeks' illness he died insensible and collapsed. The small intestine and cæcum with its appendix were healthy. The transverse colon was the seat of large sloughing ulcers, and the descending and sigmoid portions contained numerous smaller follicular ulcers. There was no perforation, but general plastic peritonitis. The liver was fatty; the stomach, kidneys, and thoracic viscera normal.

There remain cases of severe and even fatal ulcerative colitis in adults which are very difficult to explain. When cases dependent on distension (*infra*, p. 428-9), on typhoid fever, tubercle, Bright's disease, dysentery, or syphilis, have all been excluded, a certain number still remain, which must at present be classified rather by their anatomy than by their origin and pathology. Bristowe met with this condition in cases of pneumonia, Wilks in cases of diabetes. Dr Hale White showed a good specimen at the Pathological Society on December 6th, 1887; see also his interesting commentary on twenty-nine cases in the 'Guy's Hospital Reports' for 1888 (p. 131), and in 'Allbutt's System' (vol. iii, p. 950).

Membranous colitis, sometimes called "Pericolitis sinistra," plastic, diphtheritic or croupous, and desquamative colitis, is a very rare affection. It is not to be distinguished during life from sporadic cases of ulcerative colitis, unless casts of the intestine should be voided. Excessive pain and tenesmus attend the act of defæcation in some cases, but certainly not in all. Dr Goodhart narrates a marked case of this condition in a girl of eleven. There was high temperature, a purpuric rash, and excessive anæmia. After death the rectum and lower parts of the colon were found covered with a thick adherent membrane.

Dr Light has given a good account of this disease with references in the 'Practitioner' for 1893, and Dr Hale White in 1897 (l. c., vol. iii, 943). The distress is sometimes so great that colotomy has been performed, and relief has followed. In one obstinate case the writer advised this treatment, and the result was satisfactory.*

The true pathology of the *intestinal casts* which are sometimes met with conditions. Sometimes they are associated not with diarrhoea or dysentery is very obscure, and probably they are not all produced under the same enteric symptoms, but with constipation. In 1857 Mr Hutchinson showed several specimens of this kind at one of the meetings of the Pathological Society ('Path. Trans.,' vol. ix, p. 188); they were cylinders several feet long, with walls from one eighth to a quarter of an inch, yellowish brown, transparent, and gelatinous. Under a microscope their surface showed a regular arrangement of round or oval pits, which had evidently corresponded with the mouths of the tubular glands of the intestine. Their substance was almost structureless, but embedded in it were large numbers of epithelial cells. When they had been retained in the rectum, the casts were apt to be changed into hard, white, round masses, about the size of nutmegs.

* W. H. White and Golding-Bird in 'Clin. Trans.,' 1896, and *ibid.*, 1899.

More recently Dr Goodhart exhibited some specimens to the same Society ('Path. Trans.,' vol. xxiii, p. 98) which were almost exactly similar, except that they were solid. Many of them passed at their ends into a clear colourless jelly. Both in Mr Hutchinson's case and in Dr Goodhart's there was much complaint of abdominal pain. It does not appear that medicinal or other treatment led to any definite good result.

The structure of the membrane, which may be in shreds, or in broad, curled-up laminae, or in complete casts, is that of a more or less gelatinous substance in layers, either white or discoloured, with no microscopical structure beyond a few scattered opaque bodies, probably degenerated epithelium. Chemically these casts appear to consist not of fibrin, but of an albuminous substance.

Sloughing colitis.—Occasionally, as the result of severe ulcerative colitis, fragments of mucous membrane are passed. These are not mere casts, like those just mentioned, nor dysenteric sloughs, nor portions of the entire gut thrown off, as in cases of invagination.

A patient under the writer's care in December, 1887, suffered from enlarged liver with jaundice and pyrexia; and afterwards passed on several occasions fragments of mucous membrane, apparently from the colon, one of which formed a complete ring, and clearly showed the tubules of Lieberkühn. There was very little hæmorrhage, and no other distinctive character of dysentery. After being extremely ill, the patient, a man of about forty, seemed to be slowly recovering, when fresh hepatic symptoms appeared, and he died of abscess of the liver. At the autopsy was found extensive colitis, which appeared to be on the way to recovery.

Dilatation of the colon.—We frequently meet with a distended and hypertrophied colon as the result of organic or temporary obstruction, the mechanism being the same we have described in the case of the œsophagus and the stomach. But in some rare and remarkable cases the dilatation and hypertrophy of the colon are (so far as is yet ascertained) primary. Peacock ('Path. Trans.,' xxxiii, p. 18), Bristowe and Gee ('St Barth. Reports,' vol. xx), Rolleston and Haward ('Clin. Trans.,' 1895), Osler, Herringham, and Bruce Clarke ('Brit. Med. Journ.,' 1894), have described cases. The coats are greatly thickened, as well as distended. The most frequent seat of this condition is the sigmoid flexure.

DYSENTERY.*—This disease is now happily rare in England. The Greek word is classical: a passage in Herodotus clearly alludes to the dysentery of camps:—(of the army of Xerxes during its retreat from the invasion of Greece) ἐπιλαβὼν δὲ λοιμὸς τε τὸν στρατὸν καὶ δυσεντερίη κατ' ὁδὸν διέφθειρε (lib. vii, cap. 115); and the term is common in Hippocrates, who correctly refers the symptoms to ulceration of the intestine. It is also used by St Luke, himself a physician, in its natural connection with intermittent fever: πυρετοῖς καὶ δυσεντερίᾳ συνεχόμενον (Acts xxviii, 8). For dysentery is closely allied to malarial disease in its ætiology and distribution; and, like ague, has been gradually banished from the Lincolnshire and Cambridge Fens, the valley of the Thames, Essex, Kent, and London itself, where, in the seventeenth century, it was still endemic and often fatal. Dysentery is not uncommon in Spain, Italy, and Greece, in Hungary and in the Valley of the Danube; also in Syria, in almost the whole of Africa, in

* *Synonyms.*—Bloody flux.—*Fr.* Dysentérie—Flux de sang.—*Germ.* Rothe Ruhr.

Ceylon, Java and Madagascar, and in the West Indies and Southern States of America. It is also one of the most important endemic diseases in India and China. Though most severe in the tropics, it is as well known in warm temperate climates at the present day as in the time of Hippocrates, and is still most apt to break out in camps and in prisons.

Dysentery is still, after Syphilis, the most serious, by the mortality and disablement it causes, of the diseases of our troops in India and of our sailors on the China and West African stations.

Although so ancient and familiar a disease, it is difficult to define it accurately. The word, like all ancient medical terms is not pathological but clinical, and refers to symptoms, not to anatomy or causation. It originally meant no more than "Bowel-complaint," and was applied to any severe form of diarrhœa in which "blood and slime" were passed with the motions. Clinically we must still define dysentery as present when diarrhœa is accompanied by a frequent desire to go to stool, by passage of mucus, pus, and blood, with or without fæcal matter, by tenesmus with griping pain in the course of the colon, and by a feeling of heat and weight in the rectum.

As a matter of experience, when these symptoms have occurred, inspection of the body after death shows the presence of ulceration of the rectum, colon, and cæcum, with perhaps the last few feet of the ileum.

Pathologically, we shall see that it is impossible to define dysentery in terms of its essential nature and origin. It occurs under varied conditions, as an epidemic, an endemic, and in sporadic cases, under varied conditions of climate and water-supply. Modern research has brought forward many claimants among the microphytes found in the diseased tissues to be the pathogenic organism, but none has been generally admitted. Moreover a microzoon has been discovered in some of the most marked forms of tropical dysentery which has led to such cases being separated from others allied or identical in their symptoms and anatomy.

At present it appears the best course to describe first the symptoms and clinical course of the disease, afterwards the morbid changes found after death, and then to discuss its pathology and possible origin from more than one cause.

Symptoms.—If we recognise a period of incubation in dysentery, it is a short one, often not above a few hours, and seldom as long as three, four, or five days after exposure to its presumed cause. Homan and Hertwig state that in Norway in 1850 the period of incubation varied from two to eleven days.

Before the characteristic symptoms of dysentery develop themselves, there is generally a period in which the patient suffers from apparently ordinary diarrhœa, with more or less griping pain in the abdomen. This may last from three to five days, or even a fortnight. He then becomes worse, and complains of greatly increased malaise and weakness. The abdominal pain is more severe and paroxysmal, the desire to go to stool becomes more and more frequent, and, instead of abundant liquid fæces, he begins to pass only small quantities at a time, with violent straining, and burning pain in the rectum. He is then said to suffer from tenesmus, although some writers mean by it the peculiar sensation that "something wants to come away," others the straining, and others the burning pain.*

* Tenesmus (τεινεσμός, from τείνω): "Est autem affectus hic continua et implacabilis desidendi cupiditas" (Paulus Ægineta)—Quod τεινεσμόν Græci vocant: in hoc frequens desidendi cupiditas est, æque dolor ubi aliquid excernitur" (Cels., lib. iv, cap. 18).

Beside faecal matter, blood and mucus, another dysenteric product is pus, which may occasionally be discharged pure and odourless, just as if it had come from an ordinary abscess, but which is much more commonly mixed with fluid faecal matter and blood. In some cases, again, a substance is voided which looks like frog's spawn or boiled sago. This consists of rounded bodies which were once thought to be sloughs derived from the solitary follicles. But as Heubner says, they are too large for this, and they consist of mucus. Lastly, the discharge may be a brownish-red or blackish fluid, of a horribly offensive odour; this is an indication that sloughing is going on in the intestine. It is to be noted in all but the earliest stages of dysentery the matters passed from the bowel have a foetid odour, which Parkes regarded as peculiar to the disease.

The abdomen is not at first distended, but it may become so as the disease advances. Tenderness may be altogether absent, or more often pressure over the large intestine may give more or less pain.

Another local symptom sometimes present in severe dysentery is strangury, for which it may be necessary to use a catheter.

The most marked general symptom is the great prostration. The patient sometimes faints while he is at stool. Anæmia is very rapidly produced, and the face assumes a pale, waxy look.

The febrile disturbance is generally slight. Even in severe cases the temperature may be normal or below normal, and seldom rises above 100° or 101° . The pulse is not much quickened, except in certain epidemics formerly known as "inflammatory dysentery." The patient complains of thirst, and although his appetite is not always lost, griping pain is brought on by food, so that he is unwilling to take any but the blandest nourishment. Cold liquids frequently have the same effect, and therefore most patients prefer to drink lukewarm water.

Clinical progress, complications, and events.—In favourable cases, with rest and suitable diet, the symptoms quickly begin to improve. First the pain and tenesmus pass off, and then some of the evacuations begin to contain faecal matter; but formed faeces may still for a time be passed alternately with blood and mucus.

An affection which resembles acute rheumatism may attack several joints at the same time. Parotitis is also said sometimes to occur during convalescence from dysentery.

In cases which terminate fatally the prostration passes into collapse. The features become sunken, while the body is covered with cold sweat, and exhales a foetid odour. The tongue and lips are covered with sordes, hiccup sets in, and a painful sense of constriction of the epigastrium is complained of. The faeces are passed involuntarily, the anus becomes excoriated, and the lower part of the rectum is often prolapsed. The urine may be suppressed, and bedsores are formed, if the patient lives long enough. Consciousness is often retained to the end; but in some cases the mind wanders, and during the last few hours all pain may cease, so that the patient fancies he is doing well. Shortly before death the temperature of the body often rises considerably.

In the later stages of dysentery perforation of the intestines sometimes occurs, setting up fatal peritonitis.

In one case this was observed in a patient at Guy's Hospital. He was in a surgical ward for disease of the knee, when in the month of August he was attacked with "severe diarrhoea." This continued, and he died at the end of a fortnight. The large intestine was ulcerated and sloughing in its whole length, and at one spot the transverse colon was

perforated.—C. H. F. (Compare, however, the case mentioned under ulcerative colitis, *supra*, p. 385.)

In three other cases of sporadic dysentery we have found acute peritonitis without any perforation being discovered to account for it.

Chronic dysentery.—In many cases, as already mentioned, acute dysentery passes into a chronic form of the disease, which may last for months, or even years. It is probably always preceded by an acute stage. The discharges still have to some extent the peculiar odour, and they are for the most part liquid, but they vary in character from day to day. Sometimes natural fæcal matter is voided, at other times only a blood-stained mucus. Instead of gaining flesh the patient becomes more and more emaciated. The tongue is red and glazed, and the appetite fails. Abscess of the liver not infrequently forms in these cases; or death may arise from pneumonia, phthisis, Bright's disease, or lardaceous disease of the viscera; or, again, a fæcal abscess may make its appearance in the iliac fossa or elsewhere. Peritonitis from perforation may also occur, even at this late period.

Sequelæ.—Beside the multiple synovitis, miscalled rheumatism, which occasionally follows dysentery, still more rarely paraplegia from peripheral neuritis has been observed by Weir Mitchell. Chronic dysentery may lead to contraction and partial obstruction of the bowel, or to long-continued ill-health and an irritable state of the bowels with anatomical change. But the most important and dangerous result is portal pyæmia with abscess of the liver.

Anatomy.—An elaborate account of the morbid anatomy of dysentery, illustrated by photographs and coloured plates, is contained in the valuable monograph on the diarrhœa and dysentery during the American civil war in 1861—1866, drawn up by Surgeon-Major Woodward, U.S.A. Among early accounts of autopsies may be mentioned one by Hewson,* and two others (likewise communicated to Sir Geo. Baker) by Dr Wollaston.

In some instances the whole gut, from the rectum to the cæcum, shows morbid changes of the same kind and in the same stage. But in others disease is more advanced or more severe in one part than in another. Commonly the rectum is the seat of the most intense changes, and these gradually diminish towards the cæcum; but sometimes the reverse is the case, as was noted by Sydenham. The flexures often suffer more than the intervening parts of the bowel.

The appearances presented by the affected parts in dysentery are exceedingly varied, but most writers are now agreed that the processes concerned in their production may be reduced to two, to which Virchow gave the names "catarrhal" and "diphtheritic." Excellent descriptions of both forms of the disease have been given by Heubner, of Leipzig, by Woodward, by Sir Joseph Fayrer, and by Dr Maclean in Reynolds,† and Dr Davidson in Allbutt's 'System of Medicine.'

In the *catarrhal form*‡ the mucous membrane at first shows lines and

* Quoted in Sir Geo. Baker's treatise 'De Dysenteria Londinensi' (1771), who introduces the account as follows: "Iniente mense Octobri (1862) cadaver hominis dysenteria extincti secuit vir et ingenia et modestia singulari, Gul. Hewson, celeberrimo Huntero nostro in re anatomica socius atque adjutor" ('Opusc. Med.,' p. 69).

† The article referred to is in the first volume. A much shorter and chiefly anatomical account, by Dr J. W. Begbie, appears in the third volume among "Local Diseases."

‡ *Synonyms*.—White dysentery.—*Germ.* Katarrhalische Ruhr.—'Πεῦμα γαστρὸς (Galen).—Intestinorum rheumatismus (Cælius Aurelianus)—Coryza ventris—Tormina—Dysenteria fiens (Sennert), a translation of Galen's Δυσεντερία γινόμενη.

patches of a dark red colour, with points which are almost black. The summits of any ridges or folds projecting into the interior of the bowel are more injected than other parts. The mucous membrane is lined with a rather thick layer of mucus streaked with blood, and is much swollen, as well as the submucous tissue. In the earliest stage of the disease all that the microscope reveals is a dilatation of the minute blood-vessels, which are gorged with blood. Soon, however, inflammatory products are poured out. The mucous membrane becomes now still more œdematous and bulky; it is less uniformly reddened; the solitary follicles are enlarged, and appear as white points surrounded by red rings. The submucous tissue is increased from three to five times in thickness; and even the muscular coat is swollen.

Under the microscope all the tissues are seen to be infiltrated with pus-cells, which are also present in large numbers in the mucus lining the intestine. In the submucous tissue the leucocytes are most numerous in the spaces round the blood-vessels. The solitary follicles are markedly increased in size; the lymph sinus which surrounds each of them is wide, but does not contain pus-cells; leucocytes, however, are collected in large numbers under the thin mucous membrane which covers the follicles, and would evidently soon have ruptured.

After a time the mucosa softens down with the increasing infiltration of corpuscles, and ulcers are formed. The roofs of the solitary follicles give way, and minute round holes are produced, each of which leads into a small cavity having in its interior the substance of the follicle, which forms a small slough. The destruction of the mucous membrane, however, is by no means confined to the follicles. It also takes place between them, so that for a time each orifice is surrounded by a little ring, which appears to be raised, and looks like a deposit upon the surface instead of being a remnant of the original tissue. Even when the ulcers have increased in size, and run together so as to form large patches, there remain irregular islands of still undestroyed mucous membrane, which are of a bluish-red colour and covered with grey or greenish layers of tough mucus. The ulcerated surfaces themselves have a yellow or yellowish-red colour; their floor is formed by the submucous tissue.

The *sloughing or necrotic* form of dysentery* was called by Virchow "diphtheritic"—an unfortunate term, because dysentery is totally unconnected either clinically or pathologically with the disease known as diphtheria, and because there is no "false membrane" (*diphthera*) present in most cases of dysentery.†

The whole thickness of the intestine is involved from the first. Even the serous surface is injected of a dark bluish-red colour. The bowels feel hard as well as massive. They contain a thin reddish fluid, or, in some parts, a little fæcal matter. Their mucous membrane is of a greyish-red colour, and here and there exhibits what looks like a raised deposit on its surface. In its earliest stage only on the summits of the ridges the seeming deposit forms transverse lines in the ileum, the lower part of which is commonly affected; in the cæcum it becomes more exten-

* *Synonyms*.—Red dysentery.—*Germ.* Diphtheritische Ruhr.—Dysenteria facta (Sennert), a translation of Δυσεντερία ἡδὲ γεγεννημένη.

† In Virchow's sense "diphtheritic" inflammation of a mucous membrane or of the skin does not imply the presence of a false membrane, but a putrid, destructive, more or less necrotic inflammation. See the exposition of this point in the chapter on Inflammation (vol. i, p. 53), and in that on Diphtheria (vol. i, p. 316).

sive; and in the colon it occurs in large patches, or may occupy the whole surface; being, however, broken by deep groves or fissures. The parts affected look dry and granular, and to the touch feel rough and hard. Their colour varies to some extent with that of the intestinal contents, which may stain them yellowish, greenish, dark red, or even black. On making a section through the intestine one finds that it is enormously thickened: the muscular layer is much thicker than natural, and folded in and out, but the most striking change is in the internal coat: instead of the dry rough substance above described being a deposition on the surface of the mucous membrane, it is now seen to take the place of that structure, and perhaps of the submucous tissue also. The whole thickness of the intestinal wall down to the muscular coat may thus become a tough, homogeneous, yellowish-red layer, which offers considerable resistance to the knife, and in which the natural strata can no longer be recognised. Even under the microscope one can hardly make out the original elements of the tissue in the mass of extravasated blood, of amorphous fibrinous exudation, and of pus-cells.

By appropriate staining, numerous micrococci and rod-shaped organisms appear, belonging to several bacterial species.

The *Bacillus coli communis* is present in abundance, as are vibriones, *Staphylococcus pyogenes albus* and *aureus*, and streptococci. Calmette and other French observers who observed the disease in Cochin China, believe that the *Bacillus pyocyaneus* is the most constant, and perhaps the pathogenic, organism of tropical dysentery.

Since the discovery that malarial fevers depend not on the presence of microphytes, but of microzoa in the blood, these amœbæ have been sought and found in the stools of persons suffering from dysentery. This is also true of some cases in Europe, but the amœbæ are far from constant in cases of tropical dysentery.

It is evident that the apparent "deposit" or "membrane" in the so-called "diphtheritic" form of dysentery is really formed by the exudation of fibrin and the extravasation of blood into the tissues themselves, not upon the mucous surface. The mucosa as well as the epithelium is itself destroyed. This process can have but one termination—the death of the affected structures. Accordingly, whenever there has been time for the occurrence of further changes, eschars are found; and at a still later period these break down into shreds or detritus and are cast off, exposing deep and ragged ulcers of dark green or brown colour.

The changes presented by the intestine in the two forms of dysentery are so different in appearance that one would at first sight be disposed to regard them as belonging to different diseases. It is, however, certain that they merely indicate different degrees of severity in the morbid process. For they are very frequently found side by side in the same intestine, the more intense inflammation being present in those parts (commonly the rectum or cæcum) which were earliest attacked. And this being so, the fact that the solitary follicles appear not to be specially affected in even the earliest stage of the more severe form affords strong evidence that they are not the seat of any primary or special change in the catarrhal form of dysentery.

Later anatomical course.—Sometimes abscesses form in the submucous tissue, and burrow, so that undestroyed parts of the mucous membrane are detached in the form of bridges, and when pressure is made exude pus at

several points. The inflammation may extend through the muscular coat, and penetrate the serous membrane, with consequent peritonitis; or the subperitoneal connective tissue may be reached by the ulcer, and a fæcal abscess formed. In one case at Guy's Hospital such an abscess formed a large tumour filling the left side of the abdomen, extending into the psoas muscle and the spleen (which was sloughing), and denuding the ilium of its periosteum over a considerable space.

It is probable that the most intense form of dysentery, in which the whole large intestine and the last foot of the ileum are involved, is always a fatal disease; but from less extensive necrosis recovery may take place. In the catarrhal form, the inflammation probably often subsides before any breach of surface has occurred. When ulceration takes place, and the ulcers subsequently heal, a thin membrane is formed, which is at first depressed below the level of the parts unaffected; but this difference becomes less obvious as time goes on.

In the necrotic form of dysentery the ulcers left by the separation of the sloughs become covered with granulations; their undermined edges adhere to the submucous tissue, and thickened and irregular cicatrices gradually develop themselves. The cicatrices which follow dysentery are always of a dark grey or black colour, which probably results from a chemical reaction between the iron of blood extravasated during the course of the disease, and the sulphuretted hydrogen contained in the interior of the bowel.

When the disease passes into a chronic form, some ulcers remain unhealed, or fresh ones form in succession. But, according to Maclean, it is a mistake to suppose that ulcers must remain unhealed so long as symptoms of dysentery persist. Cases are often observed in which not a single breach of surface is discoverable after death. Numerous black cicatrices may be seen, but the essential pathological change is an atrophy of the coats of the bowel, the secreting and lymphatic tissues having disappeared, and the wall being so attenuated as to be transparent.*

Sporadic, endemic, and epidemic dysentery.—Probably there is no part of the world in which dysentery does not sometimes occur sporadically, but in London it is now decidedly a rare disease. Formerly it was common there and in many other parts of England, and has become rare along with ague and other results of malaria. Cardinal Wolsey is believed to have died of English dysentery.

In 1762 it occurred epidemically in London, and was described by Sir George Baker. The account of it by the late Dr Baly, as a local disorder at Millbank Prison, in 1849, is, we may hope, the last. At present most cases met with by London physicians are imported, and are nearly confined to soldiers and sailors.

There are, however, countries in which it is exceedingly prevalent, so that it may be said to be endemic there (p. 386).

Dysentery sometimes affects large numbers of persons as an epidemic, and then is apt to assume a severe type. Heubner, indeed, doubted whether "diphtheritic" (*i. e.* necrotic) dysentery ever occurs sporadically. But among the scattered cases of acute and rapidly fatal dysentery that in the course of twenty years have occurred at Guy's Hospital, there have been several in which the inflammation showed the most marked pseudo-diphtheritic or necrotic character. It is therefore evident that, so far as

* "Muco penitus absterso, nulla membrana superficiem quæ tegetet inventa est" (Hewson).

concerns the anatomical changes in the intestine, no absolute distinction exists between sporadic and epidemic cases.

As a rule, however, the disease is much milder when sporadic than when endemic or epidemic.

The relation between acute colitis occurring in asylums and true dysentery is very important pathologically and also practically. In a report to the Asylums Committee of the London County Council Drs Mott and Durham maintain that "asylum colitis" is identical with sporadic cases or restricted epidemic of dysentery, *i. e.* of the same disease which was once endemic in this country and in Western Europe, and is still common in the tropics and in the Mediterranean area, characterised by the presence of blood and mucus or pus in the stools. They regard it as infectious, but find no sufficient evidence of its being conveyed by air or water or milk. It appears to be connected with such general conditions as overcrowding and want of cleanliness, both difficult to avoid in large lunatic asylums. ('Archives of Neurology,' 1899, 'Brit. Med. Journal' of the same year, April 6, pp. 838, 846).

Ætiology.—Sporadic dysentery is commonly attributed to irritating articles of diet, such as unripe fruit, decomposing meat, or bad water.*

Virchow remarked that the cæcum and the flexures of the colon, which are particularly liable to be affected by the disease, are also especially apt to become loaded with fæcal masses; and it is evident that if there is any irritant substance among the intestinal contents, its action must be favoured by their retention as the result of imperfect peristalsis. As Annesley long ago observed, in India the disease often begins with the characteristic signs of morbid accumulation in the large bowel; and certain transverse ulcers in the colon, which have been known to perforate the bowel or to cause a fæcal abscess, are due to irritation from the fæces; indeed, they are sometimes described as stercoral ulcers.

Another cause to which sporadic dysentery has been attributed is cold. The cases of catarrhal colitis with hæmorrhage, which are not uncommon in women and children and are often called dysentery, can quite as often be traced to exposure to cold as to indiscretions in diet (p. 384).

Over-indulgence in fruit, and particularly certain kinds of fruit, as melons and gourds, preceding constipation and the chills of exposure after exertion in a hot climate, are only contributory causes of dysentery, as they are of non-specific diarrhœa.

Of the true causes of endemic and epidemic dysentery little is certainly known. On each side of the equator, to about 35° or 40° of south or north latitude, there are in all parts of the globe territories in which it prevails, but it is not endemic in every country with a hot climate. Hirsch mentions Gujerat in India (particularly the peninsula of Kathiawar), and Senegal in Africa, as regions in which the heat is intense, but in which there is no dysentery. So, again, Singapore is said to be free from the disease, which yet exists in other parts of the peninsula of Malacca.

In temperate climates epidemic dysentery occurs, at the present day,

* Trousseau urged that ingesta are not likely to leave the small intestine unaffected and to exert an irritant action first upon the cæcum and colon. But we know that this occurs in cases of poisoning by bichloride of mercury, in which inflammation and even ulceration of the cæcum and colon have repeatedly been observed though the small bowel has escaped entirely.—C. H. F.

chiefly in camps and armies. It was very fatal in 1854 among the British troops engaged in the Crimean war, as it had been among their forefathers in 1415 before the battle of Agincourt. It raged terribly in the armies of the United States in 1862—1865, and in the camps of the Northern prisoners. It was again prevalent in 1870, during the campaign between France and Germany, particularly among the miserable fugitives who sought refuge in Switzerland after the dispersal of Bourbaki's army.

Even in time of peace the great cities of Europe were formerly liable to epidemic dysentery, and Paris suffered severely from it as lately as 1859, after having been free for a hundred years. In London, in the seventeenth century, it is believed to have caused from 1000 to 4000 deaths annually; in the following century it gradually disappeared, the last general epidemic being that of 1762. In Millbank prison, however, small outbreaks of the disease were of frequent occurrence until recently.

Only fifteen years ago there was a severe local epidemic of dysentery in a lunatic asylum in Ireland. At a meeting of the Royal Irish Academy of Medicine (December 2nd, 1887), Dr Conolly Norman reported that of the 1100 inmates 120 were attacked, and 22 died. In two of the latter cases there was perforation of the colon, and in a third multiple abscesses were found in the liver.

In India the rainy season is the period of the year at which dysentery is most apt to prevail; in temperate climates and in the tropics the autumn is the season at which the disease is most apt to break out.

Dysentery occurs in persons of all ages. In this country the catarrhal form, or a closely related disorder, is not uncommon in infants who are brought up by hand. Tropical and epidemic dysentery attacks especially those whose health is impaired by intemperance.

It has long been known that even in temperate climates, and still more in the tropics, the countries in which dysentery prevails are those in which ague is common. Indeed, the two diseases frequently occur together, in the same patient and at the same time; and Aitken remarks that if a boat's crew be sent ashore in a tropical climate, and exposed to paludal miasm, of the men returning on board some will probably be seized with dysentery and others with remittent fever. Moreover the gradual extinction of endemic dysentery within the last two centuries in England has coincided with a corresponding decrease or disappearance of ague throughout the country. The discovery of amoebic organisms in the evacuations and the mucous membrane of cases of dysentery has brought this relation with ague into fresh importance (vol. i, p. 396).

At one time it was believed that dysentery, like intermittent fever, was due to a telluric poison. Dr Baly came to the conclusion that the epidemics of dysentery at Millbank prison were due to a miasm arising from the soil; and Dr Maclean attributed the poison to the decomposition of organic matter in the ground. But in the year 1854 the prisoners at Millbank ceased to be liable to dysentery; and during the next eighteen years (up to 1872) one death only occurred from that disease or from diarrhœa. Now, as Mr de Renzy showed, one, and only one, change coincided in time with this improvement in the sanitary state of the prison. Previously the water which the convicts drank was taken directly from the Thames. But on August 10th, 1854, the artesian well in Trafalgar Square was made the sole source of water-supply to the prison. The change was effected in the middle of a cholera epidemic; six days afterwards the disease suddenly ceased. Enteric fever, too, no longer attacked the convicts, and the death-rate declined. It seems impossible to avoid the conclusion that one cause of dysentery in Millbank prison was the Thames water, or the sewage it contained.

Another series of epidemics of dysentery occurred in the Cumberland and Westmoreland Asylum. In 1864, chiefly between May and August, twenty-six persons were attacked

with dysentery, and in March, 1865, five others. Dr Clouston, the medical superintendent, thought that the disease might be connected with the distribution of the sewage of the Asylum, which was allowed to flow over a field about 300 yards distant. In August, 1864, he had the sewage carried away in a covered drain to a distance; from that time no fresh cases of dysentery occurred. It was found that within a week before the day on which each patient fell ill there had always been either hot sultry evenings with no wind in the night, or northerly winds which blew from the direction of the irrigated field. Male and female patients, too, were attacked at different times, according as the exact direction of the wind was such as to carry the smell of the sewage to one or to the other of the parts of the Asylum severally occupied by men or women.

The probability that this was a real cause of the dysentery was greatly increased by the fact that the five cases of dysentery in March, 1865, all occurred within a week after the sewage was again allowed to flow over the field, during one night, when the direction of the wind was towards the Asylum.

In time of war, as well as in tropical epidemics, the conditions are generally so complicated as to defy analysis. Chevers believed that much of the dysentery (as well as cholera) occurring on board vessels in the port of Calcutta is caused by men drinking the water taken up in buckets over the ship's side, this water being loaded with sewage. Heubner was told by several military surgeons that when many severe cases of dysentery were crowded together, the disease was often spread by the latrines, and ceased when the proper precautions were taken. Maclean found that in India the barrack-rooms most exposed to the effluvia from latrines furnished the largest number of dysenteric cases.

It appears to be probable, then, that the cause of dysentery may be, and probably always is, the entrance into the body of some organic matter, conveyed usually by drinking-water, but sometimes, perhaps, through the air. So far dysentery resembles enteric fever and cholera.

Specific contagion.—The next question is whether the poison is a chemical product of decomposition, or a living organism, which undergoes a process of self-multiplication in the human body, so that the discharges can infect others with dysentery. This question cannot yet be finally answered.

All observers are agreed that the disease seldom or never passes from the sick to those who are attending on them; and Heubner says that it has hardly ever been known to spread from the military to the civil population in time of war. But dysentery may still be an infective disease, belonging to the same class of miasmatic-contagious maladies as cholera and enteric fever. No doubt it is a local disease, and its symptoms depend on the intestinal lesion; but it may still be strictly infective.

Evidence that dysentery is, at least in its severe form, a specific and infective disease was afforded by an epidemic which occurred in Norway in 1859. Dysentery had not prevailed there for half a century, and as the population was scattered, there was unusual opportunity of tracking its course. The disease was studied with great care by Homan and Hertwig, and they were convinced that it spread by contagion.

It is disappointing to find that increasing knowledge has not established a comprehensive theory of the origin and pathology of dysentery. Its relation to severe diarrhoea with ulcerative colitis may be compared with that of Asiatic cholera to sporadic cases of so-called English cholera, or of diphtheria to tonsillitis; and some cases called catarrhal dysentery may be non-specific diarrhoea or psilosis. But if we keep strictly to the definition of dysentery as diarrhoea accompanied by hæmorrhage and tenesmus, and associated with ulceration of the rectum and colon, the question remains whether the amœbic cases should be entirely separated from the rest, and if so, whether the latter are specific or no. No difference is

yet established between the bacteria found in a dysenteric colon and in the colon after death from other causes; and Dr Manson, while admitting the frequent presence of amœbæ in dysenteric stools, can find no satisfactory distinction between the cases in which they are found and those from which they are absent.

Gassen, in 1893, from a study of dysentery among the French soldiers in Algiers, and Celli and Fiona from cases in Italy and in Egypt, arrived independently at the conclusion that the presence of amœbæ in dysenteric stools is accidental, not pathogenic. In any case it seems at least premature to call all tropical cases amœbic dysentery, and to ascribe the rest to the presence of a specific bacillus or leave them under the head of non-specific ulcerative colitis.*

Diagnosis.—A question of great importance is whether dysentery is always attended by the striking train of symptoms seen in the severe tropical and epidemic forms. Tenesmus is a rectal symptom, and is absent when the inflammation is limited to the cæcum and upper colon. Dr Clouston found in the epidemic which he observed at the asylum near Carlisle, that some patients experienced scarcely any pain, and at first, having no fever or want of appetite, refused to believe they were ill, though they were passing glairy mucus mixed with blood. It does not appear that tropical dysentery is ever latent when the disease is epidemic; but Dr Fagge saw at least two well-marked instances of fatal sporadic dysentery, in each of which there was extensive ulceration of the large intestine, the presence of which had been unsuspected during life. Both patients died in Guy's Hospital, the one of an enormous hepatic abscess, the other of a large abscess in the left iliac fossa, resulting from perforation of the bowel into the post-peritoneal connective tissue. Dr Dickinson has recorded a similar case, that of a woman who died in St George's Hospital of abscess of the liver, and who, during the week which she passed as an inmate of the hospital, was so constipated as to require aperient medicines. She would not allow that she had ever had any looseness of the bowels, but after death the upper part of the large intestine was in a state of ragged ulceration.

In three cases of acute dysentery that have proved fatal in Guy's Hospital the disease was supposed during life to be *enteric fever*; the characteristic symptoms were either absent or passed under the name of diarrhœa. It is possible that a similar latency or obscurity of symptoms may sometimes occur in epidemic dysentery, both in India and in temperate climates; and we shall hereafter see that the question has an important bearing on the origin of abscess of the liver.

Even when all the symptoms of dysentery are present, the diagnosis should be made with caution.

Dr Manson says that in Africa the *Bilharzia hæmatobia* (p. 446) may affect the rectum as well as the bladder, and simulate dysentery. The ova would be discoverable in the fæces, and probably in the urine.†

In children, and particularly in male children, one must always bear in mind that *invagination* causes very similar symptoms. In this country more than one case has been mistaken for dysentery, and this very serious

* The *Amœba coli vel dysentericæ* was described by Lambe in 1859; afterwards by Loesch and by Katulis in Egypt in 1883. The fullest account of "amœbic dysentery" is given by Councilmann and Lafleur in vol. ii of the 'Johns Hopkins Hospital Reports.'

† Dr Ficket, of Liège, has published an interesting case of this disease from the Congo ('Bull. de l'Acad. Royale de Méd.,' 1897).

error must often be committed in those parts of the world in which the latter disease being endemic is likely to have its presence taken for granted.

A rectal *polypus* in children, not infrequently leads to pain, hæmorrhage, and tenesmus.

In those more advanced in years, *cancer of the rectum* may be overlooked, and its symptoms attributed to dysentery. Indeed, as a rule, a supposed case of chronic dysentery in an elderly patient, who has not been out of England, is really one of local disease of the rectum, most probably cancer; a case of this kind was lately sent up to the writer from the country. A digital examination generally clears up all doubt as to the nature of the disease; and in most cases the blood passed during defæcation is not intimately blended with the motion, but comes before or after it.

Whether cases of *ulcerative colitis* with passage of blood and mucus, usually occurring in women and children, should be called dysentery is, from a clinical point of view, little more than a question of terms; but it is better not to apply the terms dysentery or dysenteric to such cases.

Prognosis.—With regard to the grounds on which a forecast must be based in a case of dysentery, there is little to say that has not been implied in preceding paragraphs. The severity of the disease is proportional to the extent and intensity of the local inflammation, but this must nevertheless be measured during life by the gravity of the constitutional rather than of the local symptoms. Tenesmus and pain may be slight or even absent, and yet the patient may be in danger; or his sufferings may be extreme, and yet he may do well, if the disease is limited to the rectum. The appearance of his countenance, the state of the pulse, and the presence or absence of symptoms of collapse, generally lead to a correct judgment of the probable issue of the case. The rate of mortality varies greatly in different epidemics. In Dr Clouston's Asylum it was more than half of those attacked. This is enormous, for even in the worst tropical epidemics the proportion of deaths to admissions into hospital appears from a table drawn up by Sir Alexander Tulloch to be only 14·2 per cent.

In dysentery the prognosis is also affected by the presence of *scurvy* as a complication. This is so frequent that some writers speak of scorbutic dysentery as a distinct variety of the disease. But the symptoms in such cases appear to be only a combination of those due to each malady separately. It is stated that when scurvy is present, the course of dysentery is prolonged; that a patient almost always lives three weeks, and often as long as three months.

Even the most severe form of uncomplicated dysentery is seldom rapidly fatal. Death rarely takes place within the first week, or before the ninth or tenth day.

Chronic dysentery is an intractable disease, and the patient often dies at last by exhaustion, with septic febrile symptoms, even if no complication cut short his existence. Nevertheless, in the majority of cases recovery at length takes place, if they are judiciously treated. Sometimes, however, the bowels remain irritable for long afterwards, it may be for the rest of the patient's life.

Prophylaxis.—The prevention of dysentery generally depends on the good drainage, ample supply of pure water, and better diet which have nearly extirpated malaria from England.

When the first cases of an epidemic appear, among soldiers, prisoners, or townspeople, the first measure is to separate the sick from the healthy,

and to spread out the latter in huts or under canvas, instead of in houses or barracks.

The evacuations of those who are sick should, when possible, be disinfected with carbolic acid or sulphate of iron, and removed without delay. Those who are still well must clothe warmly, wear a flannel binder, and be careful to avoid chills; they should eat and drink moderately, taking no spirits or strong wine, and they should not allow the bowels to become constipated. Ripe fruit taken in moderation need not be excluded from the dietary. Every case of colic, constipation, or diarrhœa should be reported to the medical official at once.

Treatment.—The rational therapeutics of dysentery, as of other diseases, must be based upon a knowledge of its natural course. The late Dr Austin Flint once observed ten cases in succession in which no medicine was administered except a little tincture of cinchona as a *placebo*; and he found that the mean duration was eleven days and a fifth, the most protracted case lasting twenty-one days and the shortest six. Or, reckoning from the first dysenteric evacuation (instead of from the commencement of illness), he obtained a mean duration of eight and a half days, the maximum being twelve and the minimum five days. Dr Flint had before analysed forty-nine cases, which had been treated, some with calomel and opium, some with opium alone, some with castor oil, and others with astringents. The mean duration was almost exactly the same in these cases as in those in which the disease ran its natural course.

These particular figures apply only to dysentery as it occurs in the city of Philadelphia, and in strictness only to the actual period within which the observations were made. It is clear, moreover, that Dr Flint's cases were mild ones; for he states that the complaint showed no tendency to become chronic and that relapses never occurred, although he allowed his patients to eat solid food as soon as they chose to do so.

Very different is Professor Maclean's account of dysentery in India. "Speaking from large experience," he says, "I affirm that complete restoration to health, by the unaided efforts of nature, is of extremely rare occurrence; the disease either destroys the patient or it passes into a chronic form."

Even if Flint's observations appear to discredit the various remedies employed in his cases, we have in ipecacuanha another medicine which he does not seem to have tried, but which there is good reason to believe capable of cutting short dysentery. The root of this plant was first employed as a remedy for dysentery in Brazil, where it is indigenous. Towards the end of the seventeenth century it was introduced into France, where it was successfully given to the Dauphin, acquired a great reputation, and was known as the *radix anti-dysenterica*. In India it was used before mercury came into vogue, and of late years it has become the staple remedy.*

The action of ipecacuanha is "specific," *i. e.* as yet unexplained. It renders unnecessary the use of castor oil, tamarinds, rhubarb, or the sulphates of potass or soda, which are recommended by different writers. It is curious that Heubner, in 1874, knew scarcely anything of the value of ipecacuanha, except as an emetic; he had only heard that it was used by some English surgeons with good results in the war of 1870. Even Eich-

* "Dehinc ad radicem Ipecacuanha confugiendum, qua nullum præstantius aut tutius, cum vel sine sanguine, ad fluxus compescendos Natura excogitavit remedium."—"Gul. Pisonis de Medicina Brasiliensi," lib. ii, cap. xi, Lugd. Batav., 1648: quoted by Woodward.

horst, writing so late as 1887, does not mention this invaluable drug among the means of treating dysentery.

The method of administering ipecacuanha in acute dysentery, now general, was introduced by Surgeon Docker, of the 7th Royal Fusiliers, in 1858, and first tried by him in the Mauritius.

The patient having been put to bed, twenty-five to thirty grains of powdered ipecacuanha are given to him in as small a quantity of fluid as possible; a little syrup of orange-peel covers the taste as well as anything. Often thirty minims of laudanum are given half an hour before, in order to make the stomach tolerant of the ipecacuanha; but Maclean found the latter drug well borne without any such preparation. After the dose the patient should keep perfectly still, and abstain from drinking for at least three hours. If thirsty, he may suck a little ice, or have a teaspoonful of cold water at a time. Under this management he seldom complains of excessive nausea, and vomiting rarely sets in within two hours. A poultice is in the meantime placed over the abdomen, or folded flannel or spongion-piline, wrung out of hot water with a little turpentine sprinkled over it. Afterwards a little bland nourishment is given. In from eight to ten hours, according to the urgency of the symptoms and the effect of the first dose, the ipecacuanha is repeated, its quantity being somewhat reduced, but with the same precautions as before.

"All who have had opportunities of trying this mode of treating dysentery," says Maclean, "can bear testimony to the surprising effects that often follow the administration of one or two doses of ipecacuanha. The tormina and tenesmus subside, the motions quickly become feculent, blood and slime disappear, and often after profuse action of the skin the patient falls into a tranquil sleep, and awakens refreshed." The remedy may, however, be required in diminished doses for some days; and after the stools have regained a healthy appearance it is well to administer ten or twelve grains at bedtime for a night or two.

Even when the powers of life are very low this remedy may sometimes be given with safety and success. Maclean mentions the case of a lady who landed at Madras, having come from Calcutta, and who was so exhausted that her voice was scarcely audible. With some misgiving he gave twenty-grain doses at intervals of eight hours; and after the third dose she was out of danger.

When severe vomiting follows the administration of ipecacuanha, Maclean says that coexistent liver disease may be suspected, or complication with malarious fever. In the latter case he advises that quinine in ample doses should be alternated with the ipecacuanha.

In mild cases he recommends that the treatment should be commenced with a hot bath, which must be brought to the patient's bedside. He is to be kept in it until he feels faint, and after being rapidly but carefully dried he is to be put to bed, and to have a dose of fifteen to twenty grains of ipecacuanha. In some cases a few drachms of castor oil, with a little tincture of opium, may be afterwards prescribed. According to Heubner, enemata of starch and opium often give great relief to the tenesmus in these mild cases.

All Indian experience is in favour of strict confinement to bed and a very spare liquid diet—milk, if it is well boiled; and rice water, chicken broth, or white of egg. Locally, hot poultices, fomentations, turpentine stupes, dry bran poultices, hot-water bottles, or Japanese "hot-boxes,"

wrapped in flannel and laid on the abdomen, are the most useful measures.

The value of ipecacuanha in treating dysentery is attested not only by individual experience, but by statistical results, which are most striking. In Bengal, under "the old system," the average mortality among Europeans from dysentery during forty-two years was 88·2 per thousand; in 1860, under ipecacuanha, it was 28·87 per thousand. In Madras the corresponding numbers were respectively 71 and 13·5. Surgeon Mee, at Madras, treated sixty-eight cases from the 44th Regiment "in the ordinary way," with a mortality of 6 (or 88 per thousand); afterwards he treated fifty-nine cases with large doses of ipecacuanha, and these all recovered. It is also asserted that, as the use of this remedy becomes more general, the number of chronic cases of dysentery diminishes year by year, and the development of hepatic abscess becomes less frequent. Hygienic improvements, however, may have had a share in this result.

Against these statements it is right to set Dr Clouston's experience in the epidemic at the Cumberland and Westmoreland Asylum (p. 394). He found the ipecacuanha treatment useless, even if it did not take away the last chance from one or two of the patients by causing vomiting that could not be stopped, and prostration that was never rallied from. This epidemic was far more severe than ordinary tropical dysentery; but Dr Baly also found ipecacuanha wholly fail in his hands.

The experience of the American surgeons of ipecacuanha in the treatment of acute dysentery during the Civil War was also not so favourable as those in India. Dr King reported good results in the Confederate army at Richmond, and several other favourable results are referred to in the official report already quoted; but its trial at Washington ended in disappointment, as was the case in an epidemic in South Carolina in 1868. Dr Osler believes that it is far less effectual in America than in the East Indies.*

In a recent sporadic case, the present writer observed $\mathfrak{m}\mathfrak{x}\mathfrak{v}$ of ipecacuanha wine every four hours, prescribed by a medical friend who had seen much of dysentery in China, completely relieve the symptoms in twelve hours. The chronic cases of tropical dysentery which come before us in London, though far less amenable to this treatment, are also in most cases favourably influenced by it.

Calomel has been very generally abandoned in the treatment of dysentery; but Dr Manson still advises small daily doses of calomel, or of sulphate of soda, in cases which resist ipecacuanha. Sulphates of soda or magnesia appear to be the drugs most used by present colonial surgeons beside ipecacuanha. Opium is believed by all experienced observers to be injurious if systematically given, and venesection to be not only useless, but mischievous.

In severe and malignant forms of dysentery Maclean recommends the solution of pernitrate of iron; he says that he has sometimes prescribed ten drops every hour with advantage. Dr Clouston found that milk boiled with a little flour, and allowed to cool, was taken better than anything else; but he also gave strong beef-tea, jelly and eggs, and wine and water. Brandy also is often necessary.

In *chronic dysentery* an essential part of the treatment is removal to a better climate. In India it sometimes suffices to send the patient to the sea-

* The cardinal rule is too apt to be forgotten that in all the worst cases of a disease a medicine may fail to produce any appreciable benefit, and yet it may be capable of curing those which tend but a little less surely towards a fatal termination.—C. H. F.

side or the hills ; but more often a voyage to Europe is necessary. For those who are invalided home on account of chronic tropical dysentery, rest in bed is a very important part of the treatment. The late Dr Ward insisted on this, and on the diet being restricted to milk and farinaceous food. His experience at the "Dreadnought" led him to believe that ipecacuanha is useless in chronic cases ; but Maclean says that exacerbations of a subacute character are of frequent occurrence in chronic dysentery, and that at Netley he often gave the ipecacuanha in suitable doses with the happiest effect. Our experience at Guy's Hospital has certainly been that this medicine is of great value long after dysentery has been brought home from the East. In some instances it undoubtedly did good, even in out-patient practice.

Dr Ward's patients were sailors, whose food on board ship would have aggravated the disease, and it is not surprising that they derived so much benefit from rest in bed and a milk diet as to throw the effect of medicines into the background. Even when a person affected with dysentery is sent home as an invalid, with nothing to do but to take care of himself, the disease is very apt to become worse on board ship, unless great care is taken that he has proper food and puts on warmer clothing as he passes into a colder climate.

In some cases of chronic dysentery astringents are of great value. The solution of pernitrate of iron, besides checking the discharges, removes anæmia ; and krameria, catechu, hæmatoxylum, even tannic and gallic acids, may each be useful in turn. Marked benefit sometimes results from the administration of drachm doses of the extract of Indian bael, and acetate of lead or sulphate of copper is often useful. Dr Ward mentions one case in which enemata of nitrate of silver (gr. iv ad aq. ʒij) were repeated every night with good effects ; and Dr Galton found much benefit at Shanghai from enemata of sulphate of zinc, gr. v, and Liq. Opii Sed., ʒss, in ʒij of water.

In some bad cases of chronic tropical dysentery, the writer has found that, next to confinement to bed and abstinence from meat, broths, and vegetables, the most useful results have followed sulphate of copper and opium pills, and enemata of silver nitrate.

Sir Joseph Fayrer's ripe experience of the treatment of dysentery in the chronic form, seen in Indian officers invalided home, is that drugs are of far less value than in the acute cases seen in the tropics, and that an exclusively milk diet is the most important means of cure.

TYPHLITIS

“ . . . Venienti occurrere morbo
Disciteque O miseri, et causas cognoscite rerum.”

PERSIUS, Sat. iii, 64, 66.

History and name—Frequency—Anatomy—Symptoms and clinical course—of mild cases—recurrent—suppurative with adhesions—sloughing with septic peritonitis—Diagnosis—Sex and age—Pathology—Prognosis and treatment—Cases.

THERE are three parts of the intestinal canal in which the contents are apt to accumulate; the caput cæcum coli, the sigmoid flexure, and the rectum. All three are somewhat dilated, and in the cæcum and sigmoid there is a sudden change of direction which favours delay. Moreover there is the appendix cæci, which, like some other vestigial organs, is a frequent source of mischief. No wonder, therefore, that inflammation of the cæcum, or typhlitis, is a common and important disease.

In 1836 Addison wrote as follows. After describing the pain, swelling, and tenderness, with the frequent consequent suppuration and the occasional symptoms of general and fatal peritonitis, he proceeds:—"From numerous dissections it is proved that the fæcal abscess thus found in the right iliac region arises in the great majority of instances from disease set up in the appendix cæci. This organ is very subject to inflammation, to ulceration, and even to gangrene."*

Typhlitis† has been long recognised, but has been far more often diagnosed since the modern treatment by operation has become common; and in some parts of America it has even become a fashionable disease. And while typhlitis has become more common it has also become more fatal. This general impression is so far supported by some statistics collected by Dr Pitt ('Guy's Hosp. Gazette,' Feb. 27th, 1897, p. 93). In sixteen years (1880-95) the cases admitted (with the same number of beds available) were in the first ten years four times under 10 in number (5, 8, 3, and 9 cases), five times either 11 or 12, and once only (in 1883) 17; while in the next six years (1890-95) the cases admitted were only once as few as 5, three

* 'Elements of the Practice of Medicine,' by Bright and Addison, vol. i, p. 499. The second volume, which was to have been written by Dr. Bright, was never published; the first was, Sir S. Wilks informs the writer, entirely the work of Dr Addison.

† Typhlitis, from τυφλόν, cæcum (sc. *intestinum*), the blind gut, or *caput coli*. The *caput cæcum coli* of human anatomy is a mere dilatation of the colon, found only in man and certain apes. The homologue of the greatly developed true cæcum of many of the lower animals (e.g. the horse and rabbit) is the atrophied *appendix cæci*, which only occurs thus reduced in man and the anthropoid apes, and in the wombat.

times between 12 and 21, and in 1894 and 1895 as many as 30 and 40. The number of cases admitted in 1896 was 35; in 1897, 58; in 1898, 61; but in 1899 only 21.

Of late it has been the fashion to call cases of typhlitis by the barbarous name *appendicitis*, but this is unnecessary and misleading. For some writers of repute have distinguished between this and a supposed typhlitis which affects the caput cæcum coli alone, and is to be treated in a different way from appendicitis. Now if, as taught by Addison and Wilks, all typhlitis begins in the appendix, there is no need of a change of name. The appendix cæci is only the undeveloped extremity of the blind gut; so that morphology, as well as precedence, practical convenience, and euphony, is in favour of the older name of this important disease.

Typhlitis, then, is a disease which always begins in the appendix. The term Perityphlitis—another recent introduction—is ambiguous, for it may be taken to mean local peritonitis; while “paratyphlitis” might be applied to suppuration of the connective tissue round the cæcum, following the analogy of peri- and parametritis. When, as usually meant, it is a synonym for inflammation of the appendix, its use leads to the erroneous belief that suppuration behind and around the cæcum may arise independently of disease of the appendix.

No doubt there may be found “distension-ulcers” or “stercoral ulcers” in the cæcum as the result of obstruction in the colon, an abscess may form behind the cæcum from caries of the ilium or other accidental cause, and we may find tuberculous peritonitis chiefly affecting the cæcal region. But it is of great practical importance to recognise the fact that when such conditions have been excluded, we have only one and the same primary disease to deal with, of varied degrees of severity, but with the same anatomy and the same dangers.

For example, in two cases observed by Wilks after recovery from a mild attack, a second proved fatal, and in each the autopsy showed that the disease had begun in the cæcal appendix, which was perforated.

Anatomy.—Addison taught how suppuration from typhlitis, spreading upwards behind the peritoneum, may reach the kidney, and simulate suppurative nephritis, or may involve the iliacus muscle and periosteum; or if the pus travels into the pelvis, it may in women cause symptoms like those of ovarian inflammation.

In the appendix itself the inflammation spreads from the mucous to the serous coat by ulceration, or, occasionally, by sloughing; and may end in gangrene. The cause of the inflammation is often the presence of a concretion, which may be the size of a pea, or as large as a date-stone. It sometimes consists of a substance like wax, but usually is composed of hard, dry fæcal matter, with more or less of earthy salts, sometimes enough to call a calculus. A mass of this kind may look like a cherry-stone, and has often been mistaken for one. Indeed, supposed “foreign bodies” from the appendix have frequently been found on examination to be of fæcal origin. It is, however, certain that seeds, pills, bristles, pins, shot, and pieces of bone have all been found in the appendix, and that some have led to its perforation.*

* Dr Pitt quotes Dr Rochaz, of Lausanne, who found sixty-five concretions in the appendix to be laminated in structure, and to consist microscopically of epithelium, leucocytes, mucus, and oily matter, with calcium and magnesium phosphates and carbonates. The brown colour and fæcal odour are no doubt derived from the intestine, and even a minute fragment of fæcal matter may form the nucleus of a calculus.

The appendix may be so narrowed at its opening into the cæcum as to prevent the natural passage of mucus secreted from the tubular glands, which therefore accumulates, and forms a cystic tumour of the appendix. Wilks records a case in which the opening into the cæcum was closed; the appendix was dilated to the size of the ileum, and distended with three or four ounces of white odourless mucus.

The immediate cause of the inflammation is probably most often the entrance of micro-organisms from the cæcum into the appendix.

Symptoms and course.—Clinically we may assign cases of Typhlitis to three categories:

(1) Mild, sometimes chronic, and often recurrent, without suppuration, but leading to adhesion, and inflammation around the appendix.

(2) Subacute, with suppuration around the appendix.

(3) Very acute, sloughing, with perforation and septic peritonitis.

The earliest symptom of typhlitis is usually pain, referred mainly to the right iliac fossa. This is more or less paroxysmal, often of extreme severity, and associated with tenderness, so that sometimes the patient cannot bear even the slightest touch. The most tender spot is often, but not always, at what is called McBurnie's point (two inches from the anterior superior spinous process of the ilium towards the umbilicus). Nausea and vomiting are frequently present, and also in most cases marked constipation. The amount of general disturbance is variable; sometimes there is no fever, and vomiting is often absent.

The right half of the abdominal wall is tender and rigid, and there is an ill-defined swelling gradually extending upwards and inwards from Poupart's ligament. This is doubtless formed in part by the thickened coats of the affected portion of bowel, but it is due to the accumulation of faecal matters also. The size and form of this swelling may vary from day to day, and if the disease subsides it gradually disappears. The tumour is dull on percussion; but light and very careful percussion is necessary to bring this out, for resonance spreads, but dulness does not. Occasionally local oedema may be detected, and this is a sign of great importance, for it indicates suppuration. The patient often lies with the right thigh flexed, and is unwilling to stretch it down.

In the most severe cases the appendix early becomes gangrenous, and intense peritonitis lights up almost instantaneously throughout the whole serous cavity. There may then be no means of ascertaining that ulceration or sloughing of the appendix formed the starting-point of the disease; the case is set down as one of acute peritonitis, the cause of which is unknown. When such cases end favourably, it becomes possible to detect a hard swelling in the right iliac fossa, and this clears up their nature.

In many cases, however, the symptoms are mild and even obscure; there is no evidence of peritonitis, and the main clinical feature is inability of the intestinal contents to pass the cæcum. Occasionally there is diarrhoea from the first, at Guy's Hospital in about a fifth of the number.

When, as is often the case, suppuration is limited by adhesions, so that there is no general peritonitis, the pus may take one of several different courses. If the appendix is behind the cæcum, it commonly burrows upward into the lumbar region, where it may simulate a suppurating kidney. If it is more superficial in position, the abscess may open in the groin and discharge safely. If it burrows downward, as it does when the appendix hangs over the brim of the true pelvis, abscesses are formed between the

bladder and rectum which may open into either cavity, or a "pelvic cellulitis" is set up which may be indistinguishable from that which follows lesions of the Fallopian tube in women.

A curious and pathologically interesting complication of typhlitis with suppuration is the formation of a "metastatic" or "sympathetic" inflammation of the parotid gland. A case occurred in 1896, at Guy's Hospital, in a boy of thirteen, who recovered after an operation.*

Diagnosis.—As above stated, typhlitis may closely simulate obstruction of the bowels; indeed, it is a form of obstruction, though physiological, not mechanical. Diarrhoea is sometimes present, but this is the exception; and peristalsis is stopped in the cæcum, even without peritonitis.

In cases only seen after peritonitis has developed, the diagnosis may be difficult or perhaps impossible. From tubercular peritonitis the diagnosis is usually easy; but here also it is sometimes impossible to make, for what began as typhlitis not infrequently ends as *tabes mesenterica*.

Cases of typhlitis have been mistaken for enteric fever; in the former diarrhoea may take the place of constipation, and in the latter it is far from rare for diarrhoea to be entirely absent. The rose-rash is often wanting in children, and the course of the temperature will not always be distinctive, or at least not at first.

The early pain of typhlitis often closely resembles colic, and may be really due to muscular spasm of the cæcum or the appendix. Here, again, it is best to recognise colic-like pains as symptoms, not only of true colic, as defined above (p. 367), but also of typhlitis, of renal calculus and gravel, of moveable kidney, of gall-stones, and of intestinal obstruction.

In a remarkable case seen by the writer with Dr. Ransom, of Nottingham, the symptoms of typhlitis with suppuration were found—thanks to his exact examination of the pus—to be due to actinomycosis of the cæcum.

Lastly, suppuration in the right iliac region may be due, not to typhlitis, but to ovarian disease, salpingitis, morbus coxæ, caries of the ilium, or post-peritoneal abscesses originating in the kidney or the vertebræ.

Sex and age.—Typhlitis is much more common in males than in females. Out of Dr Fagge's 10 cases, 8 occurred in boys or men; of 31 patients under the writer's care, 22 were males. In 224 cases collected from recent records in Guy's Hospital by Dr Pitt, there were 160 males to 64 females. In 72 cases in America, the proportion was 2 to 1 (Fitz); but in Munich it was curiously reversed (Einhorn).

Typhlitis is a disease of early life; in 8 of the writer's 31 cases the patients were between five and twelve, in 19 between fifteen and thirty, and in 4 between thirty-eight and forty-five years old. Larger numbers yield similar results; most patients are under fifty, and few over thirty-five.

Ætiology.—The immediate cause of typhlitis is, as the anatomy in fatal cases proves, inflammation of the cæcal appendix. This may be due to a concretion, or to the entrance of a foreign body from the cæcum, but is most often caused by the admission of pathogenic microbes from the intestine.

In most autopsies no mechanical cause of the disease is found. Some cases are probably due to dilatation of the opening into the cæcum, allowing entry of fæcal matter, or at least of septic bacteria; others to constriction of the same orifice, causing retention of the natural secretion. In both cases the pathological condition begins in the mucous membrane.

* Similar parotid abscesses may follow other forms of abdominal suppuration, as Mr Stephen Paget has shown.

Dr Pitt calls attention to the mechanical disadvantage of the circulation in the appendix, due to the length its vessels have to run from the mesentery, as favouring necrosis. Moreover the mere length of the organ must often expose it to displacement and pressure.

There must be some reason for typhlitis being so much more frequent in youth, and particularly between ten and twenty-five, than in later periods of life. This has with much probability been sought in the abundant lymphatic (adenoid or cytogenic) tissue of the appendix. This in youth is well developed, active, and prone to inflammation, as is shown by the prevalence of diseases of the tonsils, the lymph-glands, the spleen, the thymus, the cancellous tissue of bones, and the lymphatic follicles of the intestine in the period of childhood and early adult life.

As exciting causes of inflammation, habitual constipation has been admitted by some writers, and others have observed the concurrence of an attack of typhlitis with rheumatic or enteric fever or influenza; and it seems not unlikely that a febrile, and especially a septic, process should light up a local inflammation.*

Treatment and prognosis.—Few cases of typhlitis which are diagnosed early end fatally, if judiciously managed; but the more severe cases, with ulceration or sloughing of the appendix, need surgical interference. Everything depends on the disease being early detected, and its progress checked. *Obsta principis* is the motto for this as for many other maladies. Delay and careless treatment in the early stages may convert a mild into a most dangerous malady. There are also a minority of cases in which ulceration and perforation of the appendix takes place with little previous warning, and these are most formidable, for purulent peritonitis may have resulted before medical aid is sought.

Dr Eyre examined the clinical records of Guy's Hospital from 1867 to 1895, and found that of 308 cases of typhlitis admitted at all stages of the disease during those twenty-nine years, 261 made a good recovery, *i. e.* over 84 per cent. Of the total number only 46, not quite 15 per cent., were complicated by abscess, and of these 16 died. Dr Donald Hood, who gives these figures in his monograph on 'The Early Treatment of Appendicitis' (1900), compares them with the experience of St. Thomas's Hospital, given by Dr Hawkins, where of 264 cases 38 were complicated by abscess, and of these 10 died ('Diseases of the Vermiform Appendix,' 1895).

In all early cases, doubtful or not, it is essential that the patient be kept entirely in the recumbent posture; that he be strictly confined to liquid diet, that he be not allowed to take a single dose of aperient medicine, and that opium should be given freely. Under this treatment the pain subsides, the fever disappears, and the patient is in the great majority of cases freed from danger.

After the attack subsides, the greatest possible care must be taken to prevent a relapse. The action of the bowels should be obtained by enemata only, and never by purgative medicines; and the restriction to fluid food should be continued for at least a week. We often see relapses occur from disregard of these precautions.

* In the first volume of contributions from the Wm. Pepper Laboratory of Clinical Medicine (Philadelphia, 1900, p. 395), Dr Frazier gives the results of experimental typhlitis in the rabbit. He finds that stagnation of the contents is the most constant and certain antecedent of inflammation. He believes that when thus confined the innocuous *B. coli communis* is converted into a virulent organism; that this result is favoured by disturbance of the local circulation, and that it is little affected by foreign bodies.

Even when the patient takes every care in diet and in the management of his bowels, typhlitis is very apt to recur, sometimes at considerable intervals of time. Repeated attacks may occur with a few weeks or months between them, until at length there comes one so severe as to place life in imminent danger. The patient then at last submits to being kept in bed for a considerable time, and observes the greatest possible care during convalescence; probably for this reason the most alarming attack is often the last.

In more severe cases, often those which have been incautiously treated or not treated at all, typhlitis ends in suppuration; the pus collects in the loose connective tissue between the cæcum and fascia iliaca, and forms an external abscess, marked first by œdema and then by fluctuation. Sometimes it passes backwards out of the pelvis through the sciatic foramen, and points below the fold of the buttock; sometimes it passes downwards under Poupart's ligament into Scarpa's triangle, like a psoas abscess; sometimes it may open into the rectum, and occasionally into the bladder, as in the case of a boy under the writer's care some years ago, who discharged the pus *per urethram* and made a good recovery. Another and a very dangerous direction of suppuration is to follow a vertical appendix upwards towards the right kidney, or to form an abscess between the liver and the diaphragm.

The thick adhesions which form are a valuable protection for the peritoneum; but when suppuration has occurred, the abscess should be opened as soon as possible by Hilton's method and drained. In one case under the writer's care, the pus had been allowed to find a way for itself; a fæcal fistula formed externally, and this ended in lardaceous disease of the viscera.

Early surgical treatment has of late years been widely followed, and still more widely advocated both in this country and in America.* It has been advised to cut down upon the appendix, ligature, and remove it, in all cases of ascertained or presumed typhlitis. Such practice, however, would in all likelihood much increase the mortality of the disease, and may justly be regarded as meddlesome.

Even when suppuration has occurred and the abscess must be opened, it is probably wise in most cases for the surgeon not to explore the seat of disease, but to be content with incision and drainage for the time. Subsequently an exploratory—often a very difficult and protracted—operation may be undertaken, to find and remove the appendix. Often, however, it has been so much destroyed by sloughing and suppuration, that if the patient escapes with his life, he is free from a fresh attack.

The recurrent cases are usually the less severe ones. When, however, repeatedly recurrent attacks of typhlitis interfere with a patient's health and prospects, an operation for the removal of the appendix in the interval when there is no active inflammation, is in certain cases justified. Such a case in a patient of the writer, who for several years had been laid aside by frequently returning typhlitis, was successfully treated by Mr. Lane in August, 1888. On the other hand, three of the writer's patients have suffered from two or three severe and several slighter attacks, and,

* See Mahomed and Symonds ('Trans. Clin. Soc.,' 1885, p. 285), and Mr Treves, valuable paper in the 'Med.-Chir. Trans.' for 1888, p. 165; and also in 'Allbutt's System,' vol. iii, p. 932, which may be contrasted with the position taken by Prof. Dieulafoy, that the only rational treatment is by operation.

after persevering medical treatment, are now well without the inevitable risks of a serious operation. They have had no relapse for seven, three, and five years respectively (1901).

When there are no signs, local or general, such as œdema, fluctuation, high fever and vomiting, that suppuration has taken place, the treatment by starvation, absolute rest in bed, and full doses of opium, is what has been followed by good results; and there seems no reason to abandon it for treatment by purgatives, still less to submit such cases to operation.

When there is frequent recurrence of the disease, it is good practice, after recovery from a second or third attack, to operate deliberately in an interval of health and remove the appendix.

When there are signs of suppuration, after the severe symptoms have subsided under starvation, rest, and opium, and pyrexia is no longer present, the abscess should be opened and drained.

In the fulminating, acute, and severe cases, when there is already general peritonitis, medical treatment is useless; and the only hope is to open and wash out the peritoneum, leaving the local lesion for subsequent treatment.

Cases.—The following was the result of the writer's personal experience between 1875 and 1894:—Out of 31 consecutive cases, seven died, but of these two were exceptional—the one being a child who had a foreign body, to wit, a pin, in the appendix, and a secondary abscess of the liver, and the other a woman in whom typhlitis was complicated by tubercles of the peritoneum and other organs. Of the 24 other cases, one died a year after from tuberculous peritonitis, and another was last seen with symptoms of lardaceous disease of the liver and kidneys. In two of the cases which recovered there was general peritonitis with high fever; in five the typhlitis was recurrent a second, third, or fourth time; in only one an abscess was opened, and in another the appendix was removed. There was no operative interference in the five fatal cases, and in three of these there was obvious reason for their ill result, which would have rendered any operation fatal.

The following statistics were taken by Dr Fawcett from the Guy's Hospital case-books during two years (1896-7). There were 93 cases and 17 deaths. Of the 93, 67 were first cases, 26 recurrent attacks. In 16 cases a local abscess was incised and drained, and of these only 3 died—one from acute peritonitis, one from secondary abscesses in the liver, and one while convalescent, from embolism of the pulmonary artery; the other 13 made a good recovery. In 14 cases the appendix was removed, with three deaths. In each of them the operation was performed while acute suppuration was going on, and general peritonitis or abscesses in the lungs and liver were found after death. The remaining 11 cases which recovered were operated on after subsidence of the acute symptoms. An operation was performed on all the fatal cases but four; one of these was the case of death from pulmonary embolism, the other three were too exhausted when admitted into the hospital for an operation to be possible, and its uselessness was demonstrated after death by the presence of pyæmic secondary abscesses.

It is important to check the results of treatment in the medical wards with those obtained by surgeons, particularly since in some hospitals cases of supposed typhlitis are sent straightway into a surgical ward, like cases of strangulated hernia. Dr Pitt states that in sixteen years (1880—1895) 77 cases of typhlitis were admitted into the surgical wards of Guy's Hospital, and the result of operation was fatal in only ten of these, including one of death from the anæsthetic.

INTESTINAL OBSTRUCTION

“Tu Maximus ille es
Unus qui nobis cunctando restituis rem.”—VIRGIL.

“Horæ
Momento cita mors venit aut victoria læta.”—HORACE.

Introduction—classification—frequency and incidence on age and sex of the several kinds of obstruction.

IMPACTION—*Gall-stones—Concretions, etc.—Fæcal masses in the rectum or colon.*

COMPRESSION *of the gut from without by tumours or other causes.*

CONTRACTION AND STRICTURE—*Cicatrices and constrictions—Adhesions and contractions affecting the ileum—Simple and malignant stricture of the colon and rectum—its locality and anatomical structure.*

INVAGINATION—*Anatomy—Obstruction and subsequent strangulation—Ætiology and pathology—Symptoms: pain, tumour, hæmorrhage—course—duration of first and second stages—Diagnosis—Prognosis—Treatment.*

STRANGULATION—*Bands—Apertures in omentum or mesentery, etc.—Internal hernia—Volvulus—its seats, mechanism, and effects.*

Anatomical results of obstruction and strangulation from whatever cause—Origin—General symptoms: constipation, pain, vomiting—Special symptoms and course of chronic obstruction—Special symptoms of acute obstruction—Diagnosis—Prognosis—Treatment: expectant—by colotomy—by abdominal section—by other mechanical methods—Summary—Cases.

THE long and tortuous course of the alimentary canal offers many opportunities for mechanical obstruction to the passage of its contents, not only from cicatrices or tumours, as in stricture of the œsophagus and pyloric stenosis, which have their analogue in cancerous stricture of the rectum and colon, but by twists and displacements of the moveable small intestine, which are made more serious by the mesentery, which tethers its movements and conveys its nutrient vessels.

Passio iliaca, mechanical occlusion or obstruction of the bowels,* depends upon many and diverse anatomical conditions, and is pathologically heterogeneous. But every case agrees in the fact that obstruction from whatever cause, whether impaction of a plug or narrowing of the gut from disease, or pressure, more or less severe, from outside, inevitably leads, if unrelieved, to distension of the part of the intestine above the stoppage, to pain, vomiting, distension, and ultimately to peritonitis and death. In

* *Synonyms.*—Ileus, twisting of the guts—Miserere.—*Fr.* Iléus, occlusion intestinale.—*Germ.* Stuhlverstopfung.

many cases these effects of obstruction are hastened by external compression being so close as to prevent not only passage of the intestinal contents through the gut, but passage of the blood through its vessels; obstruction is thus complicated by strangulation, and the course of the symptoms is increased in severity and rapidity. Either slowly by ulceration from distension, or rapidly by gangrene from strangulation, the bowel is perforated, and acute septic peritonitis from extravasation of its contents is the result. The evil is not insuperable constipation—that may be borne for weeks,—but the rapid or slow supervention of septic inflammation.

The condition therefore is one of anxiety, and liable at any time to become of extreme danger. Moreover, although the fact of mechanical obstruction is in most cases easily determined (and even this is sometimes difficult at first), it is often impossible to fix upon the nature or even the seat of the obstruction, and until this double diagnosis can be made, our treatment is empirical, and either ineffectual or mischievous.

The milder cases of obstruction by plugging or moderate compression are clinically little more than obstinate constipation; the severe and acute cases of obstruction with strangulation closely resemble those of primary infective peritonitis.

The treatment, when a probable diagnosis can be made, is often successful; when, as too often happens, the diagnosis is obscure, we have to choose between the “masterly” delay which too often means watching the inevitable approach of death, or the “bold” exploration which too often hastens the catastrophe. The decision between the two lines of action indicated in the mottoes at the head of this chapter depends partly on personal temperament, and partly on personal and particularly recent experience. This, however, may be said: that increasing attention to details of symptoms and mode of onset of the disease, continual growth of large and accurate statistical data, and increasing numbers of verifying or correcting autopsies, will make medical diagnosis more and more assured; and that when the seat and the nature of the lesion can be determined at an early stage, success will almost always follow the skill of the surgeon.

Classification.—In order of their severity we may arrange the anatomical forms of ileus as follows.

1. The lumen of the gut may be plugged or blocked by a solid body. This is called Impaction, and is the least serious kind of obstruction.

2. A coil of intestine may be pressed or squeezed from without, so that if released from pressure it would return to its normal condition—Compression.

3. The calibre may be narrowed by thickening and contraction of the intestinal walls—a condition called, by analogy to a corresponding disease of the urethra and œsophagus, Stricture.

4. A portion of the gut may by irregular peristalsis force itself into the portion next following and so block it—Invagination or Intussusception; while gradually constriction of the vessels of the impacted gut follows, and adds strangulation to obstruction.

5. Strangulation of a coil of intestine may be produced by an external or internal Hernia, a constricting Band, a Volvulus or “twist of the guts,” or in other ways.

Each of these modes of obstruction may affect either the *small* or the *large* intestine, and the symptoms or results will differ accordingly.

For purposes of diagnosis it is useful to recognise some forms of obstruction as most commonly occurring in *children* (invagination and tuberculous cicatrices), others in *young adults* (bands and herniæ and volvulus), others in *later life* (stricture and impaction).

Again, the symptoms caused by obstruction of the bowels may run an *acute* or a *chronic* course. The former is more common in affections of the small, the latter in affections of the large, intestines. Chronic cases are more often caused by impaction or stricture, and acute ones by twists or severe compression.

Lastly, some forms of obstruction to the passage of the intestinal contents are complicated by *strangulation*, that is obstruction to the circulation in the part of the gut affected, while others are not. This important and dangerous addition does not follow impaction or stricture, but only extreme external compression, as by the neck of a hernial sac. Strangulation always includes obstruction. Obstruction frequently ends in strangulation; but the two processes are pathologically distinct, and each has its own clinical effects.

The comparative *frequency* of the various anatomical forms of obstruction is important from a practical point of view, for with due consideration to age and sex and other circumstances of the case, it furnishes us (as it does in the somewhat similar question of cerebral and thoracic tumours) with a probability from frequency, or improbability from rarity, of this or the other lesion being present. We are thus helped in deciding on the most likely conclusion to be derived from the symptoms and physical signs.

Nothing of value, theoretical or practical, can be said of the incidence as to the age or sex of intestinal obstruction generally; its causes are too various, and, indeed, are only accidentally connected by their results.

We must bear in mind that the only anatomical statistics of the least value are those based on examinations after death; and this means on hospital cases. For, in spite of the admirable zeal of some of those engaged in family practice, the difficulty they meet in obtaining an autopsy or even an abdominal inspection after death is often insurmountable.

Frequency.—In hospitals, cases of strangulated hernia and stricture of the rectum are usually separated from those of internal hernia and stricture of the colon, by being sent into the surgical wards. Excluding these cases, and also those of obstinate constipation which recover without proof of mechanical obstruction, the most frequently met with of all cases of ileus appears to be *invagination*, which takes a third, or more than a third, of the whole. It is, however, rare in adults. Among children it is far the most common form of obstruction, and it is nearly twice as frequent in males as in females.

Next to this certainly comes *stricture*, most often in the rectum and lower part of the colon, but also found in the ascending and transverse colon, the valve, and the small intestine in almost regularly diminishing numbers. Stricture supplies more than a third of the cases of obstruction, probably nearly half, but most statistics exclude stricture of the rectum, or, at least, stricture of the anus, because these fall directly under the surgeon's care and are treated locally. Stricture is rare under forty, and appears to be somewhat more common in elderly men than in women.

Obstruction by *bands* and by internal *hernia* comes third, and if we included strangulated inguinal and femoral hernia, this would probably be the most numerous class of all. But the latter being capable, with few exceptions, of ready recognition and effectual treatment, is excluded from statistics of internal obstruction. Whether external strangulated hernia is included or not, this form of obstruction is considerably more common in men than women (70 to 40 in Dr Fitz's list of acute obstruction).

Impaction by *gall-stones* is rare in men; most cases occur in elderly women. Impaction of *fæces* is common enough in women of all ages, but seldom produces what can be called mechanical obstruction. When combined with atony of the colon it causes more decided symptoms, and is not infrequent in both men and women over fifty years of age.

A twist or *volvulus*, as a primary form of obstruction, is rare. It is very seldom seen in children, and most often in adults between forty and sixty. It is decidedly less rare in men than in women.

IMPACTION.—The bowel is blocked by a mass occupying its lumen, which is either a biliary calculus, or a concretion, or the residue of food swallowed.

a. In the *small intestine* this very seldom occurs, except from the presence of a large gall-stone. Of this condition 41 cases were recorded by Leichtenstern, but it is not often met with. The seat of impaction is usually in the upper jejunum, or else at the ileo-colic-valve. This accident is most frequent in women, and after middle life (forty to seventy). The symptoms are not very severe; but vomiting is early and frequent; and though there is no distension there is considerable pain.

Two cases were published by the writer in the 'Path. Trans.' for 1887, in one of which the calculus was felt (though not recognised) *per rectum*. The patient, a woman of seventy, passed it safely, as did the second patient. One stone weighed 270 grains, the other 323.

It is curious how such large masses can reach the gut. In some cases they pass directly into it from the gall-bladder by ulceration, but in others very large calculi enter it by gradual dilatation of the duct.

In idiots the ileum has sometimes been found obstructed by balls made up of fibrous materials that had been swallowed. Dr Langdon Down, in 1866, showed to the Pathological Society a mass of interlaced cocoa-nut fibres the size of a hen's egg, which caused death by occluding this part of the intestine; the patient, a boy aged sixteen, had a trick of putting a shred of cocoa-nut fibre into his mouth, and playing with it between his teeth. A similar case was recorded by the late Dr Mackenzie Bacon.

Obstruction of the smaller gut by concretions of magnesia is extremely rare. One example is preserved in the museum of St Thomas's Hospital.

It very rarely happens that ordinary articles of food cause obstruction. An instance is related by Brinton in which an abdominal tumour, the size of a pullet's egg, was believed to be formed of a mass of half-chewed filberts. Having been first detected in the right hypochondrium, it in two days moved almost entirely downwards into the left iliac fossa, and then disappeared; a few hours afterwards the bowels acted for the first time. Even such foreign bodies as coins, knives, and, in one case, the tube of a stomach-pump, find their way safely from the stomach to the anus.

Cases of obstruction of the small intestine by round-worms have been recorded by Duchaussoy and other writers. The late Dr Beavan Rake met with more than one case of it among negro children in Trinidad.

β. In the *large intestine* obstruction has occasionally been caused by concretions. Of these one variety (less rare than in the ileum) consists chiefly of magnesia.

A case in point was brought before the Pathological Society in 1855 by Mr Hutchinson. It was that of a lady who had the rectum blocked by a rough hard body, at least fifteen inches in circumference, which had to be broken down before it could be removed. It was made up partly of strawberry and other seeds, partly of concentric layers of what looked like red stone, but was found to be a mixture of iron and magnesia. The patient had formerly taken sesquioxide of iron and also carbonate of magnesia in large doses, but not for twelve years before the detection of the mass in her rectum.

In persons who eat largely of oatmeal another kind of concretion is sometimes met with, consisting of a felted mass of hairs derived from the grain. Such masses have a soft velvety feel. Several specimens of them are preserved in the museum of the College of Surgeons; their nature was first suspected by Mr Clift, and afterwards demonstrated by Dr Wollaston. As might be suspected, they have been found in Scotland more often than elsewhere. They have not generally caused insuperable obstruction of the bowels, but have been passed after giving rise to more or less distress. In one case, quoted by Sir Thomas Watson, no less than thirty-two such concretions, varying from a hen's egg to a filbert in size, were voided at different times.

Other insoluble constituents of food may form obstructive masses in the rectum, and it is strange that they pass safely through the gastric secretion.

In a curious case the writer saw with Dr Galton, of Norwood, the patient, a middle-aged lady, passed with the aid of enemata a mass of what looked like fir needles. They dissolved in acetic acid, however, and under a low magnifying power were clearly the fin-rays of fish, probably soles, which must have gradually accumulated until the large mass almost blocking the rectum was formed.

Fæcal matter in the large intestine sometimes accumulates to such an extent as to cause symptoms of obstruction of the bowels.

A remarkable case of this kind occurred to Dr Peacock, and was recorded by him in the 'Pathological Transactions' for 1872. A man aged twenty-eight died in St Thomas's Hospital after an illness of six weeks, during which he had had obstinate constipation. The bowel, from the cæcum to the rectum, was found loaded with semi-solid greenish fæces, to the amount of fifteen quarts; it measured from six to eight inches in circumference. The patient had been subject to constipation from childhood, and for twelve years before his death his bowels had never acted without an enema, aperients having ceased to produce any effect.

Another case which seemed to be of this kind was remarkable, because the patient, a woman aged twenty-two, was attacked with abdominal pain and other symptoms of obstruction twenty-four days before her death, and yet at the autopsy no cause could be discovered for her illness but masses of scybala in the sigmoid flexure and rectum, although much had been removed during life by an enema.

COMPRESSION.—Compression from outside the gut may be by a solid tumour, an abscess, an aneurysm, or a cyst. It naturally is a rare cause of obstruction in the looser parts of the bowel, and is by far most common in the rectum, confined within the bony pelvis, and next in the duodenum or the last part of the ileum, both more fixed than other parts of the gut. The symptoms are those of chronic obstruction; they are ingravescent and often intermittent. In two cases under the writer's care the compressing tumour was a pregnant retroverted uterus, which was redressed, with re-

moval of the obstruction; in another it was an ovarian cyst, which had fallen into the pelvis, and was tapped *per rectum* with relief to the symptoms.

CONTRACTION.—Obstruction may be caused by external traction and constriction, whereby its lumen is narrowed without the circulation being seriously impeded—obstruction without strangulation.

This condition Dr Fagge, in the 'Guy's Hospital Reports' for 1868 (ser. 3, vol. xiv, p. 272), described and distinguished by the name of "*contraction*," cases often confounded with strictures, in which "the disease begins not within the intestinal coats but on their exterior, sometimes on the serous surface of the intestine, sometimes in the mesenteric glands. The obstruction, when it occurs, arises not merely from a narrowing of the calibre of the bowel, but partly from the adhesion of one coil to another or to some other structure, or from puckering and contraction of the mesentery." Dr Bristowe afterwards called this condition "compression and traction." It includes cases otherwise described as "obstruction by adhesions," matting together of the intestines causing traction, compression, shrinking or bending of the gut.

In almost all cases it is a secondary effect of peritonitis, traumatic or tuberculous, and is therefore pathologically allied to obstruction from a band. Clinically, it resembles obstruction from a stricture. The morbid appearances are much less striking in this than in other forms of obstruction of the bowels, and they are not easily illustrated by drawings or preserved in pathological museums. Consequently it had remained almost unrecognised before the publication of Fagge's paper, although it is by no means of infrequent occurrence.

a. The cases of contraction there recorded affecting the *small intestine* were twelve in number. In four of them the affection was the consequence of chronic peritonitis, by which more or less of the small intestine was bound down to some part of the abdominal wall, or by which its coils were made to adhere among themselves. In two it resulted from the puckering caused by cancer affecting the serous covering of the bowel, and from adhesions which had formed. In one instance a somewhat similar condition of the intestine arose as a result of tuberculous peritonitis; and in three it was due to contraction associated with chronic disease of glands in the mesentery. The remaining two cases were of a less definite nature.

The peculiarity in this form of obstruction is that, instead of there being one particular spot beyond which the contents of the bowel cannot pass, the impediment is generally continued through a considerable length of the gut. The whole of the small intestine may be matted up, so that one is unable to say that one point more than another was the seat of the obstruction. Or there may be one or more sharp bends or twists or "kinks;" and sometimes it is clear how the part of the bowel above a bend, becoming distended, pressed on that below, and occluded it. Or, one portion of the intestine being fixed by adhesions, that above it may be stretched by accumulated fæcal matter, and hang down into the pelvis, so as to drag on the attached portion, and prevent anything passing through it. More than one striking instance of the latter kind has occurred in Guy's Hospital since 1868. One of the former kind came under the observation of Dr Bristowe, and was recorded by him in the 'Pathological Transactions' (vol. xxi, p. 185). In it the intestinal coils from the middle of the ileum to

within a foot of the cæcum were adherent to one another and to the brim of the pelvis by bands and filaments of false membrane, and were so entangled that their direction was traceable with difficulty; but there was no part of the bowel through which the finger failed to pass.

The origin of the adhesion and contraction is often the presence of an old hernial sac, or the injury inflicted by a strangulation which was relieved by herniotomy. Often it is pelvic cellulitis, with adhesions of the gut to the ovary, uterus, or broad ligament; often former suppuration of mesenteric glands, past typhlitis, and chronic tuberculous or cancerous peritonitis. As Mr Treves remarks, the fact that femoral hernia, pelvic peritonitis, and perhaps malignant peritonitis are more common in women than in men explains the greater frequency of female cases of this form of obstruction.

β. In the *large intestine*, obstruction sometimes arises from its being bound down or adherent to the adjacent structures; in other words, there is an affection analogous to "contraction" of the small intestine. Three such cases were related in Fagge's paper: in one, each end of the loop formed by the sigmoid flexure was bound down to the spine by firm fibrous tissue; in another the impediment resulted from adhesion of the transverse colon to the neck of an umbilical hernia; and in a third its cause was that the same part of the bowel had been dragged down and fixed to the mesentery over the lumbar vertebræ.

STRICTURE.—Narrowing, contraction, or stenosis of the gut owing to thickening of its walls is one of the more frequent causes of intestinal obstruction, but is almost entirely confined to the rectum and colon.

a. In the *small intestine* stricture is rare, and more often cicatricial than neoplastic. In one instance, after an operation for strangulated hernia, the patient suffered from continual vomiting, and died in two months; about an inch and a half of the small intestine was found to be narrowed, its coats thickened by hard, white cicatricial tissue, and its mucous membrane almost devoid of villi; evidently this was the part which had been in the hernial sac. Moxon noted two cases in which narrowing of the small bowel appeared to have resulted from the sloughing of an invagination, and similar instances are on record.

In the 'Pathological Transactions' for 1869 a remarkable case is related by Dr Wickham Legg, in which the opening from the ileum into the cæcum was only just large enough to admit a catheter; and similar instances have been recorded. He supposed that in his case the stricture was congenital, but it may have resulted from the shedding of an intussusception. The patient, a woman aged thirty-two, had nearly all her life been liable to attacks of what was termed colic, and six years before her death she was in hospital under Dr Walshe. It was then noticed that manipulation of the abdomen produced a peculiar crackling, which could be both felt and heard. That this was due to the presence of cherry-stones in the intestine was evident, for on one occasion she passed some. After death the intestine was found to contain almost enough fruit-stones to fill a pint measure; most of them lay in the jejunum or ileum at a distance from the stricture in the valve, but a few in the dilated pouch measuring seven inches in circumference, formed by the cæcum.

Most writers state that the cicatrization of tuberculous ulcers is a not infrequent cause of stricture of the small intestine; and that such a condition never follows the healing of the ulcers which occur in enteric fever. But no case of either kind is recorded in the 'Pathological Transactions,' and none seems to have been met with at Guy's Hospital within the last thirty years. The nearest approach to it is a case which occurred in 1858. A child died of phthisis and diarrhoea after an illness of three or four

months' duration. There were numerous large tuberculous ulcers in the bowel, some of them extending all round it. A remarkable degree of narrowing, apparently from contraction of the peritoneal coat of the intestine, was found at several parts, but symptoms of intestinal obstruction seem to have been altogether absent.

Mr Treves figures one case of double stricture of the jejunum from tuberculous ulcers, and quotes a unique case, reported by Klob, of stenosis after typhoid ulceration. He remarks that stricture in the small bowel is often double or multiple. Of 26 cases collected by him from various sources 10 were cancerous, and the rest were cicatricial; 10 after ulceration, 4 after hernia, and 2 after injury.

The seat was in 16 cases in the jejuno-ileum and in 8 at the ileo-cæcal valve.

β. In the *large intestine* stricture is by far the most common of all the lesions that give rise to obstruction. Its most frequent seat is the rectum; next the sigmoid flexure. Out of 100 cases, collected from Guy's Hospital, from the list of Mr Treves, and from that of Dr Coupland and Mr Morris (excluding the rectum), 58 were in the sigmoid flexure; while, of the remainder, 11 were in the descending colon, 8 in the splenic flexure, 7 in the transverse colon, 10 in the hepatic flexure, 2 in the ascending colon, and 4 in the cæcum. Brinton and Duchaussoy's results closely resemble these. Thus it may almost be said that the liability of the large intestine to stricture increases regularly from its upper to its lower end. Cases of stricture of the rectum of precisely the same pathological characters are most of them admitted into surgical wards.

Pathologically stricture of the large intestine differs greatly in different cases. In the rectum it occasionally consists of dense fibrous tissue, contracting the bowel and puckering its muscular coats; the result of injury in parturition or of operations for piles or fistula. Sometimes it can be traced to the cicatrization of a dysenteric ulcer; and more frequently the stricture (particularly in women) is syphilitic in origin, usually rectal in locality, and annular in shape.

But by far the most common form of stricture is some form of *malignant* growth. This often has more or less of a villous character. In some cases it forms a raised ring of a bright crimson colour, projecting with a smooth velvety surface into the cavity of the bowel. In other cases it is excavated by ulceration.

Histologically it may occasionally be a spheroidal-celled glandiform carcinoma, but it is far more frequently a cylinder-celled epithelioma. Indeed, Mr Harrison Cripps identified all reported cases of scirrhus or encephaloid cancer of the rectum as cylinder-celled; and so M. Hausmann (1882) and Mr Treves (1884). Occasionally the growth is found to have undergone extensive colloid degeneration. Secondary tumours in the lymph-glands or in other viscera are often absent; one reason being, no doubt (as in some cases of cancer of the stomach and of the uterus), that the duration of the disease is not protracted.

INVAGINATION.*—One of the most interesting and important forms of intestinal obstruction is what is known as intussusception or invagination. It occurs when one part of the gut passes into that immediately beyond it, just as the finger of a glove can be made to slip into itself. It appears to

* *Synonyms.*—Intussusception.—*Germ.* Darmeinschiebung.

come second in frequency after stricture. Invagination is, however, almost confined to the small intestine, and is most frequent at its lower extremity. Out of 500 fatal cases of obstruction collected by Brinton, no less than 215 were due to invagination, and Leichtenstern found out of 1152 cases of obstruction, 479.

The part of the gut which passes into that below it may be compared to a foreign body plugging the intestinal lumen. The coats of the invaginated portion speedily become greatly thickened by congestion and oedema, and thus produce a kind of stricture; and the pressure of the swollen gut upon itself, together with the dragging of the mesentery and its vessels, causes first congestion, and at last strangulation with gangrene. Hence three of the anatomical types of intestinal obstruction above described—impaction, traction, and strangulation—may be, and usually are, united in a case of invagination.

Invagination may occur at any age, but it is far most common in children. Indeed, it is the only kind of intestinal obstruction that is frequent under puberty. It begins acutely, but sometimes passes into a chronic condition afterwards.

Anatomy.—The direction in which invagination occurs is invariably downwards (or “forwards”); *i. e.* the sheath (*vagina, la gaine, recipiens*) is on the anal side of the intussuscepted portion (*le boudin, receptum*).

An invagination must obviously consist of three parts, or, as they are often called, “layers.” Of these we may term the outermost the “receiving,” the middle the “returning,” and the innermost the “entering” layer. The latter two together are called *le boudin* by French writers, which we may translate as “the plug.” The returning layer, unlike the others, has its mucous outside its serous coat; it is, in fact, turned inside out. The bend which connects the receiving and returning layers is situated at the upper part, and its convexity is formed by the peritoneal coat of the intestine; that which connects the returning and the entering layers is at the lowest point of the intussusception, and the mucous membrane covers it. Adhesions are apt to form where the two serous surfaces of the entering and returning layers come into contact.

Almost every portion of the bowel is liable to intussusception:—one part of the small intestine enters another, or one part of the colon another. But invagination occurs much oftener than anywhere else at the junction of the small with the large intestine; the ileum with the valve pushes forward into the cæcum, and then carries the cæcum on into the colon. Such cases are called *ileo-cæcal*, and make two thirds of the total cases of intussusception. There is very seldom protrusion of the ileum through the ileo-cæcal valve into the cæcum; perhaps less than 5 per cent. of this so-called *ileo-colic* intussusception, for which “valvular” would be a more distinctive name. Next in frequency to the ileo-cæcal variety is invagination of the ileum (less than a third), then that of the colon, and it is extremely rare for the rectum or the duodenum to be affected. A drawing of the last variety was published by the writer in an article in the ‘Illustrated Medical Magazine’ (Aug. 24th, 1889). A case of invagination of the rectum in a woman of thirty-five occurred at Guy’s Hospital in 1857, and it is recorded in the ‘Reports’ for 1860, p. 287; the rectum with just the lower end of the sigmoid flexure passed down through the anus, and there was some difficulty in distinguishing it from a mere prolapse of the mucous membrane.

In twenty-four consecutive cases of invagination at Guy's Hospital (1889), the writer found ordinary ileo-cæcal variety in eleven, the seat was in the ileum in two adult cases and in one infant, and there was one example of the rare valvular (or ileo-colic) form. The remaining nine cases recovered without operation, and their exact seat could therefore only be surmised; but they appeared to be ileo-cæcal.

Collected cases probably show an undue proportion of exceptional characters. In this as in other diseases the common type is probably more common and the rare types more rare than appears from statistics.

Course.—An invagination at first affects only a small part of the bowel. Gradually more and more is involved, and always by successive inclusion of one part after another of what had been the receiving layer. Thus the upper bend of the intussusception is constantly shifting, while the lower bend remains stationary from beginning to end. In an ileo-cæcal case, for instance, the entering layer is the ileum; the cæcum forms the returning and the receiving layers; and the lower bend is situated exactly at the ileo-cæcal valve. As the affection advances, the whole of the cæcum, the ascending, transverse, and descending colon may become included; but the ileo-cæcal valve always remains at the foremost point of the mass. Extraordinary as it appears, an invagination of this kind may pass through the anus; and the valve has actually been seen in this position, as well as the orifice leading into the vermiform appendix. The peritoneal layers of mesentery and the vessels of the invaginated gut must be stretched more than could have been thought possible; but there is no doubt that the protrusion of an ileo-cæcal intussusception from the rectum has repeatedly been observed, and it may be regarded as the natural result of the process. When the small intestine is invaginated, the mesentery tethers it much more closely; it is drawn in and forms a wedge-shaped mass on one side of the gut, which it pulls on, and drags its lower end to that side.

The next step is that the circulation of blood in the invaginated mass is interfered with. Thus to obstruction is now added strangulation of the intestine. The one condition prevents the passage of fæces, and acts in a way comparable to stricture of the urethra, stricture of the œsophagus, or stenosis of the pylorus. The other condition interferes with the nutrition of the invaginated tissues, and causes congestion, inflammation, and at last gangrene.

Sometimes, it seems, the veins alone are compressed; blood can then no longer return from the affected part of the bowel, which becomes enormously swollen with hæmorrhage and exudation of serum into its tissues, particularly at the lower bend. Moxon records an instance in which the coats of the bowel were three quarters of an inch thick in this position. The included part of the mesentery likewise becomes dark and thick with effused blood.

In other cases the influx of blood through the arteries is arrested as well as its escape by the veins, especially in the iliac form of intussusception where the diameter of the receiving layer is small. The inevitable result is gangrene; and, strange to say, this does not always lead to the death of the patient. The sloughing part of the bowel may be cast off, pass down the large intestine, and be discharged *per rectum*.*

* It generally appears as a single tube, with its mucous surface outwards; this perhaps includes both the entering and returning layers, the former having undergone inversion during the process of detachment; or it may be that the entering layer is cast off

Several instances have been recorded in which portions of bowel from twenty to forty inches long have been shed. In some cases surprisingly little disturbance is caused.

In a case which Dr Hare brought before the Pathological Society in 1862, when the patient died of phthisis three months after passing some inches of bowel, the line of union could only just be detected on the mucous surface by its shining appearance. On the serous surface there was "considerable puckering," but below the cicatrix a small pouch existed into which projected a little hollow cylinder, evidently a relic of the invaginated part.

Occasionally a local abscess may form, sometimes fæcal in character. Even when the expulsion of the separated portion of gut is followed by the recovery of the patient there is always a risk that the cicatrix may gradually contract. Moxon twice observed a fatal annular stricture of the ileum, with puckering of the mesentery, which probably arose from a former intussusception.

Age and sex.—Invagination is far more rare in adults than might be supposed from the comparatively numerous cases that Peacock, Brinton, Leichtenstern, Hutchinson, and Treves have collected. Wilks used to say that he had only seen one case in a grown-up person. In young children and infants the disease is frequent; and probably many cases are overlooked, so that it really occurs oftener than would appear from the published statistics. In fact, with rare exceptions, intestinal obstruction in an infant or child under ten is due to invagination.

Of the writer's twenty-four consecutive cases, twenty occurred in children and two in adults. The youngest patient was two months old, fourteen were less than twelve months, and the eight others were between one and seven years old. Both the adult patients were women between forty and fifty; but of the twenty-two children, fifteen were boys and only seven girls.

As Mr Treves has shown, the acute cases belong chiefly to childhood, while the chronic ones are more common in adult life.

Among children, males are far more liable to intussusception than females. Rilliet and Barthez met with twenty-two cases in boys to only three cases in girls. In Leichtenstern's collected cases of all ages, 285 occurred in males and 157 in females. In adults the difference between the sexes in this respect is far less marked. Thus in one year the Registrar-General's returns (according to Mr Gay) gave altogether 163 male to 93 female patients, while between thirty-five and forty-five the numbers were 55 men to 74 women.

Origin.—It is very doubtful whether sudden movements or violent efforts can cause invagination as Rilliet and Barthez supposed.

The immediate mechanism probably is that the contraction of the transverse muscular fibres of one portion of the bowel lengthens it, so as to push it into the succeeding portion.

It is possible by producing strong contraction of a limited portion of intestine with the faradic current, particularly when death by asphyxia has caused active peristalsis in an animal, to initiate the pathological process of invagination, and cause the contracted portion to push itself into the next

separately in the form of soft shreds, so that the inverted mass is constituted by what has been the returning layer only. A specimen was once sent up to Guy's Hospital, which consisted of twelve inches of intestine, and within its channel lay the appendix, opening on to its outer or mucous surface. Only an inch of it was small intestine. Consequently, if the intussusception was of the ordinary ileo-cæcal variety, the cast-off mass must have been derived mainly from the returning layer, the entering layer having doubtless broken down and been discharged separately.—C. H. F.

piece of gut. Moreover, on opening the abdomen of a rabbit or cat under chloroform, or recently killed by carbonic acid poisoning, one may often see more than one invagination form, and sometimes extricate itself again.

The special liability of the ileo-cæcal region to invagination is probably the result of two conditions: one, that the axis of the large bowel is nearly at right angles with that of the small intestine; the other, that the cæcum is much more fixed as well as much larger than the ileum.

Many instances have been recorded in which the starting-point of an intussusception has been a polypus hanging from the mucous membrane. This seems to have been caught by the contraction of the intestine below, and to have dragged downward the part to which it was attached. Dr Moxon met with a case in which an intussusception appeared to have been caused in a similar way by a diverticulum of the ileum; this must itself have first been inverted into the gut.*

Occasionally invagination occurs as a secondary complication of some other form of obstruction.

In the great majority of instances, however, we find no probable exciting cause of the invagination, nor can we say why it is comparatively common at an early age and rare after childhood.

In making an autopsy on a child, particularly after death from cerebral disease, we often find two, three, or more short intussusceptions in the small intestine. They are sometimes reversed or retrograde; the affected parts are not reddened, and there have been no intestinal symptoms. It has been supposed that such invaginations have occurred after death as a result of rigor mortis; but more probably they formed towards the end of life, under the stimulus of increasing asphyxia.

Symptoms.—In the progress of many cases two distinct periods can be recognised. But sometimes, particularly in infants, the former stage is so short that it is passed before the patient is seen.

The first complaint is of a paroxysmal *pain* in the abdomen, with sudden onset, without tenderness, and usually remittent. This pain is generally referred to the neighbourhood of the umbilicus. It is often so violent that the patient rolls about the bed in agony; and it is as a rule accompanied by *vomiting*. In one case the patient, a boy five years old, had for four months only two or three attacks of pain during each twenty-four hours; between them he appeared perfectly well.

At first the intestinal contents still pass through the invaginated part; the bowels act as usual, and the evacuations are natural.

The most important symptom of intussusception is the presence of a *tumour*. To detect it one may sometimes have to place the patient under chloroform, so as to relax the abdominal walls, and if the child is fat, the examination may even then yield no definite result; but doubt as to the existence of an intussusception may generally be cleared up by thorough palpation of the abdomen. The tumour which occurs in this disease is felt as a more or less cylindrical lump. The note yielded by percussion over its surface may be dull, or partially resonant, or scarcely distinguishable from that given by other parts of the abdomen. Its seat varies with the part of the bowel which is concerned. In the common ileo-cæcal variety it originally occupies the right iliac fossa. As more and more of

* The presence of the *Ascaris lumbricoides* in cases of intussusception has been noticed by several observers, and it seems not impossible that a worm might have its body grasped by the peristaltic movements of the intestine, so as to invert the part to which its head was attached.—C. H. F.

the intestine becomes involved, the tumour gradually changes its position. It moves across the abdomen, either at the level of the umbilicus or a little higher; having reached the left side it passes downwards into the left iliac fossa, and ultimately into the pelvis. Brinton says that it often forms an elongated mass, which lies horizontally just above the pubes. Another peculiarity of this tumour is that its size and form are liable to frequent changes. Perhaps hardly perceptible when one first lays one's hand upon the abdomen, it often hardens under manipulation, particularly if a paroxysm of pain should come on; and this muscular contraction is very characteristic.

As the disease advances, exploration through the anus affords further aid in diagnosis. The end of the invaginated intestine can often be felt with the finger, and ultimately it may even protrude from the anus, like a prolapsed rectum, for which it has often been mistaken. In the rectum the plug is felt as a soft, smooth tumour, in which the finger feels a dimple not unlike the os uteri in the cervix. It can be carried round between the returning and the receiving layers; and when withdrawn it is unstained by fæcal matter, but covered with blood-stained mucus.

Before the plug has reached the rectum fresh symptoms commonly develop themselves, belonging to the latter stage of the disease. As already remarked, the veins of the intussuscepted part of the bowel become obstructed, and it consequently becomes intensely congested and swollen; *hæmorrhage* occurs from its surface, and blood is discharged by stool, or a mixture of mucus and blood. The blood is red, never black, and is sometimes voided in large quantity. This symptom was present as often as vomiting, namely, in twenty-two cases out of twenty-four. Tenesmus is a frequent symptom after the plug has reached the rectum. At the same time the passage of fæces through the invaginated part is obstructed. *Vomiting* returns, there are no longer any remissions in the pain, the vomited matters become foul, and before long the patient in collapse expires. In the case already referred to of a boy who for four months had no symptoms but paroxysmal pain and tumour, death occurred within four days from the time when he began to have tenesmus and to pass blood and mucus. In infants these symptoms generally set in at the very commencement of the disease, and prove fatal on the second or third day.

Tympanites only occurs in the chronic cases; collapse is frequent in the acute ones, particularly in infants.

It has generally been supposed that the cases attended with hæmorrhage are those in which the invaginated mass is apt to slough away and to be discharged *per anum*, but Mr Hutchinson showed that in chronic cases of intussusception this very rarely occurs. Moreover, the changes which lead to the gangrene and shedding of the whole of the invaginated part of the bowel involve the complete arrest of the circulation of blood in it. Consequently one would not expect hæmorrhage from the bowels to occur while these changes are in progress, although the ulceration at the neck of the intussusception may lead to hæmorrhage when the sloughing part is being detached. Out of twenty cases collected by Peacock, in all of which the invaginated parts were shed and passed *per anum*, there was only one in which bleeding is said to have occurred, and in that one it ceased twelve or fourteen days before the expulsion of the gangrenous mass. In fact, the symptoms of those cases in which the invaginated gut sloughs can seldom be clearly divided into two stages; and often they

are undistinguishable from those of other kinds of intestinal obstruction until the intestinal slough is unexpectedly voided from the rectum.*

Among those cases which present the more characteristic symptoms of intussusception, the duration of the first period varies greatly. The above-cited case lasted four months, and similar instances have been recorded by others. A patient of Brinton's died during this stage of an intussusception which had lasted four months and a half. Such protracted cases seem to be always examples of the common ileo-cæcal variety of the affection. On the other hand, many cases, even of this variety, pass into the second stage from their very commencement; and when the small intestine is alone concerned the disease appears generally to take this course. Sometimes hæmorrhage and the other symptoms belonging to the second period set in and afterwards subside. Mr Sydney Jones met with a case in which they lasted for three days and then passed off, returning again seventeen days later, and then leading to a fatal termination.

An acute course is the rule, and is almost universal in children. Of our 24 cases, 18 lasted from twelve hours to five days; 2, nine or ten days; and only 4 were chronic, lasting three, nine, ten, and twelve weeks respectively. Two of these four cases were in adults; the two others and all the acute cases in infants or children under seven.

Dr Goodhart recorded a remarkably chronic case of invagination which lasted twenty-one months in a girl of nineteen. It was apparently occasioned by a polypus ('Clin. Trans.,' 1886).

Diagnosis.—This may either be perfectly easy or exceedingly difficult. The abdomen should always be carefully explored, by the hand laid on its surface, whenever a patient (particularly a child) complains of paroxysmal pain recurring without obvious cause. A tumour may perhaps be discovered, the form of which, and its seat, would point to an intussusception. The most likely alternative is an enlarged gland or an impacted mass of fæces. An enema (repeated if necessary) generally answers the second question; or we may feel the tumour harden under manipulation, or find that it shifts its place. A gall-stone or a cancerous nodule in the omentum might simulate an invagination in an adult.

In one instance a swelling, of somewhat cylindrical form, and only partially dull on percussion, was felt crossing the abdomen just above the umbilicus, and was at first thought to be due to intussusception. But the fixity and unchanged position of the tumour, and the absence of other symptoms, soon showed that it was not. The man died some months afterwards of tuberculous peritonitis, and the mass that had been felt was found to be the omentum indurated with caseous tubercle.

In those cases which are attended at an early stage with hæmorrhage from the bowels, there is danger of mistaking the disease for infantile dysentery. In an adult one must also remember cancer of the rectum. Doubt may often be removed by digital examination of the bowel; but even when the invaginated mass protrudes from the anus, it has been taken for prolapsus. Thus in a case recorded by Mr Hutchinson, a practitioner returned the bowel into the rectum, and fitted a cork pad to the anus to prevent its coming down again.

* Brinton says that when the invaginated mass sloughs off and is discharged, the date at which it commonly separates is the eighth day in cases limited to the small intestine, and that it is expelled on the tenth day. In ileo-cæcal cases the corresponding dates are, according to him, the fifteenth and twenty-second days. But these last figures are of little value because of the variable duration of the first stage in ileo-cæcal intussusceptions.

Prognosis.—Invagination is a dangerous disease. The bowel may occasionally extricate itself, particularly if aided by chloroform, as it did in three cases of our twenty-four. But far more frequently the invagination goes on increasing, and the symptoms pass from bad to worse.

Death happens by collapse in acute infantile cases, more gradually in adults by exhaustion from starvation, pain and hæmorrhage, or after a shorter or longer time by peritonitis. If the plug sloughs, death often results from profuse hæmorrhage or from perforation. In sixty-four cases collected by Dr Peacock ('Path. Trans.,' 1864), however, twenty-five recovered. This event belongs to chronic and adult cases, and is probably more rare than it used to be when cases were often unrecognised.

Treatment.—Our object in an early case of invagination is to replace the bowel in its natural position. This may be effected by several methods. In at least two cases introduction of a gum-elastic bougie into the rectum has succeeded, but it is applicable only when the invaginated mass lies in the rectum, and probably only in the rare cases when the part of the intestine concerned is limited to the lower part of the colon.

Copious rectal injections of warm water have frequently cured the disease. But a still more effectual measure appears to be inflation of the intestine with air. This procedure was many years ago (1838) recommended by Mr Gorham in the 'Guy's Hospital Reports' (1st series, vol. iii, p. 345); and he quoted three cases which had been successfully treated in this way in America. It is now frequently employed, and sometimes with the result of completely curing the disease. More often, perhaps, its success has been partial. The tumour has been reduced in size; or it has changed its position, returning towards the seat which it had occupied at an earlier period; or it has been made to disappear for a time, and all the other symptoms have subsided, but only to recur a few days later, and sometimes with a fatal termination.

The method of injecting air into the bowel is as follows. A plug of lint is wrapped round an ordinary pair of bellows, at a little distance from the nozzle, and fixed by adhesive plaster. The pipe is then introduced into the rectum, the plug being kept firmly pressed against the anus. Air is then slowly forced in until the abdomen becomes tense, the physician's hand being placed on the abdomen so as to feel any change in the tumour and regulate the distension.

Sometimes it has appeared advantageous to place the patient with the pelvis raised higher than the shoulders, while either air or water is being injected into the bowel.

Care must be taken that too much force is not used, for in the case of an infant five months old, which was treated by inflation in Guy's Hospital in 1873, the bowel gave way, air passed into the peritoneal cavity, and the child died in a few hours.

After successful inflation an adult patient should be kept under the influence of opium for several hours, and even infants may have minute doses repeated.

Inflation should be practised in every case of intussusception when the diagnosis is made early; but if the process of sloughing of the included bowel has once set in, the procedure would not only be useless, but would take away the last chance of the patient's recovery. Unfortunately, there are no definite symptoms which indicate that ulceration or gangrene has begun. Still, as Mr Hutchinson has remarked, the fact that in a parti-

cular case the tumour is moving onwards is always proof that the upper bend is not yet fixed. If inflation fails laparotomy should not be delayed.

If the case appears to be too late for inflation, the patient must be kept under the influence of opium, and allowed to drink as little as possible, in the hope that a natural cure by sloughing may be effected; or else abdominal section should be performed as a desperate chance and resection attempted. As far back as the year 1784 this was done successfully in Paris, in the case of a woman aged fifty.

In 1873 Mr Hutchinson advocated it in a paper read before the Royal Medical and Chirurgical Society. He had in 1871 performed the operation on a child, aged two years, who had an ileo-cæcal intussusception for a month, which was protruding from the anus. He opened the abdomen in the median line, introduced two or three fingers, and quickly drew out the invaginated mass at the wound. He then easily effected its reduction and returned it into the abdominal cavity. This was all done in two or three minutes, and the child recovered well.

In 1874 Dr Fagge was asked to see a woman who had suffered for a fortnight from paroxysmal abdominal pain. A tumour was readily detected which presented all the characters of an intussusception. She had but little sickness, and no constipation. Inflation with air was at once practised, and the swelling then receded from the left iliac fossa towards the right side of the abdomen, which had been its original seat. This clenched the diagnosis. Mr Howse was then asked to see the patient, and after repeating the injection of air without effect, he performed abdominal section. There was no difficulty in reducing the invagination, and the patient recovered well ('Med.-Chir. Tr.,' vol. lix).

The cases most suitable for this operation are the common ones of ileo-cæcal intussusception, in which the symptoms come on slowly. In such cases, as Hutchinson remarks, there is but little tendency to sloughing and detachment of the invaginated part, so that there is scarcely any prospect of a spontaneous cure, and the surgeon is unlikely to find the bowel in such a state as would make it impossible to proceed with the operation. Still the occasional difficulty of a complete reduction of the invagination is shown by an autopsy Dr Goodhart made on a child aged six months, who had died in less than twenty-four hours, yet neither by traction nor by squeezing could he replace the inverted cæcum.

The advantage of laparotomy in the case of invagination is that the surgeon knows where the obstruction is, and how to deal with it; the disadvantages are the early age of most patients, and the fatal collapse which is apt to attend a prolonged operation.

Out of 33 cases collected by Peyrot and Treves in which abdominal section was performed for intussusception, there were 24 deaths and 9 recoveries. In 10 of these cases (with 7 successful) the reduction was easily accomplished after the abdomen was opened; in the remaining 23 (with only 2 successful) it was difficult or impossible. Mr Barker, of University College, records 15 consecutive cases of abdominal section for invagination, and ten of them were successful. (See also Mr Knaggs' case in the '*Lancet*,' June, 1887, pp. 1126 and 1177, and Dr Lees' two cases, '*Clin. Trans.*,' 1898, p. 58.)

Of the writer's 24 cases, 10 were not mechanically treated; of these, 7 died and 3 recovered. Inflation was practised in 9 cases, with 4 deaths and 5 recoveries. In the remaining 5 cases, abdominal section was performed after inflation (except in one case) had failed, and all died—3 after successful reduction, 2 after resection.

INCARCERATION.—There is a condition causing Intestinal obstruction which deserves a separate place. It has a close resemblance anatomically to Strangulation, but differs in the important fact that while there is mechani-

cal detention of a coil of intestine at a certain point, and mechanical obstruction to the passage of its contents, there is no such pressure as to interfere with the circulation. It bears, in fact, the same relation to strangulation by a band, that an incarcerated does to a strangulated inguinal hernia. Like the latter, however, it may—and if unrelieved often does—end in embarrassment of the circulation in the imprisoned gut, and at last in gangrene.

The examples of this form of obstruction are cases in which a coil of gut falls over a band and, by its own weight, exerts a certain pressure, like a stocking hanging on a clothes-line; cases in which a coil passes through an opening too small to allow of its ready return, and too large to interfere with its vascular supply; and cases of internal hernia, particularly post-peritoneal hernia (jejunal, cæcal, mesocolic, or intermesenteric; v. *infra*, p. 426). In the same category may be also placed cases where adhesions have taken place, so that a piece of intestine is fettered in its peristaltic movements by what is virtually an obstruction, and yet there is no such pulling and squeezing as to obliterate the calibre.

Incarceration is most common in the small intestine, but may occur in the more moveable parts of the colon. It can scarcely affect either the duodenum or the rectum.

Besides incarcerated post-peritoneal hernia, which may lead to strangulation, as described on the next page, Albrecht drew attention to the fact that another form of obstruction at precisely the same point, the passage of the duodenum into the jejunum, sometimes occurs without a hernial sac.* It appears to depend on dilatation of a loaded stomach pressing on the gut as it crosses the vertebral column, while the mass of small intestines drag downwards the mesentery at its commencement. In Albrecht's case, he thought the trunk of the superior mesenteric artery supplied a further cause of obstruction by acting as a band. Such obstruction by pressure and traction, as in the cases referred to above (p. 414), may be complete and acute, with a rapid and fatal course ending in necrosis or perforation. Bäumler, to whom the writer is indebted for a knowledge of these cases, described one of his own in the 17th number of the 'Münchener med. Wochenschrift' for 1901, with reference to the two or three others on record.

STRANGULATION.—The remaining forms of intestinal obstruction differ from those last considered, in the anatomical fact that the constricting agent is independent of the constricted part of the bowel, and is invested with a distinct peritoneal covering. In all of them there is stoppage of the circulation beside obstruction.

a. In the *small intestine* internal strangulation forms perhaps a quarter or a third of all cases of obstruction. The exact nature of the constriction varies. It often is a *fibrous band* of greater or less length, attached at each end, but free in the rest of its course.† Such a band may be attached at both ends to the mesentery or intestine, or may be fixed at one end to the neck of an old hernial sac, or to the uterus, ovary, or Fallopian tube;

* P. A. Albrecht, "Über arterio-mesenterialen Darmverschluss an der Duodeno-jejunalgrenze, und seine ursächliche Beziehung zur Magenerweiterung" ('Virchow's Archiv,' 1899, p. 285).

† This was described by Mr Gay as the "solitary band," on account of there being usually only a single one present; but sometimes unfortunately there are more than one, and there is no reason, pathological or practical, for keeping the term.

or it may consist of a portion of the omentum, and pass to any of the structures that have been mentioned. Its mode of origin cannot always be ascertained, but it is often the direct result of cicatrization.

Gay records a case in which the transverse colon was punctured by a trocar in the operation of paracentesis abdominis; the patient recovered, but died many months afterwards of fever, when a firm and thick band, two inches long, was found, passing from the seat of the wound to the parietal peritoneum. He also quotes a case of Jobert de Lamballe's, in which a man who had been stabbed in the abdomen by a stiletto died some time afterwards from strangulation of the intestine by a band, which extended from the abdominal wall to the spot in the bowel that had been injured.

The writer has seen strangulation by cords following operation for hernia or pelvic inflammation in women. In men one of the most frequent causes of such a band is a cured case of typhlitis.

In many cases the constricting agent is a cord connected with a *diverticulum ilei*. Among fifteen cases of internal strangulation at Guy's Hospital five resulted from the presence of this relic of foetal life. The diverticulum is always situated on the side of the bowel furthest from the mesentery, and near the lower end of the ileum. According to Wilkinson King it is never more than from ten to twenty inches above the cæcum; but since his time a specimen has been placed in the museum of Guy's Hospital which was fifty-four inches from the valve. As Meckel long ago showed, this diverticulum is a relic of the omphalo-mesenteric duct, which passes in the embryo from the umbilical vesicle to the mid-gut. Before birth it ought to waste away entirely; but its intestinal end may persist and acquire adhesion to the mesentery or elsewhere. It is remarkable that this abnormality is scarcely ever found except in males; out of ten cases in which it was found at Guy's Hospital, only one occurred in a female subject.

Strangulation by a cord attached to the end of the *vermiform appendix* appears to be much less common. Duchaussoy states that females are more liable to it than males; but Leichtenstern's cases were 27 men to 13 women, while those of ileus from a diverticulum were 52 men to 14 women.

Strangulation may also be effected by the *pedicle* of an ovarian tumour, or by the edge of the *mesentery* of another coil of small intestine, which is hanging down into the pelvis, or by an adherent piece of *omentum*.

Internal hernia is another cause of strangulation of the small intestine. A remarkable and interesting form of this was first described by Treitz under the name of "retro-peritoneal" hernia, and a case was published by the writer in the 'Guy's Hosp. Rep.' for 1871. A pouch is formed at the back of the abdomen, a little to the left of the spine, passing backwards and downwards behind the curve formed by the inferior mesenteric artery and its left colic branch, and into this pouch the jejunum passes. The gut is sometimes incarcerated, sometimes strangulated. It is probable that jejunal post-peritoneal hernia was present in another case where Hilton performed an exploratory operation, and drew out a coil of intestine from an opening, apparently in the mesentery, just at the point where the jejunum became free from the spine. "Subœcal," "intersigmoid," and "intermesenteric" post-peritoneal pouches have been also observed, and have sometimes contained a coil of gut forming another variety of internal hernia.

A portion of the small intestine may pass through an *aperture* in the mesentery or in the omentum, and then swell and become irreducible,

exactly as it would under the femoral arch. Two cases of mesocolic hernia were shown at one of the early meetings of the Pathological Society by Dr Peacock in 1849.

It must be borne in mind that in any case of what appears to be internal strangulation the cause may be *external hernia*, too small to be discovered by manipulation. Hilton once opened the abdomen during life, and found an obturator hernia, the presence of which could not even then be detected in the thigh; and in a case at Guy's Hospital, in which an exploratory operation was performed, a very small knuckle of intestine was discovered in one femoral ring. Several of us had previously examined the groins most carefully, but had failed to detect any hernia.

A case of internal hernia was detected many years ago by the late Mr Cooper Forster, in which a knuckle of intestine became strangulated in a sac close to the upper border of the obturator membrane without passing through it.

β. The *large intestine* is very rarely constricted by a band, or strangulated in any of the ways just described. There are, however, two cases recorded in which the sigmoid flexure was obstructed by the mesentery of a coil of small intestine hanging into the pelvis, one in which the ascending colon was strangulated by a similar cause, one in which the same part of the bowel was constricted by the vermiform appendix, and a fifth in which the cæcum was strangulated by a diverticulum.

VOLVULUS.—A twist of the guts, the popular explanation of intestinal obstruction in general, is really a rare condition, particularly as a primary lesion. In its clinical aspect it resembles strangulation by a band.

a. When a knuckle of *small intestine* is strangulated by a band, or is "kinked" by adhesions, the condition is sometimes further complicated by the gut becoming twisted on its axis; or, when a loop has passed through an aperture or into a hernial sac, it may twist round the long diameter of the loop. Either condition may be called volvulus. Such secondary twisting of the guts makes the obstruction more complete, and may cause strangulation if it was not before present.

A primary volvulus of any kind is very rare in the small intestine. It always occurs on the proximal side if it complicates strangulated bands or pouches. Occasionally a loose cæcum or the sigmoid flexure is found twisted round a coil of small intestine so as to obstruct its calibre.

β. The more loosely attached parts of the *large intestine*—the cæcum and the sigmoid flexure—are most liable to primary volvulus. This usually consists in the rotation on its axis of a loop of the bowel, so that each limb is strangulated by the other one twisting round it. The ascending and descending parts of the loop are thus screwed up into a cord where they cross, their calibre is obstructed, and the circulation of blood in their walls is arrested. In such cases the affected portion of the intestine becomes enormously distended. We have more than once found the cæcum filling nearly half the abdomen, and reaching up into the right hypochondrium; while in another case the sigmoid flexure extended upwards, so as to come into contact with the diaphragm. Even when an attempt is made to untwist a volvulus after death, it sometimes springs back into its abnormal position with considerable force.

It is not quite clear how volvulus is brought about. Both the cæcum and the sigmoid flexure are not uncommonly found floating freely and con-

siderably enlarged in persons who have passed middle age, and perhaps such a condition is a necessary antecedent to the formation of a volvulus. When it has been the cause of death, the loop is always found full of fluid and intensely inflamed. The contents are doubtless chiefly secretion from its mucous surface, and this must have been poured out at an early stage, before the arrest of the circulation in the affected part. Bristowe suggested that enteritis is in fact the primary condition, and that the twisting occurs secondarily; he supposed that the portion of intestine becomes first inflamed and paralysed, and that, being heavy with accumulated contents, it is then pushed aside by the pressure of the active coils around it. But this explanation is insufficient to explain the way in which the neck of the volvulus is screwed up; and it is disproved by the exact limitation of the inflammation to the part of the bowel which is twisted. However the twist began, it is easy to understand how obstruction once produced leads to accumulation in the loop, and this to rapidly ingravescient strangulation.

Volvulus is most frequent in adult males; it very rarely occurs under puberty. The strangulation is early; the pain, vomiting, and other symptoms severe and acute. If seen before obstruction has led to general tympanites a circumscribed resonant tumour may be recognised.

In so severe and ingravescient a form of strangulation, the only chance lies in early recognition and immediate laparotomy.

A typical case occurred at Guy's Hospital in 1886. A young man, aged twenty-three, was admitted under the writer's care with a history of previous attacks of the same kind, and with recent symptoms of acute obstruction. The pain was intense, the vomiting severe, and it soon became stercoraceous; the meteorismus was enormous. The abdomen was opened on the fifth day, and the distended colon was seen, but the source of the obstruction could neither be reached nor redressed. After death the sigmoid flexure almost filled the abdomen, twisted on itself, and turned up, so that the returning end of the loop touched the liver. Even when the other viscera had been removed, it was found impossible to return it into its natural position.

We now pass to consider the local effect of obstruction, either alone or with strangulation superadded, upon the intestine above its seat, and the clinical effects produced by obstruction generally.

General anatomy of the obstructed gut.—Whatever the cause of obstruction may be, the bowel below its seat is pale, empty, and contracted, while that above it is distended. The jejunum or ileum may become dilated until it equals the colon in size; while the colon may reach a size which can only be described as monstrous. Moxon met with a case in which, after removal with its contents, the large intestine weighed nearly eight pounds; and some fæcal matter had previously escaped. One effect of the distension is that in the small intestine the mucous membrane becomes forced out between the layers of the mesentery, forming rounded pouches (as in two specimens shown by Dr Fagge to the Pathological Society in 1875), for which he proposed the name of "distension-diverticula." In the ileum the dilatation of the gut diminishes more or less rapidly as one passes upwards beyond the seat of obstruction; but the large intestine may be almost uniformly distended in its whole length; or there may be a great accumulation of fæcal matter in the cæcum, even when the obstruction is situated far below the arch of the colon—the result of paralysis of peristaltic movements, so that the contents gravitate on to the ileo-cæcal valve.

In chronic cases of obstruction, the muscular coat above becomes greatly

hypertrophied, forming a translucent grey layer, which gradually increases in thickness towards the affected spot. In the acute forms this is wanting, but all the coats may be swollen and injected, so that the bowel feels unnaturally thick and massive. The mucous membrane above the obstruction is apt to be ulcerated ("distension-ulcers"), in chronic cases, so that nearly the whole lining of the colon may be destroyed. Perforation is a not uncommon consequence; and sometimes a great part of the large intestine may be seen in a sloughing state, with its contents escaping from every part. Peritonitis is the necessary result of such a condition, unless the patient dies before it has time to develop. But inflammation of the serous coat is also apt to occur at an early stage in the more acute forms of the disease, independently of rupture.

In cases of strangulation the involved part of the bowel is deeply congested, and often almost black; it has lost its lustre, and is soft and readily torn. On being opened it is found full of dark blood, and the mucous membrane is swollen and softened. Corresponding with the constricting band or other pressure is an ulcerated line in the mucosa where the bowel is most liable to slough; and the same is observed in cases of invagination and volvulus.

In either case, more rapidly from the necrotic process of strangulation, more slowly from the ulceration of the distended bowel above the seat of obstruction, the result is perforation with septic peritonitis. In other cases, first local, and then general peritonitis is the result of extension of the suppuration to its serous covering of the ulcerated gut; or the final stage is reached, by septic absorption leading to pyæmic abscesses of liver and lungs.

Ætiology.—Obstruction of the bowels is, as we have seen, the result of such varied pathological conditions, that no general remarks as to its cause are applicable; and if we come to particular forms of ileus, our knowledge of the cause of gall-stones is as small as of the cause of cancer. Invagination is sometimes the result of a polypus, but in most cases there is no such exciting cause, nor do we know why it is so much more common in children than in adults.

This may, however, be said, that two forms of obstruction—that by bands and that by contractions—are both of them due to chronic precedent, and sometimes long precedent conditions, particularly local peritonitis, which causes adhesions, displacements of diverticula and of the appendix, contractions, distortions, long cords of omentum, pelvic traction, and all the other conditions which lead to these forms of obstruction. So that even in this often acute form of disease we find once more that such dangerous maladies seldom befall healthy bodies, and that the apparently sudden explosion is usually the result of long antecedent causes.

Clinical course and symptoms—(1) *of obstruction generally.*—In their clinical history, cases of intestinal obstruction divide themselves into two groups, those in which the symptoms begin acutely and run a rapid course, and those which are gradual in their onset and protracted in their course. The former correspond to the pathological condition of strangulation, the latter to that of obstruction. But the most chronic cases, if unrelieved, end with the supervention of acute symptoms; thus mere obstructions have their termination in septic processes, or in peritonitis from perforation.

As we found in diseases of the brain and cord, of the heart and lungs, symptoms become more and more similar towards the close, like paths which unite when they approach their goal.

There are three symptoms, however, which are common to all clinical forms of obstruction—acute, chronic, ingravescent, or remittent—which appear early and persist to the last; they are Constipation, Pain, and Vomiting.

Constipation may be said to be the fundamental symptom of obstruction of the bowels. It is generally absolute and immovable, whether by purgatives or injections, so long as the disease remains unrelieved.

A first enema may bring away fæces which had lain in the part of the bowel below the seat of the disease, but subsequent ones almost invariably return uncoloured. Even below the obstruction the bowel is as a rule paralysed. One would have supposed that obstruction high up would have allowed of the colon furnishing several stools before obstruction became manifest; but this is very seldom the case. In this, as in other respects, invagination is exceptional, for constipation is not always its first or even a later symptom.

It must, however, be remembered that liquid fæces may pass through a stricture which is capable of causing great distension, ulceration, and perforation of the part above it. In a case in which the writer had diagnosed annular stricture of the ascending colon, and had arranged the operation of colotomy, the passage of a large liquid motion of healthy fæces made us unwisely postpone interference. The patient died the same evening, and the cæcum was found enormously distended, ulcerated, and perforated.

Pain is always present, usually from the first, and is often of extreme severity. Brinton distinguished two kinds, of which one is a constant but moderate pain, corresponding more or less closely with the position of the obstructed part of the bowel, and often referred to the right iliac fossa. There may be some tenderness with it, but this is not usually marked. Probably this pain is always traceable either to congestion in the part of the intestine immediately affected, or to distension of that which lies above the obstruction; in the latter case it may spread over the whole abdomen.

The other kind of pain comes on in severe paroxysms, and is a kind of colic. It results from spasmodic contraction of the bowel above the seat of disease. This pain corresponds with that of strangulated hernia, which is usually referred to the umbilicus.

Vomiting occurs sooner or later in all forms of intestinal obstruction. Its severity partly depends upon the seat and nature of the affection, but is greatly increased if the patient takes much liquid into his stomach. Brinton found that in animals in which he ligatured the intestine, the quantity of fluid which they drank had more influence than anything else in determining not only the amount of sickness, but also the rapidity with which death ensued.

The vomit consists first of the gastric contents, then of bilious fluid from the duodenum, and afterwards of matters derived from the small intestine down to the obstructed part, possibly sometimes from the cæcum. These almost always constitute a thin yellow liquid, which, if the seat of disease be high in the jejunum, may have merely a disgusting mawkish odour, but when the obstruction is lower the smell becomes decidedly fæcal at last. The stench is often so powerful as to fill the room in which the patient lies,

and to be almost insupportable by those about him. The cause of this "stercoraceous vomiting" has been matter of some discussion. At first it was believed to result from a reversal of the peristaltic movements of the intestine. Brinton denied the occurrence of antiperistalsis; he showed that even though the muscular coats of the bowel should continue to contract in the ordinary way, there would be a tendency to the formation of a double current in its interior, one downwards along its walls, and another upwards in its axis. Engelmann has since demonstrated the occurrence of reverse contractions in the intestines of animals, in which he had opened the peritoneal cavity; it may frequently be observed in the physiological laboratory. In all probability antiperistalsis occurs in the human subject also; but it seems clear that the direction of peristalsis is not, as a rule, reversed; for, if it were, the part immediately above the seat of obstruction would not become distended; and mercury or castor oil, when given by the mouth shortly before the patient's death, would not be found to have passed down to the seat of obstruction.

One result of stercoraceous vomiting is that, during the distressing efforts which accompany it, some of the semi-fæcal matter may be sucked into the air-passages. This occurrence has been more than once noticed in the *post-mortem* room; when pressure was made towards the cut surfaces of the bronchial tubes, these gave exit to little yellow cylinders which certainly must have entered them during life; and in one case of strangulated hernia, in which death had arisen from peritonitis after relief of the obstruction, the lungs contained patches of gangrenous pneumonia, which had a yellow colour in the centre, due, probably, to fæcal staining.

The above symptoms of constipation, pain, and vomiting are common to both acute and chronic cases of obstruction of the bowels. The other symptoms of these two groups of cases present wide differences; and it will be convenient to deal first with the chronic.

(2) *Chronic obstruction*—of which rectal stricture is the type—is characterised by the slow or imperfect development of the symptoms already mentioned, and by some in addition.

Constipation is sometimes incomplete, scanty fæcal evacuations occurring from time to time. Indeed, for several weeks or even months before obstruction definitely sets in the patient often has considerable and increasing difficulty in procuring an action of the bowels; or he may have repeated attacks of partial obstruction before the one which at length completely closes the intestine. Even when the constipation is absolute, it is wonderful how life is prolonged; thus the late Mr Cooper Forster recorded an instance in which there was not any action of the bowels for eighty-eight days.

Pain is a variable symptom in chronic obstruction. It is seldom or never absent, but often comes on very late in the progress of the case, and when earlier present it is not constant but paroxysmal and colic-like.

Vomiting is often absent in cases of this kind for some days or even weeks after the cessation of fæcal evacuations. Ultimately it comes on, and lasts until death or relief.

Distension of the abdomen is often extreme: this depends partly on the absence of vomiting, but chiefly on the fact that chronic obstruction in most cases affects the large intestine, and the lower rather than the proximal part of this, so that the cæcum (and often more or less of the colon), as well as the small intestines, is full of retained fæces and gas.

Visible peristalsis is almost decisive of the obstruction being chronic. If in such a case the abdomen be examined during a paroxysm of pain, the peristaltic movements of the intestine can be seen through the parietes; irregular elevations arise here and there, and are succeeded by depressions, or appear to travel from one part of the surface to another. For the production of this symptom it seems to be essential that the coats of the bowel should have undergone hypertrophy; at any rate, it is seldom or never observed in cases of recent obstruction. Peristaltic movements are more often seen in the small than in the large intestine, but they may undoubtedly occur in the latter. Even during the intervals between the paroxysms of pain the position of the different parts of the bowel is often distinctly visible through the abdominal parietes; and it is to be particularly noted that the transverse colon, when distended, does not continue to lie horizontally across the upper part of the abdomen, but bends downwards, so as to form a broad loop, lying vertically and (with the dilated ascending and descending colon) filling the whole front of the abdomen. This position is, indeed, forced on it by its distension, which makes it too long to occupy its normal position. The coils of ileum, under similar circumstances, are generally arranged transversely; and since these coils are often quite as broad as the transverse colon would be under normal conditions, the uppermost one, lying horizontally just below the ribs, has often been mistaken for large intestine. On the other hand, in the case of volvulus of the sigmoid flexure referred to above (p. 428), we mistook the huge parallel and vertical limbs of the C for the loop of the transverse colon just mentioned.

The general symptoms presented by a patient suffering from chronic obstruction of the bowels are sometimes exceedingly slight, if the treatment is judiciously managed. His pulse may be natural; there may be no fever; he may sleep well at night; the tongue may be clean, and food may be relished. The urine also is passed in ordinary amount.

The patient, however, is always on the brink of a precipice. At any moment acute symptoms may supervene which may destroy life in a single day. This may depend on the obstructing external force tightening its grasp on the bowel so as to occlude its blood-vessels as well as its canal; but more often it depends upon paralytic distension of the bowel above the obstruction, leading to fæcal enteritis and ulceration, and this without symptoms; so that one can never say when perforation may take place.

It once happened to me to send up from my out-patient room into the medical ward a woman who had cancer of the lower part of the sigmoid flexure. She had been ill for three months. I went up to see her later in the afternoon, and she appeared to be perfectly comfortable, had a quiet pulse, and presented no urgent symptoms whatever. I therefore thought that the question of colotomy might be safely deferred until the following day. In the evening the nurse was turning her over to give her an enema when she suddenly expired. About a pint of liquid fæces was found in the abdominal cavity, which had escaped from an opening in the sigmoid flexure. There were also several large sloughing patches in the peritoneal covering of the cæcum, as well as its mucous lining.—C. H. F.

It would be difficult to fix an average duration for cases of chronic obstruction; and if one could be fixed, it would be of no practical value.

(3) The course and symptoms of *acute obstruction*—of which strangulated hernia is the type—must now be considered. They differ much from those of the chronic forms of the disease. The *constipation* is always absolute; any fæces that may be brought away by an enema come from below the occluded part of the intestine. Even flatus is no longer passed.

Severe *pain* is seldom absent from the first, and *vomiting* is early and severe.

The abdomen rapidly becomes distended, but seldom so much as in cases of stricture of the colon. It does not fill up the left lumbar or both lumbar regions, nor the epigastrium, but rather the central part of abdomen, where the coils of small intestine lie. Peristaltic movements are rarely, if ever, to be seen, but the form of the intestinal coils may sometimes be visible.

The most characteristic feature of cases of acute obstruction is the early development of *collapse*. The face becomes sunken, with pinched cheeks and dark circles round the eyes; the extremities are covered with a cold sweat; the pulse is very rapid and small, the voice is high-pitched and feeble or whispering. The patient, however, often retains perfect consciousness, and is able to lift himself up in bed almost until the last.

So close may be the resemblance between the condition of a man suffering from acute obstruction and that which occurs in Asiatic cholera, that during the epidemic of 1866 a case at Guy's Hospital was actually supposed to be one of cholera with retention of the rice-water evacuations, until after death the disease proved to be strangulation of the intestine.

In acute obstruction of the bowels *the patient passes little urine*, and the secretion may be altogether suppressed. The late Dr G. H. Barlow, who first noticed this symptom, supposed that it depended on diminution of the area for absorption of fluid, and indicated that the seat of disease was high up in the jejunum. Subsequently Dr Habershon attributed oliguria to the frequent vomiting which occurs when the upper part of the gut is strangulated; and Dr Brinton argued that the mucous membrane above the seat of obstruction becomes a secretory rather than an absorbing surface. Both these writers admitted the fact that when the urine is suppressed, the disease is high up in the bowel; but, as stated in the chapter on cholera (vol. i, p. 274), there is reason to believe that this symptom is really one of the phenomena of collapse, and due to temporary anæmia of the kidneys. When collapse is present, anuria occurs in all forms of intestinal obstruction, whatever their seat, including strangulated hernia.

Diagnosis.—The general diagnosis of obstruction, like its course and symptoms, is best studied under the heads of chronic cases, *i. e.* cases which begin gradually and last long, and acute cases—that is, cases which begin with severe symptoms and end soon. We have to distinguish the former from constipation with atony of the colon, and the latter from acute peritonitis. In both cases our next task is to determine, as near as may be, the exact seat and the pathological nature of the obstruction.

(1) The first question in cases of *chronic obstruction* arises from its resemblance to impaction of the large bowel with indurated fæces; or we may say that remedial obstruction by scybala with a torpid state of the colon is one form of obstruction by impaction. At first we can form no positive diagnosis, and must be guided by the effect of treatment and the further development of the case. In all cases of obstinate constipation we must remember the possibility of the cause being a mechanical one, which cannot be overcome, but may easily be aggravated, by provoking increased peristalsis.

Next, the chronic obstruction may be due to gradual compression

of the gut from without, by an abscess, or tumour, a pregnant uterus, or an ovarian cyst.

Again, there are two forms of obstruction affecting the coats of the intestines—contraction and stricture,—both of which run a chronic course. We have seen that contractions occur chiefly in the small, and strictures in the large intestine; the distinction between them is therefore to be based mainly upon the points indicating distension of the ileum or of the colon. In the first case the fulness is chiefly in the middle line and the middle and lower parts of the abdomen; in the latter, in the sides and epigastrium. There is also a difference in the shape of the distended abdomen, according as the arch of the colon is below or above the seat of obstruction. In the former case the belly is rounded, projecting well forwards, but with comparatively little fulness of the lateral and lumbar regions. In the latter and more frequent case it is more broad, and if the hand be placed on the patient's loins as he lies in bed, a feeling of resistance is experienced which is wanting when the small intestine is alone distended.

Occasionally a tumour can be discovered on palpation; and this, or the fact that the pain is referred definitely to one particular spot, may suggest the exact seat of the mischief. Moreover, all the signs which show that disease of the large bowel is the cause of intestinal obstruction really indicate more than this, and point to the conclusion that the affected spot is situated below (to the left of) the hepatic flexure of the colon. So far as these signs are concerned, chronic obstruction of the cæcum, or even of the ascending colon, is undistinguishable clinically from that of the lower part of the small intestine; for the transverse colon does not in either case become distended. We have seen, however, that the right side of the colon is but very little liable to disease in comparison with the descending colon, sigmoid flexure, and rectum.

With a view to treatment, it is of great importance to make out whether the seat of obstruction is below the splenic flexure of the transverse colon or above it. In some cases one may perhaps determine this by placing each hand under one of the patient's loins, and by raising them so as to poise the two sides of the abdomen. If a fulness is felt in the right loin which is wanting in the left, it may perhaps be inferred that the ascending colon is distended. Another procedure which may throw light upon the question at issue is the slow injection of a large quantity of fluid into the rectum. Brinton believed that this was capable of yielding trustworthy conclusions as to the seat of the disease. According to this writer, when a pint is the most that can be thrown up, the obstruction is at the upper part of the rectum; a pint and a half, two pints, three pints, correspond respectively with different segments of the sigmoid flexure. The descending and transverse colon can be made to receive larger but more irregular quantities. In one case, in which it was evident that a stricture occupied the upper part of the ascending colon, nine pints of fluid were always found to be the most that could be injected. But it is to be borne in mind that a stricture may be pervious to fluid injected from below, although faecal matter may be unable to pass through it from above. Thus, in one of the cases recorded in the 'Guy's Hospital Reports' (ser. 3, vol. xiv), in which there was a mass of disease in the sigmoid flexure, just above the pelvis, four pints of warm water were injected, of which only a small portion returned, the rest having doubtless passed upwards through the affected part of the bowel.

It is well known that often in cases of cancer of the lower part of the rectum the fæces are narrow; but this sign is far from constant, and the form of the fæces depends much more upon the anal orifice than on anything else. In any case narrow motions cannot possibly be produced by stricture above the rectum, for they would be moulded into shape lower down, and the exploring finger would reach nearly as high as this.

Digital examination of the rectum must never be omitted in any case of chronic (nor indeed of acute) obstruction.*

If plenty of fæces are found in the rectum, they should be removed by an enema, and the case is settled. But small particles of fæcal matter give only a delusive hope that the obstruction is overcome. When the rectum is "ballooned," *i. e.* spacious and empty, Mr. Bryant believes that it is a good sign of stricture not far above the anus. Probably it depends on a stricture allowing gas to pass, but not fæces. More often we find the rectum flattened back against the sacrum by the distended coils of intestine above. Now and then a stricture or invagination may be felt, or the finger may discover a gall-stone or a cancerous mesenteric gland.

Introducing a long tube into the rectum appears to be useless for purposes of diagnosis; it may catch against a fold of mucous membrane, or against the seat of obstruction, and in either case may bend upon itself so as to appear to pass up much higher than it really does.

Inflation of the rectum and colon by air, and particularly hydrogen gas, has lately been much used in America as a means of diagnosing the seat of an obstruction, and apparently with good effect. The plan is in principle like that of distending the stomach referred to above (p. 328).

Beside the determination of the seat of obstruction, the pathological character of the disease is also a question for diagnosis, and one which it would in some cases be extremely important to settle, if only this were possible. We have seen that both strictures and "contractions" may be either simple or cancerous, and sometimes the discovery of a definite tumour shows that the latter is the case in a particular instance. But it is doubtful whether there is any other way of proving it. Cancer affecting the bowel is by no means confined to persons of advanced age. Among nineteen cases collected for the paper above quoted in the 'Guy's Hospital Reports,' six occurred in patients less than thirty-five years old. Moreover malignant disease may be present in a person who looks well and has a florid countenance. On the other hand, if the patient's health is broken down, his "malignant" look may be the result of pain and sickness.

A history of tuberculous disease, or of long-standing abdominal symptoms, in a patient under thirty, is in favour of contraction; one of gradually but recently increasing constipation in a patient over forty is strongly in favour of cancerous stricture.

(2) *Mixed cases.*—It must not be forgotten that some forms of intestinal obstruction have an acute early period, which is followed by subsidence of the severe symptoms. This is sometimes the case with Invagination; and may even be observed with bands or hernia if the patient is not fed, and is kept well under opium.

* It may even be well when the finger fails to reach the diseased spot, cautiously to pass the whole hand into the bowel, the patient being under the influence of chloroform. This procedure was advocated by Prof. Simon, of Heidelberg, and in England by the late Mr Maunder. It needs a small hand and much care and patience, but is sometimes an aid to diagnosis in this and other diseases of the abdomen.

Cases of stricture and of contraction often begin with slight disturbance, pain, constipation, and loss of appetite, which last for weeks or recur at intervals for a longer period; and then, when the calibre of the bowel has been gradually narrowed by chronic local peritonitis and adhesions, it is suddenly occluded, perhaps by grape-skins or orange-pulp; or an annular stricture of the colon is suddenly blocked by a hard pellet of fæces; and thus acute symptoms really depend on a chronic obstruction.

Moreover all cases of obstruction, if unrelieved, go on, sooner or later, to the same condition of peritonitis, collapse, and death from perforation or septic infection. All roads converge as they approach their goal. We saw it was true for organic disease of the brain, the spinal cord, and the heart, and it is no less true for ileus, that exact diagnosis is only possible in the earlier stages.

(3) *Acute cases*.—As with the chronic forms of obstruction, we must first ask if there is any other condition which simulates ileus.

Strangulated *hernia* is the same pathological condition, whether it occur within or without the abdominal cavity. The only question is whether symptoms of acute obstruction can be explained by finding a hernial tumour within reach. The same remark applies to incarcerated hernia.

There is no doubt that *peritonitis* is often undistinguishable from acute intestinal obstruction. The main symptoms—pain and tenderness, obstinate constipation, and tympanitic distension of the abdomen, together with more or less close approach to collapse—are common to both diseases. Moreover acute obstruction naturally ends in general peritonitis, and acute peritonitis produces obstruction by paralysing the intestinal peristalsis.

In fact, it must be admitted that there is no single constant and trustworthy diagnostic mark between the two conditions. If seen from the first there need rarely be much hesitation; but when peritonitis is fully developed we can only form a judgment as to whether it is primary or secondary to obstruction by weighing probabilities derived from the age, sex, and previous medical history of the patient, and from the onset and course of the present attack.

The inflammatory disease of the cæcal appendix, which we have above described as *Typhlitis* (p. 402), may often simulate obstruction, and so also may abdominal *tuberculosis*. The only safeguard is to bear both possibilities in mind in every case of supposed peritonitis from obstruction.

Another disease which might be mistaken for acute intestinal obstruction is the sudden impaction of a *gall-stone* for the first time, so that there would be no history of a previous attack. The pain and vomiting so caused are often accompanied by constipation, and it may be several hours before icterus comes on and resolves the doubt.

Supposing we have satisfied ourselves as to the presence of acute mechanical obstruction, we have next to ask how far we can discriminate between the various conditions to which it may be due.

In the first place, it must be remembered that there is no form of obstruction which may not present itself with acute symptoms. Even in cases of stricture of the large intestine, constipation sometimes sets in suddenly, and quickly leads to vomiting and collapse. The explanation appears to be that, in such cases, the bowel is occluded, not directly by the disease of its walls, but indirectly by œdema, by muscular spasm, or by the bending over of the portion of intestine above, when it has gradually become overloaded with its contents. The proof is that, as in Hilton's case

mentioned below (p. 439), when colotomy is performed, fæces soon begin again to pass by the natural passage. At the autopsy, too, one can often pass the finger through a stricture which had caused obstinate constipation during life. We must therefore remember that acute obstruction is the most frequent termination of chronic obstruction.

No doubt, when acute symptoms are present in a case of stricture or contraction, they have been preceded by slighter ones for some days or even weeks. But in hospital practice it may be impossible to elicit this fact when the patient is admitted at an advanced stage of the disease. Thus there may be scarcely anything to distinguish the case from one of primary acute obstruction. The point of most importance, indicating the latter diagnosis, is the absolute suddenness of the symptoms, the patient having been well until the moment when he was attacked.

With respect to the *seat* of acute obstruction, it is in the majority of cases the small rather than the large intestine, and the lower rather than the higher part of the small intestine. Hence the utmost attention should be directed, both in exploration under chloroform and after laparotomy, to the right iliac region.

When the obstruction is high up, as in cases of post-peritoneal hernia or impaction of a gall-stone in the jejunum, vomiting is early and severe, and the epigastric region is flattened or depressed. When it is near the end of the ileum, the hypogastric and umbilical regions are swollen, and the flanks are flat or hollow.

We have seen that diminution in the amount of urine passed probably arises from excessive vomiting or collapse rather than from diminution of the area of absorption; and therefore it is no trustworthy indication of the seat of obstruction being high up. In early cases, which are free from peritonitis, if considerable increase of *indican* in the urine is ascertained, it is probable, according to Jaffé, that the obstruction is in the small rather than the large intestine; the explanation being that the indol formed in pancreatic digestion is reabsorbed and secreted as indican when the contents of the intestine are not rapidly passed on. The writer has, however, noticed increase of indican in the urine in cases of cancer of the colon and in obstinate constipation without obstruction, so that its excess can be regarded as only an evidence of checked peristalsis.

The pathological conditions, which may be regarded as the causes of primary acute obstruction of the bowels, fall under four principal heads:

1. *Internal strangulation*, affecting most often the ileum.
2. *Volvulus*, affecting the ileum, cæcum, or sigmoid flexure.
3. *Impaction of a gall-stone*, affecting the small intestine only.
4. *Intussusception*, affecting the ileum, and most often its lower end.

Unfortunately little can be said as regards diagnosis between these several affections.

We may, however, remember that obstruction by a band connected with a diverticulum scarcely ever occurs except in males, and chiefly in patients under twenty years of age.

In the case of volvulus, previous constipation is the rule; and there is not the delay in the occurrence of sickness, which occurs in the other forms of obstruction of the large intestine—on the contrary, all the symptoms develop themselves with remarkable rapidity; the abdomen becomes quickly distended to the utmost, and death may occur within three or four days. Volvulus is rare in women and in children.

Obstruction from traction by adhesions is most common in adult women as the result of pelvic inflammation, and next in those who have suffered from tubercle in childhood.

Impaction of a gall-stone in the small intestine is to be distinguished, if at all, by the fact that it occurs chiefly in elderly women. In one case of acute obstruction, which proved to be due to this cause, the writer felt the calculus *per rectum* as a smooth round tumour, very moveable, and only just touched by the finger. It was, of course, felt through two thicknesses of gut.

We can in most cases recognise invagination by the hæmorrhage and the presence of a characteristic tumour, either felt through the abdominal walls or reached by the rectum. Without these symptoms we should probably overlook it in an adult, though in a child its probability would perhaps lead us right.

Prognosis.—The prognosis of obstruction of the bowels is always grave, even with successful diagnosis and judicious treatment. The natural processes of cure, by sloughing off an invagination, or by a constricting band softening sooner than the gut it strangles, are so dangerous that we cannot trust to them.

The duration of chronic obstruction may be measured by weeks. That of acute obstruction is subject to considerable variations. Death has been recorded as occurring within thirty-three hours; but such cases are very rare, life being almost always prolonged for three or four days, and generally beyond the first week. A case of obscure abdominal disease which terminates fatally within two days is much more likely to be perforation of the stomach or intestine than obstruction of the bowels.

The fact is that prognosis in this terrible disease depends entirely upon treatment.

Treatment.—(1) In cases of *chronic obstruction* the patient has often been taking powerful purgatives for a considerable time before he seeks medical advice. All such drugs should be at once omitted, and enemata only used, for they are frequently of great service. In almost every instance there is at first a possibility that the disease may be merely an impaction of the contents of the intestine; and even when organic disease of the coats of the bowel is present, the systematic administration of enemata not infrequently removes the symptoms for a time. The constipation is, indeed, almost certain to return after a shorter or longer interval; and even if it should once more yield to similar treatment, the period at length arrives when the bowels remain occluded in spite of all that can be done. Purgative medicine must never be prescribed when peristaltic movements can be felt or seen, when the abdomen is becoming rapidly distended, or when grave symptoms appear. But small doses of olive oil are admissible when ordinary aperient drugs ought not to be given. (℞ Ol. Olivæ ʒj, Liq. Potassæ ℥iv, Aq. Ment. Pip. ad ʒj.)

The next question is that of diagnosis and the operation of colotomy. If this appears contra-indicated, we can only fall back upon opium, and must not give it sparingly; from half a grain to a grain may be given every four hours, or its equivalent in laudanum, or subcutaneous injection of morphia may be preferred.

If the patient should suffer greatly from flatulence, we may venture

perhaps to puncture the intestine with a fine trocar. The late Mr Stocker long ago used to perform this operation, which is, indeed, only the application to human patients of a common and apparently harmless operation in the case of cattle with a distended paunch. The introduction of a sharp instrument into the bowel appears to act as a powerful stimulus, for fæcal evacuations have often been passed soon afterwards. That this is not only due to the escape of the gas is evident from a remarkable case of the writer's in which puncturing the abdomen in five places with a grooved needle, although neither fluid nor gas was withdrawn, led to complete subsidence of all the symptoms of intestinal obstruction, and to the prolongation of the patient's life for several months. In chronic cases there is little fear of the escape of the contents of the bowel into the peritoneal cavity, for the mucous membrane protrudes into the minute aperture so as to close it. Although, however, puncture is safer than might be supposed, it cannot be said to be more than a doubtful procedure.

As soon as it is evident that a case of chronic obstruction will not yield to medical treatment, the question arises whether the bowel should be opened at some point above the obstruction, so as to establish a fæcal fistula. If we can clearly make out that the seat of disease is below the descending colon, the left loin and the splenic flexure should be selected for the operation. On the other hand, if there be a doubt whether the obstacle lies below the descending colon, but none that it lies below the ascending colon, the hepatic flexure must of course be opened. In either case the operation of colotomy is, as a rule, successful; the peritoneum need not be wounded, and the patient's life is often prolonged for months or even for two or three years. This operation should not be delayed after the administration of purgatives and enemata has been discontinued.

As a remarkable instance of colotomy may be mentioned a case of Hilton's recorded in the 'Guy's Hospital Reports,' 1868, p. 219. There had been absolute constipation for twenty-eight days, but four days after the operation fæces began to pass through the rectum, and in a short time the wound in the loin closed. The symptoms, however, afterwards returned, and it was necessary to reopen the colon. After this a dilator was introduced twice a day with the object of keeping open the fistulous passage; but in spite of this, it again became occluded. The patient, however, who was himself a medical man, began to regain his strength, and resumed his professional duties, being able to visit thirty patients in the day without too great fatigue. Ultimately he died of abscess in the left iliac fossa, which communicated with the interior of the hip-joint. The cause of the obstruction appeared, *post mortem*, to be a simple puckering of the coats of the sigmoid flexure at one spot.

In August, 1885, the writer saw an elderly lady with obstruction of the bowels and enormous tympanites, on whom an eminent surgeon had refused to operate. There was evidence of the seat of obstruction being above the rectum and below the splenic flexure of the colon; it was probably an annular stricture. There being no evidence of disease elsewhere, left lumbar colotomy was advised and submitted to. The patient has been frequently seen since, and is now (November, 1890) still living and able to go about. I believe she has since died.

The following case illustrates the uncertainty of even what seems to be a tolerably certain diagnosis:—A patient under the writer's care in Philip Ward (1889) had every symptom of stricture of the sigmoid flexure. Colotomy was advised, but refused until, after several days' obstruction, he seemed at the point of death. He then consented to the operation, which was performed by Mr Golding-Bird with complete success. The patient recovered his strength and returned to his work. Several months afterwards he again came into the hospital with chronic Bright's disease, the bowels acting regularly, sometimes through the rectum, more often through the splenic flexure of the colon. He died of uræmia, and *post mortem* there was not a trace of stricture, cicatrix, adhesions, twist, or contraction in the whole length of the intestine.

Mr Bryant brought before the International Medical Congress of Copenhagen, in 1884, a remarkable series of 82 cases of colotomy performed by

himself. Of these, 60 were for cancerous and 19 for non-cancerous stricture, 2 for external obstruction, and 1 for volvulus of the sigmoid flexure. As the result, 26 of the patients operated on died within a month; but for the rest the operation gave marked relief, and prolonged life—in 16 patients for between one and six months, in 8 between six and twelve months, in 12 from one to five years, and in 5 for longer periods. The remaining 8 were Guy's Hospital patients, who left the wards convalescent some weeks after the operation, but were not traced subsequently. At the same meeting Mr Henry Morris contributed 23 cases, of whom 14 recovered from the operation, and Professor Studsgaard 20, of whom 17 survived for periods varying from a month to several years.

Whether lumbar or inguinal colotomy is the preferable operation has still to be settled by the results of more extended experience. Having seen excellent results from the lumbar operation, and some unfortunate cases of inguinal colotomy, the present writer feels more confidence in the former. But in some very uncertain cases (always the least satisfactory in results) the inguinal operation is almost compulsory, and in this, as in other cases, we must remember that whatever special method of operation a skilful surgeon adopts is sure in his practised hands to be followed by better results than another to which he is less accustomed.*

When chronic obstruction by stricture or contraction affects the small intestine, or even the cæcum or ascending colon, the only feasible operation, short of laparotomy, is to open any distended coil that presents itself in the groin, as advised by Nélaton. This procedure has occasionally succeeded, but is seldom justifiable. Its only merit is its rapidity, and the immediate relief afforded.

When a surgeon prefers an inguinal to a lumbar colotomy, he is sometimes tempted to "complete the operation" by removing the tumour. But this appears to be a mistake. It prolongs the operation and the condition of anæsthesia, and therefore adds to the danger. If the distension above the stricture is relieved, and the patient got to bed again as quickly as possible under the influence of opium and brandy or strychnine, a good result is far more probable; and a week or a fortnight later the tumour may be removed with good hope of success.

(2) In the treatment of *acute obstruction*, the first point to be considered is whether one should recommend the operation of opening the abdomen (*laparotomy*), and searching for the band or other constricting agent, with the object of mechanically releasing the bowel. The analogy of herniotomy is all in favour of such a course, if only one can be sure of the nature of the disease; and every pathologist has met with cases in which, as soon as the body was opened after death, a band was seen that could have been divided without difficulty. Moreover, in many cases this operation has undoubtedly saved the patient's life.

By carefully selecting for abdominal section cases which presented in the most typical form the symptoms of acute obstruction, one could probably make it a matter almost of certainty that the operation should not be undertaken in a case of stricture or even of contraction. Volvuli and the different

* The reader interested in the history of the subject will find the case for lumbar colotomy set forth by Mr Bryant in his Bradshaw Lecture before the College of Surgeons, and that for the inguinal operation in Mr Harrison Cripps's work on 'Diseases of the Rectum and Anus.' See also the debate at the meeting of the British Medical Association at Birmingham, reported in the 'Brit. Med. Journ.' for October 11th, 1890.

varieties of internal strangulation would all be fair objects for operation, although no doubt some of them would be far more favourable than others.

One of the most striking operations on record was performed on a case of volvulus by Dr Senn, of Milwaukee, U.S.A., in October, 1889. The patient was an elderly man who had suffered from intestinal obstruction for a week. The sigmoid flexure was found twisted round its mesentery and distended with liquid fæces. It was untwisted, opened and emptied, sewn up again, and its mesentery "taken up" by a few stitches, so as to shorten it and prevent future volvuli. The wound was closed antiseptically and the patient made a good recovery ('Philadelphia Medical News,' Nov. 30th, 1889). Another equally remarkable case of successful abdominal section for the same condition (volvulus of the sigmoid flexure) had previously been published by Dr H. E. Clark, of Glasgow, in the 'Lancet' for Oct. 20th, 1883 (vol. ii, p. 678).

Another question is, whether cases of obstruction are sure, if left to themselves, to terminate fatally. Dr Fagge once searched the records of *post-mortem* examinations at Guy's Hospital very carefully to find any case of internal strangulation of the intestine in which recovery had taken place, and the patient had subsequently died of some other disease. The only instance discovered was that of a man admitted with constipation and stercoreous vomiting under the care of Dr Addison, who recovered and afterwards died of phthisis. But in that case, although there were two loose bridles, either of which might have strangulated the bowel, there was also adhesion of a coil of small intestine, and the appendix cæci was firmly bound down. Anatomically, therefore, the case probably belonged to the class of "contractions" rather than to that of internal strangulation, and no operation could have relieved it. When there is mechanical constriction of a part of the bowel, it is conceivable for the intestine to be disengaged by the peristaltic movements of the portion above, or for the constricting band, which is often very thin, to give way. When the cause of strangulation is a band attached to a diverticulum of the ileum, this is often found softened at the time of the patient's death; but unfortunately it is apt to give way just where it joins the bowel, so that the result would be a fatal extravasation of fæces.

The writer once saw a band so soft that it broke as soon as touched, and the constricted gut was not damaged beyond the likelihood of recovery; but in this case the peritonitis which was present would probably have proved fatal.

No doubt recovery sometimes takes place in cases which have presented symptoms of internal strangulation. (See on this point Mr Bryant's lecture, 'Brit. Med. Journ.,' 1884, vol. ii, p. 1128.) But the diagnosis may have been wrong.

A student under my care in 1874 was seized with abdominal pain one Sunday morning, and soon afterwards had slight vomiting. There was absolute constipation, although he took several doses of purgatives. On Tuesday his abdomen became distended, the coils of intestine being visible through the parietes. On Wednesday, when seen for the first time, his face was shrunk and his extremities were cold. The sickness was severe; and on Thursday afternoon he ejected a large quantity of brownish liquid, which evidently came in part from the intestine, though it did not actually possess a fæcal odour. That night, however, he passed an offensive stool containing numerous scybala; his urine at once became copious, and all his threatening symptoms quickly passed off. As he got better, one could feel an indurated mass in the region of the cæcum, and he had a relapse of short duration, in which there was an increase of tenderness in this part of the abdomen. There is little doubt that the case was one of typhlitis, and not of mechanical obstruction.—C. H. F.

A course which may be regarded as intermediate between opening the abdomen and trusting entirely to the efforts of nature is what Mr Hutchinson has termed *abdominal taxis*, *i. e.* inverting and shaking the patient, inject-

ing large enemata from a height, and kneading the bowels, in the hope of mechanically reducing the displacement. This is undoubtedly sometimes followed by immediate recovery, as in the following case in which the writer called in Mr Hutchinson's skill for the purpose of applying taxis.

A tall, thin man of thirty-seven, who had had his first attack of gout, was taken, on coming home to dinner, with severe pain in the abdomen. His family doctor got him to bed; but when I was sent for later in the evening I found him still pale, with weak fluttering pulse and nausea, although the pain had subsided. In spite of a grain of opium he passed a very bad night, and next morning the pain was worse again, with vomiting, but no tympanites or pyrexia. Rectum empty. No motion or flatus passed since first attack. On the third day he seemed relieved, being still under opium and taking scanty liquid diet. Urine scanty and high-coloured. No sign of biliary or renal calculus. On the fourth day an enema, very properly ordered by his doctor, was followed by renewed and severe pain, and this increased towards evening with absolute constipation. The next morning, though a full dose of opium had procured sleep, he was no better, and there was no sign of relief. I thought it probably a case of incarcerated small intestine. An eminent surgeon was then called in who I knew would not wish to open the abdomen, for which the patient in my judgment was quite unfit; and we agreed to give chloroform and try the effect of enemata with raised pelvis. The same evening therefore, a little flatus but no fæces having been passed during the day, the patient was put under anæsthesia, and, after manipulating the abdomen, warm water from a height of seven feet was sent into the rectum, while the pelvis was raised high above the shoulders. The result was the welcome sound of borborygmi, a strong fæcal odour, and the passage of a large motion mixed with the water injected. This was soon after followed by another passage of fæces, and he made a good recovery from this time. He continued well for several years, and died, without any return of obstruction, of chronic Bright's disease at the age of forty-six.

Sir Thomas Watson relates the case of a lady who observed that the hands of two other medical men who were seeing her with him in consultation were heavy as they manipulated her abdomen; she fancied that their pressure had displaced something within, and almost directly afterwards she passed a liquid motion. The procedure of kneading the abdominal parietes was adopted in a case which Dr Fagge had seen in consultation a few days before, and within a very short time the bowels acted.

Striking as such cases appear, it is quite possible that even in recent cases manipulation in the dark may do more harm than good. The same remark applies to faradisation of the abdomen, and to the almost forgotten treatment by making the patient swallow metallic mercury, which has been recently revived in Germany. This last method is surely irrational as well as dangerous.

If we do not feel justified in recommending operation, there can be no doubt of the advantage derived from the free use of *opium*—a dose of one grain given every four, three, or two hours, according to circumstances. It often affords marvellous relief, peristalsis is stopped, pain is relieved, and sickness may entirely pass off for the time: at the worst the patient's death is freed from the suffering which would otherwise have attended it; and if spontaneous subsidence of the disease be possible, this chance is greatly increased. Under no circumstances should a single dose of purgative medicine be prescribed, even at the very onset of what may prove to be a case of intestinal obstruction.

Much relief is afforded by Kussmaul's plan of emptying the stomach by a siphon-tube and feeding the patient by the rectum. The extremities should be kept warm, and the patient should lie on his back with the pelvis raised higher than the shoulders.

Summary.—It is, no doubt, theoretically possible to treat every case of acute intestinal obstruction by abdominal section, followed by operative

release of the gut, or, when this is impossible, by resection of the strangulated or invaginated portion or of the volvulus. If this attempt failed, a permanent fistula between the gut above and below the obstruction might be established, or at worst an artificial anus. But practically the difficulties would be often insuperable and the dangers great. We must remember that the actual mortality after laparotomy is much higher than is indicated by the published cases, and that the unavoidable, and also the unexpected, delays in the course of opening the abdomen and searching for the seat of obstruction are far greater than might be supposed beforehand.

It is, on the other hand, only fair to exclude the mortality after operation before the prevalence of Listerian principles, and even before surgeons had acquired that mastery of aseptic practice in abdominal cases which the present generation have reached. Things can now be done with reasonable hope of safety, which before were in the highest degree hazardous. Apart from the danger of septic infection and of peritonitis, which we may hope to see still further reduced by more absolute cleanliness, there is always the danger of shock, of cold, and of interference with the heart and respiration, which protracted abdominal operations bring. These may be met by warm rooms, heated tables, bandaging the limbs, subcutaneous injection of strychnia, rectal injections of brandy and beef-tea.

Another serious difficulty is the great distension of the bowels which we have seen to be so constant in the later, and not infrequent in the earlier stages of ileus. It is often desirable to empty the stomach by a siphon-tube, if it can be safely done, and even to puncture a distended coil of gut.*

In cases of internal obstruction, the seat and nature of which can be diagnosed with some approach to certainty, the results of an exploratory operation with a defined object are likely to justify the risk. But where, as unfortunately is often the case, we have no such approach to certainty, it is possible that the expectant treatment—emptying the stomach and using repeated enemata, followed by starvation and the full exhibition of opium—affords the patient as good a chance of life as laparotomy.

We must, however, remember that the want of power on the physician's part to form a diagnosis frequently leads to so long delay, that it is vain to expect a good result from the most skilful surgical treatment. Here, as elsewhere, improved practice must wait upon improved diagnosis.

Meantime, as a practical guide to those who have not seen many of these deeply interesting but most difficult and responsible cases, the following remarks are submitted as being in accordance with our present knowledge and experience. If they err on the side of caution, that is perhaps the more easy for the reader to correct.

(1) In some cases, perhaps in most, we are able to make a more or less probable diagnosis by careful study of the symptoms, of the previous history of the patient (when it is obtainable and trustworthy), and of the probabilities which result from the natural history of the several forms of obstruction, their frequency and their incidence on certain periods of life.

We are then justified in adopting a definite plan of treatment in accordance with our belief. We treat invagination by inflation, opium, and warmth with stimulants; with adults, and in some cases with children,

* The late Mr Greig Smith, of Bristol, advocated systematic enterostomy or tapping the distended intestines, and his views were shared by some experienced surgeons who took part in the debate ('Lancet,' March 12th, 1892, p. 582).

abdominal section is indicated should these measures fail. We treat impaction of fæces by belladonna and enemata, impaction of a gall-stone by patience, or possibly by laparotomy and incision. We treat stricture of the colon by colotomy, and internal strangulated hernia by abdominal section.

(2) There remain a large number of cases in which obstipation, pain, vomiting, and distension prove the existence of a mechanical obstruction, but leave quite uncertain its nature, sometimes even its seat and whether it is originally acute or chronic.

In these difficult cases the writer believes that some lives would be saved by putting the patient under chloroform, manipulating again with a view to diagnosis, shaking and inverting the trunk (supposing the case to be recent), and giving large and repeated clysters. If this fail, we have two courses open. One is to wash out the stomach, keep the patient fully under opium, with the pelvis elevated, and kept alive with as little food and drink taken as possible.

The other, and in many cases the wisely bolder plan is to perform an exploratory operation in these anxious circumstances. This, when decided on, should be done as early as possible, before tympanites has become marked, before the patient has been exhausted by pain and abstinence, and before the symptoms are obscured by opium. The regions of hernia, the sigmoid flexure, and, above all, the right iliac fossa and groin are the parts to be carefully examined; and if nothing can be found wrong by the exploring hand, it is probably often safest to close the wound after washing out and draining the peritoneum.

If there is reason to fear, or to be sure, that peritonitis is already present, that is no reason for rejecting an operation; for abdominal section is the only successful treatment of peritonitis itself.

There is no doubt that the experience of twenty years has greatly altered the opinion of the profession with respect to operation, for since Dr Fagge wrote the first edition of this chapter in 1882, aseptic surgery and improvement of the important details of procedure have made justifiable, and even imperative, operations which would then have been rash. The one thing still needed is more certain and accurate diagnosis; and this depends on increasing knowledge of the results of examination of these cases after death, which in hospital or in private should never be neglected.

At present, opening the abdomen is far from being free from risk, and at present diagnosis is often painfully at fault; but there is reason to hope that every year will improve medical diagnosis and surgical skill. The best cases the writer has seen have been those where a correct conclusion was arrived at of what was the seat and probable nature of the lesion; the worst have been where the laparotomy was wholly exploratory.

The other conditions which have seemed to him most unfavourable have been—(1) the early age of the patients in cases of invagination, and their advanced age in cases of malignant stricture; (2) delay in recognising the serious character of the case, and further delay in accepting the advice to submit to an operation; (3) the patient being corpulent, or otherwise in an unhealthy state for bearing a serious operation; (4) protraction of the operation owing to absence of diagnosis, or to delay in putting the patient under anæsthetics, or to disturbance of the respiration, or to unforeseen obstacles and complications, or to an attempt to do more than what is immediately urgent.

Cases.—*a.* The following are the results of eighteen consecutive cases of internal strangulation or other severe and acute obstruction (excluding herniæ, strictures, and impactions) collected by Dr Shaw, from the clinical and *post-mortem* records of Guy's Hospital, ten years ago.

Eight patients out of the eighteen recovered, after periods of complete obstipation varying from five to eight days in six cases, extending to twelve and fourteen days in the other two. In all these vomiting and other serious symptoms were present, the cases were acute in course, and were believed to have their seat in the small intestine. All were treated by opium or by opium and belladonna with the frequent use of enemata, and without an operation.

Five patients died without operation, under the same general treatment as was used in the preceding cases. In all these cases examination after death showed obstruction to be in the small intestine:—namely, a kink of the gut, fatal on the thirteenth day; strangulation by a band in two cases, one fatal on the eighth day, the other protracted for four weeks; volvulus of the small intestine in two cases, fatal on the eighth and ninth days.

Five of the patients died after an operation. In all the bowel was found strangulated, in three by a fibrous band, and in one by the appendix cæci. In three the constricting band was divided and the gut liberated; in two a fistulous opening was made in the bowel above the constriction. The operation took place on the seventh, eighth, ninth, or eleventh day; later, no doubt, than the surgeon would have wished.

b. The following results refer to fifty cases of obstruction (acute and chronic) which have been under the writer's personal observation. Thirty-seven of the patients were male and thirteen female.

There were eight cases of obstruction due to invagination, six in children between 4 and 7, and two in adults. Twenty cases of strangulation (or contraction with adhesions) occurred at various ages, from 5½ years to 46; two of volvulus at 23 and 31; two of impacted gall-stone in women at 60 and 78; four of probable fæcal impaction in men at or above 50; and fourteen of cancerous stricture of the colon or rectum at 24, 38, 41, 44, and the other ten between 60 and 78.

In the cases of stricture of the large intestine the treatment adopted was colotomy, with relief in every case but one, which was fatal on the second day. The five cases of impaction with distension and visible peristalsis recovered under belladonna and enemata: both cases of volvulus died, one, an infant of eighteen months, after laparotomy; both cases of impacted gall-stone recovered without operation; and one case of invagination died after laparotomy, the others recovering without.

The remaining twenty cases were acute obstruction of the small intestine due to bands, internal hernia, adhesions, or contractions: of these, one recovered after abdominal taxis, and four under expectant treatment by starvation and opium; six died under the latter treatment, and five after laparotomy; while three recovered from obstruction by traction (the result in each case of a precedent strangulated hernia), after an exploratory operation from the groin.

WORMS*

(CESTOID AND NEMATOID ENTOZOA)

“They have made wormsmeat of me.”—*Romeo and Juliet*.

- Tapeworms*—*Anatomy and transformations*—*Tænia solium*—its distinctive characters and distribution—its relation to *Cysticercus cellulosæ* of pork—*Tænia mediocanellata*—its specific distinctions—its relation to the *cysticercus* of beef—its geographical distribution—*Bothriocephalus latus*—its characters and distribution—its probable origin—*Symptoms of the presence of tapeworms generally*—*Preventive and curative treatment*.
- Bladderworms*—*Relation of cystic or hydatid forms to the strobila form*—*Hydatids infesting the human body: Cysticercus echinococcus*.
- Round-worms*—*Ascaris lumbricoides*—its anatomy—natural history—distribution—symptoms, diagnosis, and treatment—*Threadworms*—anatomy—symptoms and treatment—*Trichocephalus dispar*—*Eustrongylus gigas*.
- Sclerostomum duodenale*—its discovery—anatomy—habits—*Ancemia caused by its presence*—*Treatment*.
- Trichina spiralis*—its discovery—anatomy, transmigrations, and encapsulation—*Trichiniasis*—its symptoms, diagnosis, treatment, and prevention.
- Filaria sanguinis hominis*—its discovery in the blood—its transmigrations—its relations to chyluria and elephas.
- Dracunculus*—*Distomum*—*Bilharzia hæmatobia*—its habitat—relation to hæmaturia—*Echinorhynchus*.

THE condition of parasitism has already come before us in the case of microscopic plants (vol. i, p. 19), and in the case of microscopic animal-

* The English term worm has been very variously applied (1) to fabulous dragons and serpents (in the ‘Faery Queen’ and in ‘Paradise Lost’), including the slowworm or blind-worm—also a reptile, (2) to earthworms (*lumbrici*) and other Annelida, (3) to maggots or larvæ of dipterous insects, (4) to the class Vermes of Linnæus, and to the scarcely less heterogeneous group of the same name recognised by most modern zoologists, and lastly (5) to the parasitic Entozoa, which form the subject of this chapter and belong to widely separated zoological groups, Cestoda and Nematoda. The popular confusion between the poisonous worm of the Nile, the worms which Mercurio expected to feed, and the earthworms which appear in digging, has involved “mawworms” also, as Pope’s epitaph on “the egregious Mr John Moore” illustrates:

“O learned Friend of Abchurch Lane,
Who sett’s our Entrails free,
Vain is thy art, thy Powder vain,
Since Worms must eat even thee.”

cules (*ibid.*, p. 397), and we found that many, perhaps most, of them are not pure (or to use modern phrase, "obligatory") parasites, but pass part of their life outside their host. This is the rule in the case of the larger and more familiar, but less dangerous, parasites with which the present chapter deals. All whose life history has been completely ascertained spend only part of their life as parasites, and the study of this phase of their natural history is the more important from a prophylactic point of view. Beside these two varieties of constant and occasional parasitism, there is a third group of animals (as of plants) which usually live independently, and only by accident assume a parasitic condition in man. Of these "casual" parasites, as Dr Payne happily calls them, we have examples in the occasional presence of the grubs of *Diptera* in the human body, or of *Fungi* in the lungs. These are so rare as to be little more than curiosities.

The human alimentary canal, like that of other animals, often harbours animals which are known as intestinal worms. They belong to two quite distinct zoological groups, *Cestoidea* and *Nematoidea*, which agree in little but their parasitic habit.

There are other vermiform parasites inhabiting the muscles and the blood, which may also conveniently be described in this chapter, particularly as it is probable that they all inhabit the intestines at some period of their lives. The hydatid forms of *Cestoidea* will also be dealt with here, since they cannot be regarded separately, either zoologically or practically.

The most common human *tæniæ* (ἐλμινθες πλατεῖαι) have been known from classical times, as were also the *lumbrici intestinales* (στρογγύλαι), and the threadworms, or *ascarides* (ὑσκάριδες). Hydatids were regarded as pathological cystic formations, not as parasites, until after the middle of the nineteenth century. *Trichocephalus* was described in Baillie's 'Morbid Anatomy.' The other parasitic worms were only discovered within the last fifty years.

The *Cestoidea*, or "tapeworms," are flat, riband-like structures, not really vermiform, for each is a compound organism made up of a number of joints, arranged in a line. The compound *strobila* is a colony consisting of the parent "head," and of coherent products of gemmation. There is no alimentary canal. Each "joint" has a double sexual apparatus, both male and female, and lives after detachment from the colony.

The *Nematoidea*, or "round-worms," have long cylindrical bodies; they have an alimentary canal, the sexes are distinct, and they undergo but slight changes of form after leaving the egg.

CESTOID WORMS.*—It was once debated whether the "individual" is the whole "tapeworm," or each separate joint. There is a so-called "head," provided with suckers and hooks, which fastens itself to the intestinal wall; and, as Leuckart remarked, the movements of the creature are transmitted from one joint to another, so that large portions of it shorten or lengthen together. Nevertheless each segment of a tapeworm is more truly an "individual." It is one of a colony, and yet separate, as are the components of a compound salpa or of a polyp colony, or sponge, or the individual flowers of a tree. It is preposterous to apply the term to the *unconnected* products of gemmation, some living and some dead; yet this is

* *Synonyms.*—*Tæniada*—Tapeworms, including Flat-worms and Bladderworms or Hydatids.—*Fr.* Vers cestoides, Kystes hydatiques.—*Germ.* Bandwürmer und Blasenwürmer.

logically required by adopting the definition of an individual as "the total product of a single fertilised ovum until fertilisation is repeated." The fact is that the conception of an individual was originally derived from human consciousness, and is applicable to many of the lower classes of animals and to most plants.

By zoologists the tapeworm as a whole is called a *strobila*; the "head" before budding is a *scolex* or budding larva, and the segments are known as *proglottides*.

In the course of their development the Cestoidea pass through an extraordinary series of changes, which bring them into pathological importance at various points; and it will be convenient to give a general account of these before describing individual species of tapeworms.

Let us suppose that a tapeworm is present in the intestine of a man, or of a carnivorous animal, a dog or a cat. Its joints or proglottides nearest the head are very small, and appear almost structureless; for the development of new and imperfect proglottides is constantly going on at this part of the strobila, and these, as they are formed, separate the scolex or head further and further from those which preceded them. Thus the greater the distance from the head, the older and more developed are the joints; and towards the distal end of the tapeworm they are fully developed, with an hermaphrodite sexual apparatus. Here the ova are formed and impregnated. In each is presently formed an embryo, a globular body, enclosed in a thick capsule, and provided with six curved hooks arranged in pairs.

The ova are not discharged from the proglottis through the genital canal or "vagina;" they are, indeed, too large to pass through it, and remain *in situ* until the proglottis ruptures, and allows of their escape. We have seen that the tapeworm is constantly forming new joints near its "head." At its other end the mature joints are as constantly being cast off. Thus every proglottis in turn is pushed on, until by the time that its ova with their embryos are fully developed, it reaches the distal extremity of the tapeworm, and in its turn becomes detached. When this has happened, it is either discharged with the fæces of its host, or wanders out of the rectum by its own movements; or possibly may be ruptured while still within the intestine, so that its ova are expelled with the fæces.

Having reached the external world, the proglottides creep about for a time. If warmth and moisture favour them, they remain alive and active for some days. Leuckart supposed they might crawl up a blade of grass, and with this be swallowed by some herbivorous animal; but more probably they die and become disintegrated, or the growth of the multitude of ova within causes them to burst. In either case the ova escape and become scattered in all directions. Some perhaps are carried into streams and ponds, others on to the stems or leaves of plants, where they retain life for several days under favourable circumstances. The immense majority no doubt perish, but from time to time an ovum meets with the condition necessary for its further development.

This is being swallowed by some particular species of animal, generally herbivorous, either in the water which it drinks, or in the food which it eats. As soon as the now ripe egg reaches the stomach of this animal, in future to be its "host," its membranous shell is dissolved by the action of the gastric juice, and the six-hooked embryo (*larva* or *scolex* or *proscœlex*) is thus set free. It immediately starts upon its destined migration. By

means of its six hooks it bores through the walls of the stomach or intestine of its host. In this way it is very likely to enter some radicle of the portal vein; and then by the current of the circulation is carried to the liver. Less frequently it burrows through the tissues until it has reached some other organ far from its starting-point. Probably, however, in most cases of hydatids found in other regions, they first reached the liver, and thence invaded more distant parts through the hepatic veins, the lungs, and the systemic arteries. In any case, the scolex ultimately comes to rest in the liver, or lungs, or brain, or some other part of its host, and there undergoes a new phase in its development. It begins to grow, it loses its six hooks, and becomes surrounded by a layer of exudation from the tissues of its host. Within four or five days from the time when a rabbit was made to swallow the ova of a tapeworm, Leuckart found on killing it, that its liver and lungs were studded with minute white grains, like miliary tubercles, each containing a tapeworm embryo.

The embryo still goes on increasing in size, and when it has reached a diameter of 0.6 to 0.8 mm., it becomes hollow by its central cavity being filled with a transparent watery fluid. From this time it presents the character of a globular vesicle, cyst, or bladder. It was recognised in this condition long before its relation to its tapeworm parent was understood, and was formerly known as a bladder-worm, or hydatid. The *echinococcus* of the human liver, the *cysticercus* of "measly" pork, and the *cœnurus* found in the brain of sheep, are all examples of the hydatid or cystic stage of a tapeworm. Each of them is surrounded by fibrous tissue derived from the tissues of their host, in which they lie free and unattached; and it is from the blood-vessels of this capsule that they derive their nourishment.

Next, the growing bladder-worm begins to show a projection from one part of its inner surface, and this gradually increases in size and becomes pear-shaped. Soon four suckers make their appearance, and a circle of numerous minute hooks; the deutoscœlex or daughter-cyst thus acquires a resemblance to the head of the parent tapeworm, and after a time a kind of constriction or neck is developed. In strictness, however, the likeness is not to a tapeworm head as it is, but as it would appear if it were withdrawn into its body, like the finger of a glove turned inside out.

The bladder-worm may long remain quiescent in the tissues of its host. It may die there, and its remains shrivel up until only a caseous or calcareous relic is left. But if its host should die first it may be set free, and then its transformations begin again. The condition required for its further development is that it should be swallowed by a carnivorous animal, along with the tissues in which it is embedded. Thus the *cysticercus* hydatid or "measle" of pigs is swallowed by dogs; and the *echinococcus* hydatid of sheep by dogs or wolves.

When it has reached the alimentary canal of a new host, the bladder-worm enters on a fresh series of changes, which end by its conversion into a tapeworm. In the first place, the parts which have been described as resembling a tapeworm's head and neck, now turn themselves inside out. Thus, instead of being suspended in the interior of the bladderworm, the head and neck come to project from its exterior. The original sac or "caudal vesicle" speedily disappears, except a small remnant attached to the neck. The head and neck resist the solvent action of the gastric juice, and pass on into the intestine. There they become attached and begin a new linear process of growth. Within a few days transverse lines show

themselves on the neck, and the segments thus formed increase in size and multiply by gemmation, until in the course of some weeks a jointed tapeworm or *strobila* is developed. The cycle of changes undergone by the parasite is thus completed; we have arrived at the point from which we started.

In certain species some of the steps in this series of transformations deviate slightly from the account just given of tapeworms; but all the Cestoidea, without exception, require two different hosts for the completion of their existence. The one host in which the ovum assumes the form of a bladder-worm may be either herbivorous or carnivorous, but is most often the former. The other, in which the scolex becomes a tapeworm, must always be carnivorous, since the larva can only be swallowed with the animal tissues of its first host, and so gain entrance to the alimentary canal of its second one.

As may well be supposed, this marvellous series of transmigrations has only been discovered by patient investigations continued through many years. As early as 1769, Pallas noted the close resemblance between human tapeworms and the *Cysticercus tenuicollis*; but even in 1845 it was supposed that hydatids were tapeworms which had "strayed" into a wrong animal, and had consequently become dropsical and degenerated. In 1851, however, Küchenmeister administered the *Cysticercus pisiformis* of the rabbit to dogs, and succeeded in rearing in their intestines the *Tænia serrata*; he also gave the *Cysticercus fasciolaris* of the rat or mouse to cats, and found that it became developed into the *Tænia crassicollis*. In 1853 the converse experiments were performed by the same observer; proglottides of the *Tænia cœnurus* of the dog were given to sheep and lambs, with the result that bladder-worms (*cœnuri*) were found in their brains after the appearance of the symptoms of "staggers," a disease long known to be caused by this parasite. During the last fifty years similar investigations have been prosecuted with many different species, and the result is that we have now complete experimental proof of the relations and mode of development of many of the Cestoidea.

There are seven or eight species of tapeworm which have been known to occur in the human alimentary canal, but of these only three are anything but curiosities.

1. *Tænia solium* (Linn.) was formerly believed to be the most common human tapeworm. When fully developed the colony measures from seven to ten feet in length, or possibly more.

The so-called tapeworm colony, as above explained, is not like an earthworm or maggot, but an individual produced by gemmation, like an anthozoic coral. The proximal end, or "head," attached to the intestine of its host, is the scolex, derived from a cystic form, and only indirectly from an egg. It is not so large as a pin's head, and is provided with four discoid suckers, and with a small central proboscis. Between the proboscis, or *rostellum*, and the suckers is a circle of about twenty-six hooks, ranged with their points outwards. They are of two sizes, and are large and small alternately. The "head" is often black, from the presence of pigment. The "neck" measures an inch in length. The joints are at first very small and broader than they are long. They gradually increase in breadth and still more in length, so that at about a yard from the head

they are square, and towards the distal end of the strobila their length is considerably greater than their breadth. The ripe proglottides measure about half an inch long by a quarter of an inch in breadth. They have often been compared to melon-seeds, and are, in fact, not unlike them.* The "genital pores" or orifices of the sexual apparatus are placed in a little papilla, which is easily recognised on one of the free edges, more or less regularly on the alternate sides of each successive joint. The "uterus" consists of a central passage, running in the length of the proglottis, and giving off at right angles from seven to ten branches on each side, which again have complex secondary branches. A good way of observing their characters is to compress a tapeworm-joint (*proglottis*) slightly between two plates of glass, and hold it up to the light.

The eggs are nearly globular, and measure 0.03 mm. in diameter. They have a shell or capsule of considerable thickness, with rod-shaped projections, which closely cover its surface, and give it the aspect of being marked with minute radiating lines.

The cystic form or "bladder-worm" which forms a previous stage in the development of the *Tænia solium* is called the *Cysticercus cellulosæ* (*sc. telæ*). It is found chiefly in the pig, occasionally in the monkey, the dog, and some other animals, including man himself. In the pig it occurs in the liver or the brain, but more often in the connective tissue between the fasciculi of the voluntary muscles. Pork so affected is said to be "measly" (*i. e.* spotted). Its relation to the *tænia* is indicated by exact similarity of the scolex to the head of that tapeworm, and has been proved by experiment. Van Beneden, Leuckart, and others administered proglottides of *T. solium* to pigs, and the result was that the flesh of the animal became full of cysticerci. Two months and a half are required for the full development of the cysticercus. From observations made by Stich it is probable that its life within the tissues (at least in man) is limited to from three to five years; he found that at the end of such a period cysticerci in the subcutaneous tissues, which had been plainly felt through the integument, became flaccid and shrank away until they disappeared.

The converse experiment to that of feeding pigs with the proglottides of the *tænia* is the administration of cysticerci to human beings. This has been occasionally done, the subjects being either criminals condemned to death, or persons who volunteered for the purpose. Küchenmeister gave a criminal twenty cysticerci on two occasions, once four months, and again two months and a half before his execution; nineteen tapeworms were afterwards found in his intestines. A young man who of his own accord swallowed four cysticerci in Leuckart's presence began, for the first time in his life, to pass proglottides three months and a half afterwards, and a month later took a dose of kousso, with the result that he passed two tapeworms, each about two yards long.

The name *Tænia solium*,† given to this parasite by Linnæus, was meant to imply that it occurred singly in the intestine; and the same notion is expressed by the French title, *ver solitaire*. But this is a mistake; two

* Hence they were called *cucurbitæ*, and the worm *Tænia cucurbitina*. According to Küchenmeister, the Arabs call the complaint "Chabb-al-kar," *i. e.* pumpkin-seed. Aristotle makes the same comparison: "The flat mawworm produces bodies like colocynth seeds, by which physicians detect its presence."—"Hist. An.," lib. v, cap. xix.

† It should have been written *T. sola*. The same notion was expressed by the specific name *T. solitaria* (Bradley). Of other synonyms, *T. cucurbitina* (Pallas) referred to the proglottides, *T. dentata* (Gmelin) and *T. armata* (Brera) to the hooklets. It is often called the "armed tapeworm," the mawworm, or the "pork tapeworm."

or three are occasionally present at once, and cases are recorded in which many have been passed by a single patient.

This *tænia* is more common in adults than in children, and it has been often found in butchers and in cooks, as might be expected, since it is derived from measly pork. Out of Europe it has been observed in India, Algiers, and America. The duration of life of *T. solium* was estimated by Leuckart at from ten to twelve years; but Cobbold mentions the case of a patient who was infested with it for sixteen years.

The cystic form, *Cysticercus cellulosæ*, is also sometimes found in the human subject, and this is the only known instance in which man is liable to both the larval or cystic, and the mature or strobila form of a cestode entozoon. As a bladder-worm the parasite has been most often observed in the eye and in the brain; but it is also present in the muscles and subcutaneous tissue, where it is apt to escape notice. It is often solitary, or present only in small numbers; but in Stich's case, at Berlin, more than three hundred could be felt through the skin.

The writer only twice saw cysticercus of the subcutaneous tissue; in one case the cysts were very numerous, and were found after death under the skin of an old man.

In the other, a young and healthy man was found to have a multitude of small painless tumours under the skin of the trunk and limbs; one of them only was inconvenient—in the lower eyelid. This was removed and found to be a cysticercus. The case is described and figured in the 'British Journ. of Dermatology' for 1892 (vol. iv, p. 366).

A person who has a tapeworm in the intestine does not derive cysticerci directly from its ova; they must first pass through the stomach, where their shells are removed by the action of the gastric juice.*

Very few of those who have a tapeworm become affected with cysticerci; although von Graefe found that among thirteen patients with cysticercus in the eye five had tapeworms.

2. *Tænia mediocanellata*† (Küchenmeister) was long confounded with *T. solium*. The "head" in *T. mediocanellata* is flattened, and has neither proboscis nor circle of hooks; there is often much pigment deposited round the four suckers. The strobila is much broader than that of *T. solium*, so that each segment has a square form; and its water-vascular system is more simple in its arrangement. It is often called the "unarmed" tapeworm, to distinguish it from *T. solium*, which is armed with its circle of hooks.

The strobila also presents peculiarities which enable us to determine the species before the head can be obtained. It is considerably longer than *T. solium*; Leuckart says that it may reach 12 feet in length. It is also

* It is remarkable that such patients do not more commonly become affected with bladder-worms. For the ova are very apt to hang about the anus, and must frequently be carried thence at night-time, and finally might reach the alimentary canal. Moreover, long-continued retching might bring some of the joints into the stomach.—C. H. F.

† *Synonyms*.—*Tænia inermis*—*T. saginata*—Beef-tapeworm. Küchenmeister gave the specific name "*mediocanellata*," believing that it had a median water-vessel beside the two lateral ones; but this was apparently an accidental peculiarity of a malformed specimen. This tapeworm has only been recognised as a distinct species since Küchenmeister's original account of it was published in 1852. Bremser, indeed, had previously noticed that the *tæniæ* which he obtained from human beings in Vienna had no hooks; but he thought that they had dropped off by reason of the age of the worms.

firmer in texture and flatter, and of a darker colour towards its distal extremity.

In *T. solium* the uterus is full of ova at about the 200th joint; in *T. mediocanellata* not before the 360th or 400th joint. The ripe proglottides are larger than those of *T. solium*, measuring three quarters of an inch in length and a quarter to one third of an inch in breadth. They more generally rupture and discharge their ova while in the intestine, so that those which are passed *per anum* are often shrivelled and empty.

The specific peculiarity of the proglottides of the beef-tapeworm is the form of the uterus. This has from twenty-five to thirty branches on each side of its longitudinal channel (*T. solium* having only from seven to ten); they are, therefore, packed much more closely; they are simply forked over and over again, and terminate in round, club-shaped ends, not in the broad, notched, or leaf-like pouches which are seen in the pork-tapeworm. The ova closely resemble those of *T. solium* in their minute size and thickness of the capsule, but are somewhat oval in shape.

Another peculiarity of *T. mediocanellata* is its liability to malformations. Sometimes there are two or three genital pores in a single proglottis, each corresponding with a separate double sexual apparatus; sometimes the segmentation is incomplete; sometimes a supernumerary proglottis projects by the side of the continuous line of joints. The most remarkable malformation is the existence of two distinct chains, united in their whole length by one edge, so as to form a "double monster."

The source of *Tænia mediocanellata* remained long undetermined. It had, however, been observed that the tapeworm which was known to be common in Abyssinia belonged to this species, and that the inhabitants ate, not raw pork, but raw beef and mutton. It was also noticed that infants to whom grated raw beef was given under medical advice were liable to tapeworm, and this was in one instance found to be "unarmed." Küchenmeister related the case of a patient who had harboured this parasite ever since a particular period when he had fed several times on raw beefsteaks. Putting these facts together, Leuckart came to the conclusion that the bladder-worm corresponding with this tænia probably occurred in horned cattle. He therefore in 1861 gave part of a strobila on two occasions to a young calf. Twenty-five days after taking the first portion of tapeworm the calf unexpectedly died. The muscles (including the heart), the lymphatic glands, and other parts, were full of minute round or oval vesicles, embedded in an opaque, whitish substance, which made them much more conspicuous objects than they would otherwise have been. They looked like tubercles, but were the cystic form of *T. mediocanellata*.

This bladder-worm has but a brief existence; if its host is allowed to remain alive, it perishes and calcifies in about eight months.

The frequency of *T. mediocanellata* as compared with *T. solium* varies, as might be expected, in different countries, according as the people live more on beef or on pork. It is stated that in Bavaria and Würtemberg the armed tapeworm is never met with, whereas in North Germany it occurs almost to the exclusion of the unarmed species. In the United States *T. mediocanellata* is commoner than *T. solium*, and *Bothriocephalus* probably only occurs in the case of immigrants from countries where it abounds. In England Cobbold found that *T. solium* is more common among people of the lower class, who eat much pork, whereas *T. mediocanellata* occurs in those who are better off, and eat more veal or beef.

He believed that the latter, on the whole, is the tapeworm most prevalent in this country; and this conclusion has been confirmed by subsequent experience in London.

A third species of the same genus, *T. nana*, was once discovered by Bilharz in large numbers in the intestine of a boy at Cairo, and others have been described in isolated cases, from Iceland, America, and the West Indies. *T. echinococcus* has never been found as a strobila in man.

3. The only other species of tapeworm which is common in man is *Bothriocephalus latus* (Bremser), or the "broad tapeworm." It is said to have been originally distinguished in the seventeenth century by Felix Plator, who called it *Tænia prima*, to distinguish it from the worm after named *T. solium* by Linnæus, which he called *T. secunda*. It is larger in every dimension than any other human parasite. It measures eight to twenty or even twenty-six feet in length, and has from three to four thousand joints. In the middle of the strobila they are nearly half an inch broad by one seventh of an inch in length; but towards the distal end they increase in length and diminish in breadth until at last their form is almost square.

This tapeworm has a longitudinal projecting ridge traversing its whole length. Its head is unarmed; it is club-shaped, and has two deeply grooved longitudinal suckers, one on each side, whence it takes its generic name of "pit-headed." The reproductive organs differ altogether in appearance from those of the tæniæ. The genital pore lies in the middle of each segment, opening upon its ventral surface. The uterus is an unbranched tube, which is bent on itself four or five times each way; and when distended with ova, its loops make it resemble a rosette.

The eggs are larger than those of the tæniæ; they measure 0·07 mm. in length, and are oval in form, with an operculum or lid at one end which allows the escape of the embryo. The capsule is comparatively thin.

A peculiarity of this tapeworm is that its joints do not come away singly, but in fragments of the strobila from two to four feet in length.

Like the tæniæ, the broad tapeworm is usually single, but like them, two specimens are occasionally found in the same patient. In the 'Pathological Transactions' for 1890, Dr Montague Murray records the occurrence of no less than seven tapeworms of this species, which were found unattached, in an entangled mass, in the cæcum of a man who died of tubercular meningitis without abdominal symptoms. He was an Englishman by birth, but had lived several years in Sweden, Finland, and Russia.

The *Bothriocephalus latus* is almost limited to the inhabitants of certain countries of Europe. The locality in which it is best known is the western part of Switzerland; in Geneva one person in every four is said to harbour it. It occurs in the north-west of Russia, in East Prussia, in Sweden (where the whole population of one province seems to have been infested with it), and also in Poland, Holland, and Belgium. It is doubtful whether it has ever appeared in England.

The source from which this parasite enters the human body has not yet been certainly determined. The observation has long been made that the districts in which it is met with are low-lying regions, situated either near the sea, or at least near some large lake or river, and it has been suspected that the corresponding bladder-worm inhabits some kind of fish, or possibly a fresh-water mollusc. Salmon, trout, and pike are especially likely to be

the resting-place of the immature form of the bothriocephalus. This view derives support from the fact, first discovered by Schubart, but more fully investigated by Knoch in 1862, that after keeping the ova several months in water, each of them gives out an embryo possessing the usual six hooks, but enclosed in a membrane covered with long delicate cilia. These enable it to keep up a constant rotatory movement, like that of a volvox. After four to six days, it escapes from the ciliated membrane and becomes free. Its further fate has as yet eluded observation.*

Another species, *B. cordatus*, has been more than once observed in human beings in Greenland, and a third, *B. cristatus*, twice in France.

Symptoms of tapeworms generally.—The effects of the presence of a cestoid worm in the human intestine are due not to the loss of material by the development of the parasite, but to the reflex symptoms produced by its movements. Adults and healthy children seldom experience discomfort; it is only when proglottides or portions of their strobila are evacuated, that a suspicion arises that they are not perfectly well. When this has once happened, the patient often begins for the first time to complain of pains and other symptoms, of which nothing had before been heard, although the parasite must have been present for several months. The sensations caused by it are described as an "uncomfortable feeling in the abdomen," "a colicky pain," a "gnawing pain at the epigastrium." Sometimes the patient is convinced that he can feel the movements of the worm; and it must be remembered that the sluggish contractions of the strobila outside the body are not the limit of its activity in the warmth of the intestine. Leuckart speaks of the vigorous movements of its segmented body, the continual play of its suckers, and the bendings of its neck. It usually hangs at length, but sometimes it is bent on itself, or rolled up.

Foulness of the breath, irregular and craving appetite, constipation, or diarrhoea, have been ascribed to the presence of a tapeworm in the human subject. It is also supposed to cause itching of the anus, itching of the nose, grinding of the teeth, headache, giddiness, lassitude, and faintness. Patients are said to have been cured of hysterical fits, epilepsy, and even mania by the expulsion of the worm. That hysteria may thus be cured may be well believed, as the following case seems to prove.

Graves relates the case of a young lady, who was attacked with what were regarded as alarming symptoms of bronchitis. She had a dry, hollow cough, which was repeated every five or six seconds, night and day, whether she was asleep or awake. Bleeding, tartar emetic, blisters, antispasmodics, were tried in turn, but without result, until Graves gave up the case in despair. At last she had a sudden attack of colic, for which an old servant of the family gave her a full dose of oil of turpentine with castor oil. She passed a large piece of tapeworm, and from that moment every pulmonary symptom disappeared.

The bothriocephalus is said to give rise to more marked symptoms than the tæniæ, but it also may be altogether latent. Bremser mentions the case of a Swiss, who had been eleven years away from his native country before he discovered that he was the host of this parasite.

Prophylaxis.—To prevent the development of tapeworms in the human intestine, meat which contains cysticerci should not be eaten at all; and all meat—and also fish, particularly salmon and pike—should be so well

* Knoch thought that he had proved that the administration of proglottides of bothriocephalus to puppies led to the direct development of the tapeworm in their intestine, but the validity of his experiments was disproved by Leuckart.

cooked before being eaten, as to destroy any cysticerci that might be present.

Measly pork may often be easily recognised; the bladders are of considerable size and may be present in very large numbers. But it is remarkable that in the flesh of horned cattle cysticerci have never yet been seen, except after the experimental administration of proglottides of *T. medio-canellata* to the animals (p. 453). The ox or heifer is a more cleanly feeder than a pig, and so its only chance of being infected with the cysticercus is by swallowing stray ova on the grass it eats or in the water it drinks.

Happily cysticerci cannot survive the temperature of boiling water; but, for more reasons than one, sausages which are underdone in the middle should be avoided.

In this country several cases have been recorded of tapeworms infecting persons who have been addicted to eating meat raw. But the most striking instance is that given by Kaschin, of the Bürater of the Baikal. These people live almost exclusively upon flesh, which they neither properly clean nor thoroughly cook; and they eat from tables that are never washed, and are used for cutting up the meat. Even when stationed as Cossacks at Irkutsk, after they had been away from their native country for years, they were infested with tapeworms to such an extent that in 130 autopsies only two bodies were found to be free from the presence of the parasite; often there were several, and once as many as fifteen in the intestines of the same individual.

The curative treatment.—This consists in the administration of some substance which has the power of killing the parasite without injuring its host. At the present time no drug is used so largely for this purpose as the liquid extract (or “oil”) of male fern. A dose of from fifteen to thirty minims may be effectual, but at Guy’s Hospital we have been in the habit of giving a drachm. Sir William Gull published in the ‘Guy’s Hospital Reports’ (3rd series, vol. i, 1855) a series of 200 cases thus treated with much success. It never does any serious harm, but often produces sickness; and Cobbold recorded unpleasant effects on the nervous system from a large dose.

Another useful drug, derived from Abyssinia, is kousso, which consists of the dried flowers of the *Brayera anthelmintica*; from a quarter to half an ounce of this is infused in boiling water, and swallowed, powder and all.

Oil of turpentine, again, is often effectual: from half an ounce to two ounces may be taken, and a single dose is less apt to cause strangury. A decoction of the bark of the pomegranate root is another valuable anthelmintic; three or four doses of one or two ounces each should be given at intervals of an hour; but it often causes faintness and giddiness. Lately thymol has been highly recommended.

Whatever medicine may be chosen, it is advisable for the patient to fast for several hours before taking it, so that the alimentary canal being empty, the drug may with more certainty come into contact with the tapeworm. For the same reason a dose of castor-oil is sometimes given three or four hours before the anthelmintic.

The administration of one of the remedies above mentioned almost always brings away a large portion of the tapeworm, if there is a fully developed strobila in the patient’s intestine; but, unfortunately, it usually breaks at the neck. The head, or rather the scolex, remains behind, and at once begins to form fresh segments.

If the parasite is a *tænia*, it is remarkable that an interval of three months (Cobbold says thirteen weeks) is almost invariably found to elapse before proglottides again begin to be passed. Dr Fagge repeatedly saw this occur the very day which had been predicted. This period of three months corresponds exactly with the length of time which is required for the full development of the tapeworm from the scolex; so that it seems to follow that under the influence of anthelmintics the usual line of fracture is close to the head.*

The patient must always be told to look very carefully in his evacuations for the head, the appearance of which should be described to him. An enthusiastic practitioner may himself search for it. Cobbold recommends that the whole mass of *fæces* should be passed through a sieve. If the head be not discovered, the patient may either wait for three months to learn whether the treatment has been effectual, or he may take a second dose. One would have thought it doubtful whether drugs would act satisfactorily upon a tapeworm of which nothing but the head is left; but Cobbold relates one instance in which, having brought away almost the whole of a tapeworm with one dose of male fern, he gave another dose the next day, and succeeded in finding the head with its four suckers in the patient's *fæces*.

HYDATIDS.—*Natural history and development.*—We have seen that every tapeworm in the course of its development passes through a remarkable phase, in which it forms a bladder embedded in the substance of a host, and filled with a transparent fluid. What is commonly called a hydatid of the liver is the cystic stage of a minute tapeworm, which inhabits the intestine of the dog, and is called *Tænia echinococcus*. When fully grown this measures only four millimetres, or about the sixth of an inch, and consists of only three or four segments, of which the last alone contains developed sexual organs. It is very common in London dogs, and is often present in large numbers. Its ova are discharged with the *fæces* of the host and enter the human stomach either in drinking-water, or on the leaves of lettuce, cresses, or other raw vegetables.

When an ovum of the echinococcus-tapeworm has thus found its way into a human host, it at once enters upon the wonderful transmigration above described. The gastric juice dissolves its shell and liberates the embryo as a larva, or *scolex*, with its six chitinous hooks, to bore its way through the walls of the stomach or small intestine. Its further course is not always the same (p. 449); but hydatids are far more common in the liver than anywhere else, because the embryo, in piercing the wall of the stomach or intestine, generally falls into one of the rootlets of the portal vein, and is at once washed away by the stream of blood, until it is arrested in a portal capillary within the liver.

Having thus reached its destination, the scolex acquires four suckers and more numerous hooklets, twenty-eight to fifty-two, arranged in two circles. As it grows larger it becomes converted into a laminated cyst, lined with a layer of granular cells and containing a transparent fluid. It also becomes surrounded by a layer of vascular connective tissue which

* It no doubt sometimes happens that the creature breaks in the middle, particularly if the dose of the anthelmintic is inadequate. But this, at any rate, may be said,—that if a portion of tapeworm be brought away, in which part of the narrow neck is recognised, and if the patient should in much less than three months begin to pass proglottides *per anum*, it is certain that more than one *tænia* is present.—C. H. F.

grows with its growth. This capsule of a hydatid is formed from the human tissues, with which the hydatid itself lies in contact.

Up to this point the development of the echinococcus is like that of the cystic stage of any other *tænia* or a *cysticercus*. But the further steps are very different. The *cysticercus*, in order to complete its development, has only to form a single "head" or "scolex." But in the echinococcus, instead of a single bud, many form on the inner surface of the parent cyst, growing from the "granular layer" or endocyst, and pushing into the vesicle, like an invagination. These buds, or *deutoscolices*, do not develop into heads, but form fresh cysts, which for a time remain attached by a pedicle, but soon become detached. It is then called a "daughter-cyst," while the original hydatid that encloses it is termed the "mother-cyst." Each daughter-cyst, again, may develop one or more "granddaughter" cysts in its interior. In this way the echinococcus becomes filled with smaller vesicles of various sizes, which may amount to thousands. Occasionally certain echinococci, described as "pill-box hydatids," contain smaller vesicles arranged like a "nest" of pill-boxes. Sooner or later the little buds or protrusions cease to form detached vesicles, and develop into thin membranous sacs, the pedicles of which are persistent; these are called "brood capsules," because they give origin to a variable number of "scolices" or "heads," each of which has its row of hooklets and its four suckers, and is capable, under favourable circumstances, of growing into a *tænia*. These scolices or heads alone used to be called echinococci; and this use of the term accords both with its derivation (*ἔχινος*, hedgehog; *κόκκος*, grain or berry) and with the intention of Rudolphi, who invented it. But at the present day the term echinococcus is applied to the whole animal, with its daughter-cysts, brood-capsules, and scolices.

One difference between a *cysticercus* and an echinococcus is that, whereas the former gives rise only to one scolex, and can therefore ultimately form only a single tapeworm, the latter may develop thousands.

Echinococci do not, however, always pass through the changes above described. Sometimes they fail to produce scolices and daughter-cysts, and are then described as "sterile hydatids" or "acephalocysts." The term acephalocyst was invented by Laennec, who believed that the hydatid which infests the human subject never produced scolices, although he was aware of their presence in hydatids from the lower animals.* It is said that Bremser, in 1821, first discovered scolices in hydatids taken from the human body. Bright gave a drawing of them in the 'Guy's Hospital Reports' for 1837. Even after it was recognised that the hydatids of man usually contain scolices, they were still called acephalocysts, and the term may properly be applied to those hydatids which are really sterile. This is said to be more frequent in the case of hydatids infesting the brain than in those of any other organ.

Multiple hydatids.—Sometimes, but in the human subject very rarely, instead of budding internally to form daughter-vesicles, the echinococcus produces them externally. In this way the liver may become riddled with hydatids, not contained in any mother-cyst, but penetrating its tissue in all directions, and even invading the neighbouring organs.

* "There is no doubt that the hydatids in the livers of sheep are animalcules; they have been often seen to move when taken out of the liver and put into warm water."—Matthew Baillie, 1797. The discovery of the nature of hydatids was made by the naturalist Pallas in 1760.

A remarkable case of this exogenous echinococcus once occurred in Guy's Hospital, under the care of Dr Rees. A boy was admitted with what seemed to be effusion of fluid into the right pleura, and enlargement of the liver; but when the chest was punctured with a trocar, hydatids escaped. Ultimately he died, and it was found that the liver, diaphragm, and right lung were full of hydatids, which were all budding externally.

A remarkable form of hydatid, developed by this process of external gemination, is styled "multilocular" by Continental pathologists. It forms a solid globular mass in the liver, as large as a fist or a child's head. Its periphery is well defined, and it can be shelled out of the tissue in which it lies; but on section it is found to be divided by trabeculæ into a number of small cavities of irregular form, each containing a mass of gelatinous hydatid membranes pressed closely together and small cysts containing scolices. The individual cysts are never larger than peas, and are often as small as millet-seeds. Virchow supposes that the parasite occupies the lymphatic vessels. A multilocular hydatid tumour often undergoes softening in its centre, and forms a suppurating cavity, sometimes with secondary peritonitis and jaundice. This form of hydatid tumour seems hitherto not to have been observed in England.*

Dr Mitchell Bruce and Mr Sheild have recorded a curious case in which a hydatid cyst of the liver underwent degeneration into a tough, gelatinous substance, which prevented it from collapsing after puncture ('Med.-Chir. Trans.,' Feb. 9th, 1892).

The hydatid fluid has characters different from those of any other liquid that is met with in the chest or abdomen, but not unlike those of the cerebro-spinal fluid. It is limpid or very slightly opalescent, its sp. gr. is 1007 to 1009, or a little higher; it contains no albumen, so that it does not coagulate either when boiled or on the addition of nitric acid.† There is only a trace of sodic carbonate and chloride. When a glass containing hydatid fluid is held up to the light, one can often see floating in it delicate white bodies, so minute as to be only just visible, which rapidly settle to the bottom of the vessel. These are clusters of scolices, either still enclosed in their brood capsules or (if the latter are ruptured) kept together by their common stalk. They appear under the microscope as round bodies, with oval calcareous corpuscles scattered through their transparent substance, and each with its crown of hooklets and its four suckers, usually retracted into the interior of the cystic body. Often they are still alive, and can be seen to move. It was formerly supposed that the scolices, or "echinococcus heads," became detached from the main wall of the parent cyst in the course of their growth, and that they could swim about in the fluid. But this was a mistake; they are naturally fixed, and are only set free during the operation of paracentesis.

The discovery of a hydatid scolex, or of one of the indestructible hooklets, in a cyst of doubtful nature, is of course conclusive, and the membranous outer wall of a hydatid cyst also has distinctive microscopical characters. It is made up of very thin concentric layers; and the smallest portion is seen under the microscope to be marked with delicate parallel lines. A piece of hydatid membrane rolls itself up, so that the originally

* Frerichs suggested that a specimen in the museum of Guy's Hospital, which is labelled "colloid cancer of the liver," might be a multilocular hydatid. But some years ago I carefully examined this specimen, and could not discover any trace of a parasite.—C. H. F.

† Hydatid, like cerebro-spinal, fluid contains a trace of grape-sugar, and succinate of ammonia has also been found in it.

inner surface is outermost. Chemically it consists of a modification of chitin.

Events.—It is an interesting question whether there is any natural limit to the life of the echinococcus. Reynal is said to have met with a patient in whom a tumour of the neck had existed from the age of seventeen to sixty, and when punctured gave issue to hydatids, all apparently living; and Budd recorded the case of a lady who died at the age of seventy-three, and who was believed to have had two hydatid tumours since she was eight years old. But dead hydatids are frequently discovered when the patient has died at a much earlier age.* The fibrous capsule of a dead hydatid is often of cartilaginous hardness, or in great part calcified.

Anatomical distribution.—Echinococci are seldom found anywhere else than in the liver.† Leuckart supposed that for every three cases of hydatid of the liver there may perhaps be one in some other organ, but even this estimate is probably above the mark.

Davaine collected very carefully all published cases of hydatids occurring in other parts of the body than the liver, and he found that among 200 cases there were about 40 of hydatids of the lungs, about 30 of the muscles and subcutaneous connective tissue, 30 of the kidneys, 26 of the pelvis, 20 of the nervous centres, 17 of the bones, and 10 of the heart.

When an echinococcus develops itself in one of the *lungs*, it is found as a rule in the base of the right lung, a fact probably due to the six-hooked embryo having penetrated into the chest from the liver, or to a primary hepatic cyst opening into the lung. Thus pulmonary echinococci are almost always migrated parasites of the liver. Clinically, hydatid disease of the lung is scarcely likely to be suspected until one or more of the daughter-cysts have been expectorated. It produces hæmoptysis or purulent expectoration with fever, and has frequently been taken for phthisis.

Hydatids of the *brain* have occasionally been met with in the dead-house; the symptoms are undistinguishable from those of other cerebral tumours. The capsule of a cerebral hydatid is exceedingly thin and delicate, and the cyst is sometimes acephalic (p. 458).

Echinococci have been found in the *heart*, where they may give rise to varied symptoms, or sometimes to none at all.

The *spleen* is very rarely the seat of a hydatid cyst. In one case of the kind there was a large tumour in the left hypochondrium, but until an autopsy had been made, it remained uncertain in what organ the parasite was seated. Hydatids of the *kidney* are not nearly so rare.

In the *pleura* and the *pericardium* the echinococcus cyst may grow to considerable size, without the formation of a capsule round it; but this is said not to be the case with the *peritoneum*, in which hydatids are described as always having a proper capsule. Hydatid of the peritoneum may be mistaken for cystic disease of the ovaries; and an attempt has several times been made to remove such a tumour by ovariectomy. Occasionally hydatid cysts are found in different parts of the abdominal cavity, some of

* Cruveilhier supposed that the entrance of bile into the capsule may cause the death of the parasite. But this seems very doubtful, for I once found two dead hydatids in the same liver, one of them containing bile-stained matter, while the contents of the other were colourless; and it seems unreasonable to attribute the one death to the toxic action of the bile, and to leave the other unexplained.—C. H. F.

† On the treatment of hydatid tumours in the liver, see an important discussion at the Clinical Society in December, 1887 ('Trans.,' vol. xxi).

them having been developed in the omentum, and others in the interspaces between the different viscera.

Lastly, there is a local variety of hydatid, which develops itself in the *pelvis*, between the bladder and the rectum, or behind the rectum, in men, and behind the uterus in women. In these cases the six-hooked embryo doubtless falls into the serous cavity when it has penetrated the walls of the stomach, and rolls into the most depending part of the peritoneal sac. The result is the formation of a tumour which may assume an oval form exactly like that of a distended bladder, and may occupy precisely the same situation. In a case of this kind which occurred in our wards it was supposed that the bladder was full, but of course the catheter failed to give relief; the patient died, but until the autopsy its real nature was not even suspected. Bright relates a similar instance, and several others have been placed on record by different observers; so that whenever a fluid tumour is felt in this position, which cannot be reduced by passing a catheter, the possibility of a hydatid should be considered.

Nematoid worms.—The remaining intestinal worms belong to the Nematodea*—a group of uncertain affinities, parasitic, but widely differing in structure from the Cestoid worms, and without the same remarkable transformations.

The common round-worm (*Ascaris lumbricoides*, Linn.) is an almost universal parasite in the human intestine all over the world, and most animals are infested by allied species and genera.

The distinction between what is natural and pathological fails here, as in many other cases. Parasitical animals and plants exist everywhere and by as good a right as their hosts. It is as "natural" for a man to have worms in his bowels as pediculi in his hair, and both entozoa and epizoa are found in quadrupeds and all other mammals. The cleanliness of the most civilised communities is only maintained by constant effort, and freedom from external and internal parasites is as "artificial" as freedom of a well-kept garden from weeds.

As its specific name implies, the ascaris is not unlike the common earth-worm (*Lumbricus terrestris*) in appearance. When alive it is of a reddish-brown colour with a tinge of yellow; but after its death this colour slightly fades, and it becomes greyish. It has a disagreeable smell, which cannot be removed by washing, and which, according to Leuckart, has its seat in the deeper muscular layers.

Dr Bastian and some others have suffered from irritation of the eyes, sneezing, and other symptoms like those of hay-catarrh after dissecting this worm.

The female is fifteen inches long; the male, which is comparatively seldom met with, measures only ten inches; its circumference is also much less than that of the female. They are both cylindrical in form, tapering at each end, but rather more gradually towards the head than the tail.

The ova are elliptical in form, much larger than those of the tænia, and not unlike the eggs of bothriocephalus in size and shape.

The life-history of this parasite has not yet been completely ascertained.

* Sometimes spelt "Nematoda." The word is derived regularly from *νήμα*, a thread, and *εἶδος*, appearance. The group nearly corresponds with the Cœlminthia, *vers entozoaires cavitaires* of Cuvier.

The female discharges ova which certainly do not undergo any development while they are in the human body. After their escape with the fæces, however, an embryo slowly appears in each egg if it be kept in water or in moist earth. Davaine and others formerly supposed that the ova were swallowed in this condition, either in drinking-water or upon uncooked vegetables or fruit, and that after passing through the stomach, the embryos gradually became developed into full-grown worms. But several German investigators have deliberately swallowed large numbers of ova, and no specimen of the ascaris has hitherto been obtained in this way.

Leuckart thought it most probable that the ova are swallowed by some intermediate host—perhaps the larva of some insect—and that within its body the embryos pass through changes which fit them to be transferred to the human intestine; but there is no proof that another host is needed.

The *Ascaris lumbricoides* is rare in infants under a year old, and is most common in children between three and ten years of age, as was observed by Hippocrates. It is more common in rural districts than in towns, and particularly in low and damp localities; and it is met with more often in the autumn than at any other season. Those who are poor and dirty are more subject to it than others. In the insane it is very common; among thirty lunatics of dirty habits in the Hofheim Asylum there was not one who was free from this parasite. In the Southern States of America, the West Indian islands, Cayenne, and Brazil, the negroes at all ages are, with scarcely an exception, infested with round-worms. In Europe they appear to be particularly common in Finland and Holland.

The round-worm inhabits the small intestine. It may be solitary, or there may be two, three, or any number of them. When numerous they are often coiled together in knots, and they have sometimes been found obstructing the intestine. Children have been known to pass some hundreds of them in the course of a few weeks; and Cruveilhier found more than a thousand in the intestine of an idiot.

It is probable that each individual worm remains only a few months within the body of its host. If they pass down into the large intestine they are voided from the anus, either alone or with the fæces. If they make their way upwards into the stomach they are generally vomited. Sometimes a worm is discharged through the nose; and it has even been known to enter the larynx, and cause death by suffocation.

A curious point, to which Cobbold drew attention, is that the ascaris is apt to insinuate itself into any kind of small ring that may be swallowed by its host, such as the "eye" which answers to a "hook," or the shank of a button. A single worm has been found with two buttons thus attached.

The parasite sometimes makes its way into the bile-duct or gall-bladder, setting up jaundice, or even suppuration in the liver. It has sometimes escaped into the peritoneal cavity through the floor of an intestinal ulcer, and has been found in an abscess at the umbilicus or in the groin. After its removal the abscess has generally been found to heal.

The *symptoms* produced by these worms vary according to their number and the irritability of their host. A single *Ascaris lumbricoides*, or even two or three, would scarcely cause appreciable discomfort. When symptoms arise, they are said to be uneasiness in the abdomen, nausea, foulness of breath, irregularity of appetite, flatulence, and the presence of mucus in the stools.

In certain cases the presence of round-worms is reported to have caused

reflex symptoms like those ascribed to tapeworms: dilatation of the pupils, squinting, irritation of the nostrils, and grinding of the teeth in sleep.

When the round-worms occur in large numbers it is possible that the loss of material involved in the formation of its countless ova may directly affect the nutrition of the host, especially if the host is a child.

But far more serious cases have been recorded, of fatal obstruction caused by masses of the parasites rolled up together. Some of these are probably not genuine; but Dr Beaven Rake reported an undoubted case which occurred in a negro child in Trinidad ('Guy's Hospital Gazette,' March 8th, 1890). It is also possible that the presence of worms may lead to invagination.

Increasing cleanliness of habits and care in the preparation of food have no doubt diminished the frequency of round-worms in civilised countries. In China, where the population is excessive, the food scanty, and sanitary precautions absent, the expulsion of enormous numbers of this parasite forms part of the daily routine of practice, and the effects of the ascaris are far more serious than those commonly seen in Europe.

Diagnosis.—There are two conditions under which one has to deal with this parasite:—either when a patient, generally a child, presents some of the symptoms just enumerated; or when one ascaris has been passed *per anum*, and there is a question whether there are still others in the intestine. In this case one has to bear in mind that impostors have been known to bring earthworms with them, which they pretend to have passed from the bowels. The true lumbricus, however, is readily distinguished from an ascaris; it is much redder, it tapers less at its extremities, and it has rows of small bristles, which aid it in locomotion; its mouth is a short fissure on the under surface of its rounded head, whereas the mouth of the *Ascaris lumbricoides* is a triangular aperture at the more pointed end of the animal, surrounded by three tubercles or lips.

When the presence of this parasite in the intestines is suspected, the question may readily be answered by a microscopical examination of the patient's fæces, which, if the worm is there, are sure to be full of its eggs.*

The ova are elliptical in form, measuring $\frac{1}{340}$ of an inch by $\frac{1}{440}$ of an inch; they are of a brownish colour and nodulated on the surface, from a thick layer of an albuminous substance deposited outside their proper shell.

Dr Ransom gives (in Reynolds' 'System of Medicine,' vol. iii, p. 197) the case of a child who was admitted into hospital for abdominal pains and disordered digestion, and because she had passed two round-worms previously. The evacuations contained the eggs of the parasite. Medicines on several occasions brought away one or more specimens of the ascaris, and her symptoms entirely disappeared. But ova were still detected in the stools, and therefore she was kept under treatment three months and a half longer, until seventeen worms in all had been passed. No more of the ova could then be discovered, and she was accordingly sent out of the hospital.

Treatment.—Santonine appears to be the most efficient drug in expelling the ascaris. The dose, for an adult, is from three to six grains twice daily, and for a child one to three grains; but while it is being taken, an occasional purgative should also be prescribed. An inconvenience sometimes

* The round-worm is estimated by Eschricht to produce in the year some 60,000,000 eggs. Though the egg of an ascaris is extremely minute, its diameter being a twentieth of a millimetre, the total weight of the yearly produce of the eggs comes, according to Leuckart, to a mass 1740 times that of the parent worm. The queen bee only produces 130 times her weight in the form of eggs. . . . To equal the fertility of the ascaris the human female would have to produce about 25,000 children in the year.

produced by santonine is a curious temporary disturbance of vision, objects appearing of a yellow, green or blue colour. The urine may also be reddened; but what is of more consequence, santonine sometimes produces tenesmus, and even hæmorrhage from the bowels. Dr Ransom speaks of *Dolichos pruriens* and oil of turpentine as being also worthy of trial.

As to preventing the *ascaris ovum* from entering the body, all that can be said is that one should be careful to drink only pure water and to have all solid food thoroughly cooked.

The allied species *Ascaris mystax* has occasionally been observed in the human intestine by both German and British helminthologists.

The *Oxyuris vermicularis* (Bremser), or "threadworm" (*Germ.* Maden-wurm), is very much smaller than the round-worm just described. It was formerly called *Ascaris vermicularis*, and this name is not quite obsolete, for threadworms are still called "ascarides;" but the term is incorrect, and liable to mislead.*

The oxyuris is like a small piece of white thread. The female measures four tenths of an inch in length, the male one sixth of an inch. The latter is much less often seen; it is supposed that there is about one male in ten. They taper towards the tail, as their generic name denotes.

Threadworms occur only in the large intestine, the cæcum, and colon, as well as the rectum. The bile-pigment can be recognised in their bodies. They are often present in vast numbers, and are found either singly in the mucus of the bowel, or matted together into little balls.

The eggs of oxyuris are oval in form, and flattened on one side, with a smooth surface and thin membranous shell. They measure $\frac{1}{1100}$ of an inch by $\frac{1}{190}$ of an inch. Unlike those of the *Ascaris lumbricoides*, they already contain embryos when deposited. It might therefore be supposed that the oxyuris could multiply in the human intestine for an indefinite period; but some good observers believe that the ova are incapable of undergoing development until they have passed into the external world, and been again swallowed. This theory is supported by analogy; and Leuckart never found *young* oxyurides in number approaching that of the ova, whereas, if they were developed directly from the eggs, they ought to be abundant. But Dr Guillemard ('Allbutt's System,' vol. ii, pp. 1032, 1035) considers that both round-worms and threadworms are probably developed from ova without the help of any intermediate host.

The oxyuris emerges from the rectum of its host, especially at night; it creeps about the anus, and in women may pass into the vagina. It is therefore conceivable that, as Küchenmeister supposed, the worm might obtain direct access to a fresh host. But the migration of threadworms probably occurs as follows. The worms and their ova often become adherent to the skin and hair about the anus; they dry up, and ultimately break down into dust, containing enormous numbers of ova still capable of springing into life if brought under suitable conditions. Thus every opportunity is afforded for "self-reinfection." Grassi ascertained by experiment that a single infection by ova through the mouth will keep up successive swarms of worms and eggs for five or six weeks.

Again, every fæcal evacuation of a person infested with threadworms probably contains numberless ova, which may be carried into drinking-

* Etymologically the word ἀσκαρίς (from ἀσκαρίζειν, to leap) is applicable rather to the threadworm than to the less active round-worm.

water, taken up by flies, deposited upon vegetables and fruits, and so in countless ways gain access to a fresh host.

The *symptoms* produced by threadworms are due to the irritation they produce by creeping about the anus. Pruritus comes on generally soon after the patient is in bed, but sometimes earlier in the evening. Marchand quotes the account which a man gave of his own sufferings, as follows:—"Every evening about 5 or 6 o'clock, when I first feel the worms, I become pale and troubled, and sometimes I have even shivered; my companions often notice it; I am restless and obliged to walk about; if I am at a place of entertainment, I leave instantly and hasten to employ a cold enema; this does not always give me relief, and I am then in torture; I tear my perinæum and scrotum, and am obliged to micturate every instant." Irritability of the bladder is sometimes caused by the presence of threadworms, and they sometimes excite priapism or nymphomania. Another occasional symptom is tenesmus; and the fæces may contain a large excess of mucus. Cruveilhier recorded the case of a child who was awakened every night at the same hour by an agonising pain in the anal region, so that he screamed and writhed about in bed. The periodicity of the attacks led to the administration of quinine, but with no success, until threadworms were looked for and the cause discovered.

The *treatment* is a less easy matter than might be expected. Watson often relieved patients of threadworms by prescribing infusion of quassia as an injection. Lime-water, solutions of chloride of sodium, or of perchloride of iron also have been recommended, injected every third or fourth day for two or three weeks.

Since the oxyuris occurs in the cæcum and in the upper part of the colon, Cobbold recommended active saline cathartics, repeated for several days in succession, before enemata are given, also large draughts of infusion of gentian, and an Indian remedy—*Aristolochia bracteata*. The introduction of a little mercurial ointment within the verge of the anus before the patient goes to bed will effectually prevent threadworms from creeping out of the rectum; but this plan requires some caution, lest salivation should follow. Relief is afforded to the itching by the application of carbolic oil or dilute red oxide ointment or cocaine.

Children, particularly about five or six years old, are much more commonly infested with threadworms than grown persons, but they are also more easily freed from their presence than adults.

If the above view of the life-history of the oxyuris is complete, scrupulous cleanliness in all the surroundings of a patient is the only prophylaxis.

The *Trichocephalus dispar** (Rudolphi), another nematode worm, has its seat in the cæcum. It is remarkable for its very long thread-like neck, which forms about two thirds of its whole length of one and a half to two inches; and it was formerly called *Trichurus* from a mistake between the head and the tail. The female, which is more numerous, lies coiled up in the mucus. This parasite appears to give rise to no symptoms; it has scarcely ever been discovered in the evacuations, and possesses no clinical interest.

As its ova may be found in the fæces, it is well to mention that they are bluntly spindle-shaped, with transparent ends, and that they measure

* *Ascaris trichiura* (Linn.), *Trichurus* (Buttner).—*Ger. Peitschenwurm.*

0.023 mm. in breadth by 0.051 mm. in length, somewhat less than those of oxyuris.

The *Ancylostomum duodenale** (Dubini) is of far greater importance, although happily it is not found in this country. It is occasionally met with in Italy, and was discovered at Milan by Dubini in 1843; it occurs also in Sardinia and in Sicily. It is almost universal among the natives of Egypt, where it was discovered by Griesinger and Bilharz in 1851. (See Dr Sandwith's monograph, read at the Congress held in Rome in 1894.) It is common in Spain, in Northern and Central Africa, the East and West Indies, Peru, and Brazil. In Northern Europe—Bonn, Chemnitz, St Etienne, and Liège—it has been found in miners. At a meeting of the Pathological Society in 1867, some specimens of it from Brazil were exhibited by Dr Hermann Weber. (See his paper, with figures, 'Path. Trans.,' xviii, p. 274.) It is still very common in Egypt and in some parts of Hindoostan (Dobson).

It must be borne in mind that this parasite is often present without the serious symptoms to be described; in fact, the host may appear to be in ordinary health.

The sclerostomum inhabits chiefly the jejunum, generally lying between the valvulæ conniventes, with its mouth firmly fixed in the mucous membrane by means of its four conical chitinous teeth. The female occurs in larger numbers than the male.

The sclerostomum is a small round worm with its head bent nearly at right angles. The male measures nearly half an inch in length; the female seven tenths of an inch. It may be present in enormous numbers, for 1250 have been counted in a single patient. It feeds, not upon the intestinal contents like other worms, but upon blood, which fills its digestive canal, and gives its body a red colour. It fixes itself firmly by means of two pairs of teeth (of which the ventral pair is the larger) into the mucous membrane of the duodenum or jejunum; and within its mouth there are two moveable blades, which doubtless serve to incise the tissues.

The spot to which it is attached is indicated by ecchymosis; and probably, if it shifts its position, the punctures which it leaves may go on bleeding. The intestines are sometimes found full of blood after the patient's death, although hæmorrhage from the bowels seldom occurs during life. The body of the worm usually hangs free within the gut, protected more or less by the ridges of the mucous membrane; but sometimes it is rolled up.

The female sclerostomum lays numerous eggs, which are oval bodies, with a thin, transparent shell, of nearly the same size as those of the oxyuris, but less elongated, measuring only $\frac{1}{20}$ mm. in length. They also differ in having no operculum, and in their yolk being undivided or only just segmented at the time of their expulsion in the fæces, whereas the eggs of the oxyuris already contain embryos.

The life-history of the sclerostomum has not been directly traced, but it is believed to be the same as that of the allied *Dochmius trigonocephalus* of the dog. If this be correct, the ova become hatched when they pass into mud or water, and produce slender worms which exhibit active movements. These require no intermediate host, but develop into sexually mature animals before they are swallowed and reach the human alimentary canal.

* Also known as *Sclerostomum duodenale* (Cobbold), as *Strongylus* or *Dochmius duodenalis* (Leuckart, Diesing), and as *Str. quadridentatus* (von Siebold).

The parasite is met with chiefly in hot climates, where men are often compelled to drink water from dirty pools exposed to contamination in every way.

Since hundreds and even thousands of these parasites are sometimes present in the same individual, it is not surprising that they should give rise to grave anæmia.

It was Griesinger who showed, in 1854, that the form of anæmia known as Egyptian chlorosis was due to the presence of this parasite ('Arch. f. phys. Heilkunde,' xiii, 557). Wucherer, of Bahia, found in 1886 that the same symptoms depend on the same cause in Brazil; and Dr Strachan has reported cases from Jamaica ('Brit. Med. Journ.,' June 27th, 1885). In fact this form of severe and sometimes fatal anæmia has been met with in all the warmer parts of the world.

The ancylostomum was the cause of epidemic anæmia among the workmen in the St Gotthard Tunnel in 1880 (see a paper by Dr Bugnion, 'Brit. Med. Journ.,' March, 1881, p. 882). One of these cases came under the care of Prof. Bäumler, of Freiburg. Similar epidemics of anæmia have been observed among miners in Hungary, Germany, Belgium, and France, the warmth of this underground habitat being apparently essential to the life of the parasite.

It is remarkable that at least in Egypt the disease is very rare in women, and in children under twelve years old. Among 402 patients Dr Sandwith found only three females—one grown woman and two girls about thirteen. It is much less common among the negroes and in the Soudanese regiments than among the Egyptian fellaheens; and it is only very rarely seen in Europeans. But the 'Lancet' (Feb. 1st, 1890) relates the case of an Englishman who was subject to anæmia in India, was treated in London, sent to Australia for his health, and the cause of his malady was at last discovered and cured by a physician in Heidelberg.

The resulting anæmia may prove fatal in a few weeks, or it may run on indefinitely, unless death occurs by dysentery. At first the patient appears well nourished, but at length he becomes wasted and dropsical. The symptoms are those which follow constant loss of blood from any cause:—giddiness, sleepiness, dyspnoea, and in advanced cases, œdema. The temperature is usually normal or subnormal. The number of red blood-discs is diminished sometimes from 4 to 1 million per cubic millimetre. The hæmoglobin is reduced to 50 or 30 per cent. of the normal.

If unrecognised, this "tropical anæmia" may last for years, until the patient is at length carried off by diarrhoea, pneumonia, or some other accidental malady. More rarely the fatal termination is by dropsy.

Microscopically the eggs may be recognised by diluting the fæces with water, stirring, and examining the sediment. They are much like those of oxyuris, but more slender in shape. The worms themselves remain fixed in the intestine, but after a purge may be discovered in the fæces.

Detection of the ova or worms is the only way of distinguishing anæmia thus produced from other kinds of symptomatic anæmia, and from the severest form described by Addison as idiopathic, and since called pernicious.

The drugs which have been used in the *treatment* of this disease are oil of male fern, santonine, oil of turpentine, assafoetida, and aloes; but thymol in large doses (introduced by Bozzolo in 1880) is now found to be the most effectual treatment. Prophylaxis depends entirely on enforcing habits of personal cleanliness.

*Strongylus (Eustrongylus) gigas** is a very large nematoid worm inhabiting the pelvis of the kidney in certain Carnivora, and very rarely occurring in human beings. It is not very uncommon in dogs and wolves, in the coatimondi (*Nasua*), racoon, otter, and seal, and has also, it is said, been met with in horses and oxen. Most recorded cases in man are spurious, and refer to ascaris or to fibrinous clots passed *per urethram*. Küchenmeister quoted fourteen cases, but only those of Grotius (1595), Ruysch, and Blasius, and a more recent one of Moublet, seem to be authentic. Diesing can only adduce three clear cases. A specimen in the Hunterian Museum is said to be taken from the human kidney.

This species is the largest nematoid entozoon known. The male grows to a length of ten inches, and the female to thrice that length. One huge strongylus of this species was found free in the abdominal cavity of one of the Esquimaux dogs which McClintock took on his arctic expedition in search of Sir John Franklin. It was sent from Greenland to Steenstrup, at Copenhagen, and by him given to Leuckart, who based his account of the strongylus on that specimen from the dog and two others, one from a coati, and the other from an American mink (*Mustela vison*).

An allied but much smaller species (*Strongylus armatus*), after it has wandered from the cæcum or colon, affects the blood-vessels, and produces aneurysms in the mesenteric arteries in horses and asses.

Trichina spiralis† (Owen).—This nematode is truly an intestinal worm, although it was long after its discovery only known as occasionally found in the muscles. Its life-history is not unlike that of the other nematodes which infest the human intestine; the principal difference being that, instead of its ova passing out through the rectum, they develop within the parent worm into young trichinæ, which, as soon as they are born, begin an active migration among the tissues of their host.

Tiedemann, in the year 1822, first noticed the presence of white stony concretions in the human muscles, but he did not describe them further, and Leuckart doubts whether these were trichina capsules. In this country Hilton was the first, in 1832, to record the fact that he had met with the encysted trichinæ.‡ He could not determine their precise nature, but thought that they were probably very small cysticerci. Next Sir James Paget, then a student of St Bartholomew's, independently described them. Two years later Owen showed that they were hollow capsules, and that each of them had coiled up within it a minute nematoid worm, to which he gave the name of *Trichina spiralis*, and fully described its anatomy.

The capsules enclosing the worm are just visible to the naked eye: they measure $\frac{1}{78}$ inch in length, and $\frac{1}{130}$ inch in breadth. They are lemon-shaped, and their long diameter is always in a line with the muscular fibres among which they lie. They occur in all striped muscles, even in the tensor tympani, but are most numerous in the diaphragm and the muscles of the trunk. The heart is sometimes affected, but in unstriated muscles they are never met with, so that the œsophagus contains them only in its upper part. They have, however, been found in the subcutaneous fat, and in the intestinal walls. They feel gritty when touched with a knife.

* *Strongylus* (στρογγύλος), round.

† Hair-worm—θρίξ, τριχός, a hair.

‡ 'London Medical Gazette,' 1833, p. 605. Owen's paper is in the 'Zool. Trans.,' vol. i, p. 315 (1835). The oldest preparation of trichinæ is one of the sterno-hyoid muscle in the Guv's Museum, put up by Hodgkin in 1828.

owing to the deposition of calcareous matter; this sometimes renders them altogether opaque, but is usually present only in their extremities, leaving the middle transparent, so that under the microscope the little worm in the interior is at once visible. There is an excellent description of the trichina, with figures by Dr Bristowe and the late Mr Rainey, in the 'Pathological Transactions' for 1854; and good plates are given in Cobbold's 'Entozoa.'

Not only swine (the usual bearers of the disease to man), but rats above all, also cats and dogs, hedgehogs and moles, horses and calves, rabbits and guinea-pigs, are liable to infection. The muscles of birds remain free from invasion, though the worms multiply in their intestines; and trichinæ do not breed in cold-blooded animals.

The earliest experiments which threw light upon the way in which the trichina gains access to the human body were those of Herbst, who in 1851 showed that when flesh containing trichinæ was given to animals, they in turn became infested with the parasite. He did not trace the intermediate steps, and naturalists were led astray by Küchenmeister's hypothesis that the trichina was a stage in the development of *Trichocephalus dispar*. In 1860, however, Virchow and Leuckart showed that this was not the case. They fed animals with trichinous meat, and found that the worms at once escaped from their capsules and developed into sexually mature entozoa of a previously undescribed kind.

The experiment has since been repeated by many observers, and with uniform results. An animal to which trichinous flesh has been given is killed at the end of forty-eight hours; and the mucus lining its alimentary canal is found to contain numerous minute living worms already sexually mature. They are not visible to the naked eye, but are easily recognised under the microscope. The majority of them are females; these measure one twelfth to one ninth of an inch in length (2—3 mm.). The males are smaller, being only $\frac{1}{15}$ to $\frac{1}{14}$ of an inch long (1.2—1.6 mm.). If the host be left until the sixth day before it is killed, the female worms contain free embryos, which may be watched under the microscope as they are extruded and move about the field. It is estimated that at least 150 young are produced by each female trichina.

It is therefore clear that from the sixth or seventh day onward after the ingestion of trichinous flesh by man or any other animal, living trichina-embryos are poured in large numbers into the alimentary canal. Thence they find their way to the peritoneum, the lymph-glands, the viscera, and the muscles. Probably the embryos enter the blood-stream, and ultimately reach the tissues through the walls of the systemic capillaries. In the voluntary muscles alone do they find the conditions necessary for their further development; for if they enter other tissues they either perish or migrate again until they reach their proper seat. Even when they have reached the muscles they seem at first to move on in the course of the fibres, for they are found in larger numbers towards their tendinous insertions than elsewhere, as though these formed obstacles arresting their further progress. They have sometimes been distinctly seen within the sarcolemma, but more often between the fibres. Once lodged in the muscles they grow rapidly, and acquire an alimentary canal, and rudimentary sexual organs. At this stage they roll themselves up, and round each of them a capsule is developed about the fourth week after their immigration. It consists originally of an exudation due to the irritation

of the tissues which their presence causes; and after a time calcification begins. Dr Thudichum says that in rabbits he has seen the capsules perfectly opaque within ten weeks; but in the human subject calcification is not complete in less than a year, and Rupprecht found capsules still transparent in the muscles of a man who had been attacked by trichiniasis two years before.

Calcification of the capsule does not always destroy the trichina within, which remains quiescent, waiting for the death of its host to call it into activity; but in course of time it may itself perish.

The number of trichinæ which may be contained in the muscles of the human body is enormous. From data obtained in animals it has been estimated at from twenty to thirty millions.

After the invasion of trichina-embryos, the muscle-fibres lose their striation, become brittle and homogeneous, and show numerous minute fissures. To the naked eye the muscles appear of a pale reddish-grey colour.

Trichiniasis.—At the time when Leuckart and Virchow were working out experimentally the life-history of the trichina, Zenker had just observed a case in which the parasite caused a fatal illness in the human subject. On January 12th, 1860, a girl was admitted into the Dresden Hospital suffering from what at first appeared to be fever. She died, and on *post-mortem* examination the characteristic lesions of enteric fever were absent, but the muscles were full of living trichinæ as yet unencapsuled. The girl had been in the service of a butcher, who had killed a pig about a week before her illness commenced. She had been employed in making sausages of the pork, and had very likely eaten some of it in an uncooked state. The sausages and a ham from the pig were examined and found to contain trichinæ. It was also ascertained that the butcher and two other persons had been taken ill about the same time, but had recovered.*

Symptoms.—Many cases have since been recorded, so that the clinical aspect of trichiniasis is now well known. At first it is undistinguishable from those of other febrile diseases. The patient complains of loss of appetite, sleeplessness, and a sense of extreme lassitude and depression, and it may be of nausea and vomiting; but at the end of a week, or a little later, the arms and legs begin to be stiff and painful. The elbows and knees become flexed, and great pain is produced by any attempt to straighten them. After a time the limbs are sometimes rigidly extended, and the body is as though affected with opisthotonos. The muscles are tender to the touch; when grasped they feel hard and swollen, and as if they were distending the fasciæ in which they are enclosed. The jaws are often closed for several weeks, after which the muscles may become suddenly relaxed again, with an audible crack. Movements of the eyes are painful, and the power of accommodation is lost. The breathing becomes shallow and hurried from implication of the thoracic muscles, until coughing, sneezing, and yawning become almost impossible.

About the end of the second week the eyelids are observed to be œdematous, and sometimes the whole face and neck. Afterwards the legs and the parts round the joints become swollen.

In trichiniasis the temperature seldom rises above 102° Fahr. There

* Leuckart ('Die menschlichen Parasiten,' vol. ii, p. 525) recalls the early observation of a case of "pneumonia and pericarditis with muscular rheumatism," *i. e.* of trichiniasis, at Bristol, by Dr Wood, in 1835 ('Lond. Med. Gaz.,' p. 190).

is often profuse sweating, and a miliary eruption may appear. The pulse is sometimes very rapid; the tongue is red, slightly furred, and rather dry; and diarrhoea may occur, especially in severe cases.

If the patient dies, it is usually in the fourth or fifth week of the attack, but sometimes much earlier—from pyrexia, pneumonia, or ulceration of the colon. If he recovers, convalescence is tardy, lasting three or four months.

Diagnosis.—Well-marked cases are not difficult to recognise; for the symptoms, taken together, are unlike those of any other disease. Manson says that the nearest approach to trichiniasis is Beri-beri—one a myositis, the other a neuritis. The diagnosis may be established by the microscopical examination of a minute portion of muscle removed during the patient's life. Rupprecht found many adult worms in the stools of patients to whom large doses of calomel had been given.

The disease often occurs as an epidemic. Thus, at Plauen, in 1862, thirty persons were attacked about the same time. At Hettstädt, near the Hartz Mountains, four separate outbreaks of trichiniasis occurred between September, 1861, and March, 1864; in the most important of these 158 persons were attacked, of whom twenty-eight died. Other epidemics have been observed at Stassfurt, Dessau, Leipzig, and elsewhere.

In England the only cases of trichiniasis which have been detected during life, or attended with symptoms (since Wood's in 1835) are those recorded in 1871 by Mr Dickinson, of Workington, in Cumberland. The patients were a farmer's wife, his daughter, and a serving man. They had all been eating sausages and pork from one of the farmer's home-fed pigs, the flesh of which was found by Cobbold to be full of trichinæ.

Possibly cases due to the migration of trichina-embryos into the muscles do really occur from time to time in this country, but are overlooked. Indeed, the fact that encapsuled trichinæ are now and then found in the bodies of those who have died of other complaints, shows that the necessary conditions for the entrance of this parasite into the human body are not entirely absent in England. But even encapsuled and harmless, the trichina is rarely met with here as compared with its frequency in Germany. In Dresden, Zenker detected it in four out of 136 *post-mortem* examinations; and in Berlin Virchow found it six times in the course of a single year; but often only a small number of trichinæ were present, so that they might easily have been overlooked. Probably in such cases the symptoms, if any, would be very slight, and might be attributed to "rheumatism." Even on the Continent the disease is far more common in Northern Germany than elsewhere, and most common of all in Saxony. In districts where pork is not eaten raw it is very rare. Next to Germany, it is most common in the United States; but is also recorded in India, and by Guillemard, at Amoy.

The severity of a case of trichiniasis appears to depend upon the number of embryos which penetrate into the muscles. Thus the most important guide to *prognosis* is believed to be the state of the patient's limbs, as regards mobility and pain on movement. The mortality in different epidemics varies greatly—from 2 to 30 per cent. Children are said almost always to recover.

Treatment.—The first indication is to expel as many of the trichinæ as possible from the alimentary canal. For this purpose castor oil is recommended, or calomel in full doses, repeated at intervals. Manson advises

the use of thymol. When once the worms have left the alimentary canal and reached the muscles, it is very unlikely that any drug will avail to destroy them.*

For the *prevention* of trichiniasis, animals intended for human food must be kept out of the way of trichinous flesh. Pigs doubtless derive trichinæ from the rats which infest the sty, for rats are the most frequent trichinæ-bearers of all animals, and devour their own dead. The trichina retains its vitality after the flesh containing it has become putrid; and after smoking, pickling, freezing, and even heating to a degree as high as is sometimes reached in the deeper parts of half-cooked pork.

But even if flesh containing trichinæ should by accident be used for human food, all danger is obviated if the meat is cooked throughout.

Filaria sanguinis nocturna.†—In 1866 Wucherer detected, in cases of chylous urine occurring in Brazil, certain minute living organisms, the embryos of a nematoid worm. Six years later, in 1872, the late Dr T. R. Lewis, in India, discovered similar embryos in the blood. It was soon found that this was no isolated occurrence, and that the hæmatozoon (as it was called) was by no means limited to persons affected with chyluria. In Southern China, for instance, among 1000 natives taken at random, about 100 are said to be infested with this parasite. In that district similar larval entozoa (as of *Filaria immitis*) are very common in the blood of dogs and in many species of birds, so that their presence in man did not surprise experienced helminthologists. Among those who harbour the *Filaria sanguinis hominis* (as Lewis termed it) some appear to be in perfect health, but others suffer from lymphangitis with varicosity of the inguinal lymphatics, lymph-scrotum, elephantiasis, or chyluria.

The discovery of the parent worm from which the embryos found in the blood are derived, was effected in 1876 by Dr Bancroft, of Brisbane, in Australia. He first obtained a dead specimen from a lymphatic abscess in the arm, and afterwards four living ones from a hydrocele of the spermatic cord. These he sent to England to Dr Cobbold, who gave to the entozoon the name of *Filaria Bancrofti*. It is now ascertained to be the mature form of the nocturnal *Filaria sanguinis*. The length of the female is from three to three and a half inches; its breadth from $\frac{1}{100}$ " to $\frac{1}{90}$ ". It has a circular mouth, destitute of papillæ, a narrow neck, and a bluntly pointed tail. Its body is smooth, and of an opaline appearance, and it has been described by Dr Manson as looking "like a delicate thread of catgut, animated and wriggling." This observer, in 1880, while operating on a case of lymph-scrotum, removed at the same time a portion of a living worm, and showed that it lay in the interior of a dilated lymphatic. He also proved that the parasite is naturally viviparous, for he saw fully formed embryos, exactly like those which are found in the blood, escaping from the vagina. The embryo in the blood is always enclosed in a delicate sac or sheath, which fits it accurately, except that a collapsed or unoccupied part is seen projecting behind either the head or the tail, according to the direction in which the worm happens to be moving; and

* Friederich gave picrate of potash in one case which ended in recovery; but live trichinæ were afterwards found in this patient's muscles. Glycerine has been administered in cases of trichiniasis in the United States, in the hope of killing the trichinæ by abstracting water.

† *Filaria*, a threadworm (*filum*, a thread). The worms discovered in the human blood belong to at least three species—*F. nocturna*, *F. diurna*, and *F. perstans*.

it seems now to be certain that this sheath is nothing else than the envelope or shell of the ovum, which, as the embryo develops, yields before its vigorous spontaneous movements, and so continues to be stretched out over the worm. The size of these embryos is such that they can traverse the lymph-paths through any glands that may come in their way, and so passing on from the lymphatic vessel in which the parent worm lies into the thoracic duct, at length reach the blood-vessels. Their diameter, indeed, is only about $\frac{1}{3200}$ " , not more than that of the leucocytes which circulate through the lymph-glands; their length is about $\frac{1}{50}$ " .

So long as the embryos of the filaria remain in the blood, they continue the same size, and show no indication of further development. This fact is of itself sufficient to suggest that they are waiting to be transferred to some other host; and it seems to have occurred independently both to Dr Bancroft at Brisbane, and to Dr Manson at Amoy, that this host might probably be some species of mosquito which feeds on human blood. Dr Manson came to this conclusion, in 1877, and afterwards discovered that the filaria above described is only present in the cutaneous blood-vessels during the night. At about 6 or 8 p.m. they begin to make their appearance; by midnight their numbers reach the maximum; as morning approaches they become fewer and fewer; by 8 or 9 a.m. they cease to be discoverable. Probably during the day they may be all collected in the pulmonary capillaries, or in those of the deeper structures generally, and at night visit the vessels of the skin.

Dr Stephen Mackenzie recorded in the 'Pathological Transactions' for 1882 observations made at intervals of three hours for weeks together, and the periodicity was found to be as complete as it possibly could be. Dr Mackenzie also submitted his patient to the experiment of having his habits of life reversed, so that for nearly three weeks he remained out of bed all night, and rested in the daytime, the hours of his meals being arranged accordingly; the result was that during this time the filariæ were found in the blood during the day, but not at all, or only in much smaller numbers during the night. Moreover, they were not tempted to come out during the day by even the thickest London fog.

The filaria's habit of entering the capillaries of the integument at night-time is adapted to bring them within reach of the proboscis of a nocturnal blood-sucker like the mosquito.

Dr Manson persuaded a Chinaman, known to be infested with the filaria, to sleep in a "mosquito house." Next morning the gorged insects were caught and examined. The blood in their stomachs was found to contain filariæ in even larger numbers than that of the man from whom it had been derived. It is supposed that they become entangled in the proboscis of the mosquito, and are so removed from the blood-vessels. Having reached their new host, they lose their sheaths, grow to the length of one thirtieth of an inch, and acquire a distinct alimentary canal, a mouth with three or four nipple-like papillæ, and rudiments of generative organs. Their movements also become extremely active. These changes are completed in from four to six days. During this time the mosquito has been digesting her single meal and maturing her own ova; she now deposits them on the surface of water, after which she dies, and probably falls into the water on which her eggs are laid.

The filaria next makes its way out of the body of the dead insect into the water. The next step is that it is swallowed by a human being, from whose stomach it bores a passage into the thoracic duct or into some lymphatic vessel; and along this it then works up stream, in

obedience to some strange instinct, until it reaches a spot which it takes for its permanent abode. Here we must suppose that it is joined by another parasite of the opposite sex, after which it furnishes to the lymph-channels and to the blood-current of its host those swarms of larvæ which formed the starting-point of our inquiry as to the life-history of the entozoon. How long the parent worm lives we do not as yet know; but a case observed by Dr Manson shows that it may be not less than thirty-two years; he found living filariæ in the blood of a man aged fifty, who had had lymph-scrotum from the age of eighteen. There is some reason for supposing that it may be killed by an acute disease in the host.

Dr Stephen Mackenzie's patient was attacked with rigors as the result of going out of the hospital on a cold and windy day in October, and after the following day no embryos were ever discoverable in his blood. Pleurisy set in, and an abscess formed near the left collar-bone; and when he died two and a half months later no trace of the worm could be found. Dr Mackenzie supposes that it became dislodged during the rigor, and reaching the termination of the thoracic duct on the left side of the neck, excited both the pectoral abscess and also the pleurisy. However this may be, it seems clearly to have perished from an early period of the man's fatal illness, and its body to have disappeared.

An important matter to determine is how the filaria injures the lymphatic vessels. Dr Manson's ('Path. Trans.', 1882) hypothesis is that so long as the discharge of embryos goes on after the manner above described, the parasite is perfectly innocuous to its host. But from some cause or other it happens in certain cases that instead of the larval filariæ enclosed in their sheaths, ova in a much earlier stage of development are extruded from the maternal vagina. Manson has twice obtained such ova from the lymphatics. According to Manson, they measure $\frac{1}{750}$ " in breadth by $\frac{1}{500}$ " in length; according to Cobbold, $\frac{1}{500}$ " by $\frac{1}{1000}$ ". In either case their transverse diameter is far greater than that of the embryos; and they thus fail to pass along channels which the embryos would find no difficulty in traversing. Manson supposes, for instance, that when they are carried by the lymph-stream to a gland they become impacted in the small channels formed by the afferent vessel. The result must be, as anastomosing paths become one after another obstructed, a more or less complete stasis of lymph, not only in the neighbourhood of the spot where the parent worm is situated, but also in the whole of one or both of the lower limbs, and in the scrotum.

The pathological effects of the filaria will be described hereafter in the chapter on affections of the urine and in that on elephantiasis.

Filaria sanguinis diurna is only known on the West Coast of tropical Africa. It appears in the cutaneous blood-vessels only during the day. Its host is unknown, but is probably some dipterous day-fly. Manson conjectures that its mature or parental form may be *Filaria loa*.

A third worm whose embryos may infest the human blood is *Filaria sanguinis perstans*, which may be found either by day or by night. It also has only been discovered on the Congo and the Guinea Coast. Like the diurnal species, it does not appear to have any relation to chyluria and lymphatic diseases.

The Guinea-worm (*Dracunculus medinensis**) is an enormously long

* The specific name refers to their frequent occurrence in Medina and other parts of Arabia (cf. Plutarch, 'Sympos.', viii, 9).

nematoid worm from one to three, or even five, feet in length, of which the female only is known. It inhabits the subcutaneous tissue, usually of the legs, to which it gains access from the water-tanks in India, Egypt, Arabia, the coast of Guinea, and other hot countries. Its effects are purely those of local irritation, but they are often severe.

Dr Manson has made out its history. The worm lays her eggs, or, rather, embryos, for it is viviparous in water. They then enter the body of a common fresh-water crustacean, *cyclops*, which apparently conveys them to the human stomach in drinking-water. Thence the embryo bores its way to the subcutaneous tissues, always seeking the lower extremities, where it is most likely to find water. The worm (how and where impregnated is not yet known), as soon as the sore it makes is opened under water, pours out its living embryos. If the leg be moistened with water even, the instinct of the worm makes it eject a little milky fluid containing thousands of them.

A suppurating sore is thus produced which may persist for years. The natives draw out a portion of the intruder and wind it round a stick, which they attach to the patient's leg; but before it is all thus extracted, the worm often breaks. The modern treatment is to cut down on the parasite, lay open its track, and dress the wound with phenol or other germi- and vermi-cidal application.

Trematoda.—Two members of this very distinct group of animals, mostly parasitic in habit, have been found in the human body. One of these, the liver-fluke (*Fasciola hepatica* or *Distomum hepaticum**) infests the gall-passages, but it is very rarely found in man, at least in civilised Europe—it is said to be more common in Bosnia. In sheep it causes a common disease, the sheep-rot, after passing through an intermediate stage, first as a free cercaria, then in the tissues of a fresh-water snail, *Limnæa*, and lastly as a free-living inhabitant of wet pastures.

The other Trematode worm (*Bilharzia hæmatobia*) inhabits the pelvic veins of persons living in Egypt and Natal, and produces serious hæmaturia. Its ova are passed out in the urine, and are recognised by a pointed process at one end, or in some cases by a second spike projecting from the side of the egg. This worm will again be noticed in the chapter on Hæmaturia. It was described by Griesinger, who named it after Bilharz, its discoverer in Cairo in 1855.

Acanthocephala.—A thorn-headed worm, *Echinorhynchus*, sp., has only once been certainly discovered in the human intestine by Lambl ('Prager Vierteljahrschrift,' Feb., 1849). A second case, reported from Netley in 1872, is doubtful. It is common among pigs in England.

Acarina.—A parasite belonging to the class Arachnida, and to the same order of mites as that to which the *Acarus* and the *Demodex* appertain, is occasionally found in the liver, but it is always dead and encysted. It is called *Pentastoma tænioides*, and has no clinical significance. We have frequently noted it at Guy's Hospital.

Psorospermia have been occasionally recorded in the human liver.

* *Fasciola*, dim. of *fascia*, band or tape. *Distomum*, a name given under the strange mistake that the two suckers are mouths.

These oval encysted parasites, probably a phase in the development of Gregarinidæ, are frequently found clustered into opaque white nodules in the livers of rabbits, and when extremely numerous produce emaciation and death. They have been recognised in the alimentary canal and liver or other glands, not only of mammals and fishes, but also in insects, mollusks, and worms, and are probably ubiquitous. They are rarely met with in man, and still more rarely produce symptoms; but possibly some cases of tubercles in the liver have really been examples of this disease. A remarkable case was published by Mr Silcock in the 'Path. Trans.' (xli, p. 320). An excellent account of our present state of knowledge concerning these parasitic monozoa, with plates and ample references, is contributed by Dr Delépine to the same volume of the 'Pathological Transactions' (1890, p. 346). The parasitic nature of the organisms described in cancerous cells, and regarded as allied to psorospermia, has been much contested, and at present has not been established (vol. i, p. 97).

The remaining animal parasites of the human body are the amœbæ, or plasmodia, which have been described in the chapter on Malaria (vol. i, p. 397).

DISEASES OF THE PERITONEUM

June 28th, 1754.—By way of prevention therefore, I this day sent for my friend Mr Hunter, the great Surgeon and Anatomist of Covent Garden ; and tho' my Belly was not yet very full and tight, he let out ten quarts of water, the young sea-surgeon attending the operation, not as a performer but as a student.

FIELDING : *Voyage to Lisbon.*

ACUTE SEPTIC PERITONITIS—*Clinical symptoms—Origin : secondary to visceral disease—bacillary—puerperal—uræmic—extreme rarity of idiopathic acute peritonitis—Morbid anatomy—Local and circumscribed peritonitis—Sub-phrenic suppuration—Diagnosis—Treatment : medical and operative.*

CHRONIC PERITONITIS—*Peritoneal adhesions—Thickening, general and local—liver, spleen, omentum—Locular and general effusion—Frequency—Causes.*

TUBERCLE OF THE PERITONEUM—*Anatomy—Symptoms—Age and sex—Diagnosis—Prognosis—Treatment.*

CANCER OF THE PERITONEUM—*Commonly secondary—Anatomy—Symptoms.*

ASCITES—*Physical signs—diagnosis—Chylous ascites—inflammatory and obstructive ascites—Prognosis—Treatment by drugs and by paracentesis.*

THE peritoneum is part of the great body-cavity (*cœlom*) formed by the mesoblast splitting into somatopleure and splanchnopleure. It is a huge areolar space or lymph-sac, and its most intimate pathological relations are not with skin or mucous membranes—not even with the joints or the so-called arachnoid space—but with the pleura, pericardium, and tunica vaginalis.

The diseases of these three divisions of the same original cavity are very similar : acute inflammation, serous or purulent, traumatic or septic ; chronic adhesive inflammation, with more or less thickening and hypertrophy ; chronic irritative effusion, and passive dropsical effusion—hydrothorax, hydropericardium, and ascites. All three serous membranes are liable to be invaded by tubercle, and also by cancer. All three are prone to follow the pathological fate of the viscera which they cover ; they are all apt to suffer in the course of Bright's disease ; and lastly, we not infrequently see them all affected together, often by inflammation or tubercle, and more rarely by cancer.

On the other hand, rheumatism seldom or never affects the peritoneum as it does the pericardium, and peritonitis is seldom or never the result of exposure to cold, as pleurisy undoubtedly is.

Although peritonitis is not so constant a companion of inflammation in any abdominal viscus as is pleurisy of pneumonia, yet it is most often secondary to inflammation of one or other of the organs it covers, the effect ranging from the adhesions which slowly form about an enlarged liver, spleen, or ovary, to the rapid and violent inflammation which blazes up when pus or fæcal material finds its way into the cavity. The most important viscera from this point of view are, for men-patients the intes-

tines and the stomach, next the gall-bladder, and then the urinary bladder. In the case of women the ovaries, Fallopian tubes, and uterus set up peritonitis more frequently than any other viscera.

We may divide peritonitis, as we see it clinically, into the following varieties:

1. Acute, infective, suppurative, virulent, affecting the whole cavity of the abdomen: always secondary to perforation or to some septic process, as in puerperal fever after childbirth.
2. Acute, sero-fibrinous, or sero-purulent, but local and circumscribed: traumatic, or secondary to visceral inflammation or to Bright's disease.
3. Chronic, adhesive, local, hypertrophic.
4. Chronic, with serous effusion, sometimes latent.
5. Tuberculous, with adhesions, thickening, and effusion, serous or sero-purulent, occasionally hæmorrhagic.
6. Cancerous, with adhesions, thickening, and effusion, usually of blood-stained serum.

ACUTE SEPTIC PERITONITIS.—This disease is both fatal and common. In 1873, taking a year at hazard, of 434 inspections made at Guy's Hospital, in at least 52 death was directly attributable to acute peritonitis, or nearly one in eight. In 1898, twenty-five years later, of 449 inspections made in the same hospital, 42 were due to peritonitis. In almost every case fatal peritonitis is septic, and secondary to injury or disease of the abdominal viscera, or to an infective process.

Symptoms.—These vary greatly in different cases, and they are often so masked as to make recognition peculiarly difficult.

If we take the case of an apparently healthy person, who is suddenly seized with peritonitis, we shall find him lying in bed on his back, with his knees drawn up, his features pinched and drawn, his eyes sunken and dark. There is no sweat, and his face is pale, but its watchful, anxious look is like that of a patient with rheumatic fever.

He complains of sharp, sometimes cutting or burning pain in the abdomen. This is constant, but liable to aggravation if he changes his posture, if he coughs or sneezes or strains, and also when there is movement of gas in his intestines. He dreads pressure, even the lightest application of the hand. Tenderness may either be diffused equally over the whole surface, or may be most intense at some particular spot, probably the starting-point of the inflammation. The movements of the diaphragm cause so much pain, that in breathing the patient instinctively uses the upper ribs only; the inspirations are therefore shallow, and are repeated forty or fifty times a minute.

An attack of peritonitis sometimes begins with sharp rigors. These are followed by more or less fever. The *temperature* may rise to 104° or to 105°; but it is important to remember that a normal temperature gives no certainty that peritonitis, and even purulent peritonitis, is not present. We found the same exceptions in cases of pleurisy, and even of empyema. When death is approaching, the temperature falls to normal, or below it, and the hands and feet are icy cold. As Mr Symonds has remarked, the temperature keeping up during the period of repose in perforative peritonitis—when shock is over, and vomiting and pain have yielded to starvation and opium—is often a valuable sign that the danger is not over (Address to the Hunterian Society, Feb., 1899). The *pulse* is frequent, ranging from

100 to 150. At first it is often small, hard, and strong—the wiry pulse. In the later stages it becomes still smaller, feeble, compressible, irregular,—the thready pulse; it may be imperceptible for some hours before death. In fatal cases this usually occurs by collapse, the mind often remaining clear to the last moment. But there may be great restlessness towards the end, the patient tossing about in delirium for an hour or two before death, and careless of the posture he assumes.

The fact that the inflammation penetrates to the subserous and muscular coats may be one reason why there is *constipation* in peritonitis. The bowels can, however, be moved by giving purgatives, if this dangerous mistake is committed; and in puerperal peritonitis diarrhoea is as frequent as constipation.

Vomiting is sometimes the earliest symptom of acute peritonitis. The *tongue* is small, furred, and dry. When the case is approaching a fatal termination the patient is sometimes tormented by obstinate hiccough; micturition is often painful and difficult, particularly in pelvic peritonitis, and the urine is scanty and high-coloured.

The surface of the abdomen is not only tender to the touch, but also much harder than natural by a conservative reflex action, which interferes with physical examination, but is of great diagnostic import. After a time it is distended, sometimes enormously, with gas which accumulates in the paralysed bowels—a condition called *tympanites* and *meteorismus* by the Greek writers. The abdominal walls early become immovable in breathing; the recti and other muscles are rigid, and the semilunar and transverse markings may be plainly discerned through the integuments.

At first the percussion-note is everywhere tympanitic, usually of a higher pitch than in health, but after a time it may become short and tympanitically dull from extreme distension. Sometimes we can detect one or more circumscribed regions of dulness, which point to local effusion. A friction-sound like that of pleurisy may be detected by the stethoscope occasionally, but seldom in acute general peritonitis. Fluctuation points to the case becoming chronic, and an audible rub to its being local.

As stated above, symptoms are often so little marked that it needs much care and experience not to overlook the presence of peritonitis.

Ætiology.—Acute peritonitis is probably always of septic origin, and is most commonly caused by extension from a viscus—as a rule a hollow viscus. For the cavities in the abdomen, besides being liable to undergo perforation and to discharge their contents into the serous cavity, are also more subject than the solid viscera to those septic forms of inflammation which, when they reach the peritoneal surface, excite the same unhealthy action there. Thus cirrhosis of the liver does not set up acute or general peritonitis, nor does chronic inflammation of the ovaries, nor the swollen spleen of ague or of enteric fever; but a ruptured hepatic abscess, a perforated gall-bladder, a sloughing appendix cæci, a ruptured bladder or ovarian cyst, or septic conditions of the uterus or Fallopian tubes after delivery or abortion—these are almost certain to produce acute and fatal peritonitis. An embolic block in the spleen from ulcerative endocarditis may occasionally set it up, and, in a few instances noted in the *post-mortem* records at Guy's Hospital, its starting-point was suppuration of the kidney. Another exceptional origin is in softening of tuberculous lymph-glands.

A perforating ulcer of the stomach at once sets up general septic peri-

tonitis; and a perforating intestinal ulcer will do the same; most frequently a typhoid ulcer, but even tuberculous ulcers, which are much less likely to perforate, have done so in several cases in our experience. Intestinal strangulation always causes septic peritonitis; but chronic obstruction and mere faecal impaction may also produce fatal peritonitis by ulceration and perforation of the dilated part of the gut above the seat of obstruction. Lastly must still be mentioned surgical operations on the abdominal organs as occasional causes of peritonitis, although this is now very rare.

Typhlitis in both sexes and pelvic suppuration in women are probably the two most frequent of all causes of secondary peritonitis; and next, perforation of the stomach or bowel.

In some of these cases the cause is obvious; in others obscure. The patient may be attacked when apparently in perfect health, as when the starting-point of the disease is a deep ulcer of the stomach or duodenum; and even when it is a typhoid ulcer of the ileum, the occurrence of peritonitis may be the first indication of illness, for enteric fever may in exceptional cases be latent. Another of these obscure causes of peritonitis is typhlitis.

In most, if not in all cases, puerperal peritonitis starts from a septic or sapræmic state of the lining membrane of the uterus, which infects the peritoneum either along the Fallopian tubes or through the sinuses and venous channels, which are often filled with pus. Miscarriages, again, are not rarely followed by peritonitis; and it may also be set up by extra-uterine foetation, a pelvic hæmatocele, gonorrhœal suppuration of a Fallopian tube, or sloughing of an ovary. The most careful vaginal examination should therefore be made in every case of obscure peritonitis in a woman.

When we cannot trace acute peritonitis to a primary local disease, there is no doubt that it is always secondary to some general septic infection. Sometimes the bacilli of tubercle, sometimes the streptococci of suppuration, and sometimes the specific microbe of Enteric fever, or a virulent form of *Bacillus coli* is found in the exudation. Peritonitis is very rare after Scarlatina; but epidemic peritonitis of doubtful origin has been ascribed to the specific microbes of erysipelas or of influenza.

Of predisposing causes of acute peritonitis, Bright's disease is the only one well ascertained. Of this sixteen cases were observed in Guy's Hospital between the years 1854 and 1872. The inflammation was generally suppurative; and there was often a marked absence of vascular injection of the serous membrane. The kidneys were, as a rule, enlarged, and in a more or less advanced stage of tubal nephritis; but in three cases they were contracted, with a wasted cortex.

Rheumatic peritonitis has been described. If the term is used in its proper sense, as denoting relation to rheumatic fever, it can only be said that such cases are exceedingly rare, if they exist at all. It is certainly strange that the peritoneum should not share the so frequent fate of the pericardium and pleura in rheumatism, as it does in renal disease; but such exceptions meet us everywhere in pathology.

In 1874 several children at a school at Wandsworth were attacked at the same time with acute peritonitis. The late Dr Anstie investigated this epidemic; and the conclusion at which he arrived was that the disease was caused by exposure to the influence of sewer gas. It was in making an autopsy in one of the fatal cases that he received the wound in his finger which cost his valuable life. Dr Shirley Murphy has

since met with the following case, which appeared to be attributable to a similar cause.

A woman, aged thirty-six, died on her way to the Homerton Fever Hospital. The autopsy showed that acute peritonitis was the cause of death. The coils of intestine were matted together by lymph, but the intestines, uterus, and other viscera were healthy, and no local starting-point for the inflammation could be discovered. It was afterwards ascertained that the patient had been living in a house the drain-pipe of which was obstructed, so that for two or three weeks the sewage had been spread over the yard. When first taken ill she shivered, fainted, and vomited; next day she complained of pain in the left iliac fossa, with purging; and two days later she died.

Anatomy.—The changes which occur in acute peritonitis present considerable variations, but rather of degree than kind. They are essentially the same as in other serous membranes. The surface first becomes reddened, from injection of the minute vessels. This injection is often not uniform, but is especially marked along two longitudinal lines, which run over the bowel, at a little distance from one another, parallel with the attachment of the mesentery. The explanation of this appears to be as follows:—In health atmospheric pressure keeps every part of the serous surface in contact with some other part; the intestines are not (as one is apt to suppose) regularly rounded tubes; they are flattened against one another and the abdominal wall. But the distension caused by peritonitis leads them, by physical necessity, to assume a cylindrical form; and the result is that blood is forcibly drawn into the angular spaces between them. The red lines so produced were therefore called “suction-lines” by Moxon. They are wanting when the intestines fail to become distended, when air has access to the peritoneal cavity, and perhaps also when inflammatory effusion is poured out very early and in large quantity.

The further morbid appearances vary according as the inflammation leads to the effusion of lymph or to suppuration without adhesions. In the former case the membrane becomes dull and lustreless, and very soon it presents shreds and small patches of fibrin; in the latter case it is even more lustrous than in health, and feels greasy to the touch.

The coagulated lymph (*i. e.* fibrin) forms a layer of greater or less thickness, which may either be limited to certain parts or cover the whole surface of the serous membrane. Microscopically it consists of fibrinous threads which cross one another in all directions, leaving interspaces in which are masses of leucocytes. The observations of Rindfleisch, Ranvier, and Klein have proved that some of the cells are derived by proliferation from the endothelium; others are exuded leucocytes. Klein, in his ‘Anatomy of the Lymphatic System,’ describes the lesser omentum and mesentery as oedematous and swollen to five times their natural thickness in cases of experimental peritonitis in animals; and effusion into the sub-serous tissues accounts for the fact that after death from peritonitis the serous membrane can be stripped off so readily.

When the inflammation is severe, more or less turbid fluid is also effused, and under the microscope this always contains numerous leucocytes. When they are numerous enough to cause opacity, the liquid is said to be purulent; but every gradation may occur between inflammatory serum and pure pus. All depends upon the proportion of leucocytes to the fibrin and serum.

If the peritonitis is of moderate intensity, it leads to adhesion of the opposed surfaces of the serous membrane. This appears to be brought about by the cells embedded in the fibrin. Some pass into spindle-cells,

and ultimately form perfect connective tissue; while others develop into blood-vessels, the walls of which at first consist entirely of opposed cells. These new vessels are very soft, and readily give way if the exudation-fibrin is subjected to pressure or traction. Spots of hæmorrhage are therefore often seen, and sometimes the amount of blood effused is very great. What is called hæmorrhagic peritonitis appears to arise in this way.

The adhesions resulting from peritonitis may be universal, the cavity being obliterated and the abdominal organs united together by dense connective tissue, from which they have to be dissected out when a *post-mortem* examination is made. More frequently the opposed surfaces adhere in certain places only. They still move on one another as in health; and thus the tissue which connects them becomes stretched into bands or cords which may acquire a considerable length. These, as we have seen, are frequent causes of intestinal strangulation (pp. 414, 425).

Even after effusion of a large quantity of fluid, the two surfaces may at last come together and unite. The connecting fibres seem to be formed from the cells of a layer of granulation tissue, which covers each surface, and is derived from the endothelium of the serous membrane. Even pus may dry up and become converted into a caseous mass embedded in the fibrous tissue of the adhesions.

Circumscribed peritonitis.—In many cases of peritonitis, and those the most virulent, the inflammation starts from some one spot and rapidly spreads over the whole abdomen. This process is accelerated by the movements of the intestines, so that parts already inflamed are brought into contact with others and infect them. Moreover, when the stomach or intestine has been perforated, the extravasated matters may be afterwards discovered in the most distant parts of the cavity.

But even the acute septic peritonitis of which we are now speaking, does not always spread over the whole of the peritoneum. The omentum often seems to check its progress, or the pus poured out may be limited by agglutination of two serous surfaces. Thus peritonitis starting from the uterus may lead to a circumscribed abscess occupying the pelvis; in one case of this kind the pus was discharged through the bladder during life, and in another through the umbilicus. So also peritonitis arising from ulceration of the intestine often gives rise to localised collections of pus, especially when the cæcal appendix is the starting point of the disease. The abscess then forms a swelling in the right iliac fossa. It sometimes points near the crest of the ilium, but not infrequently it passes down below Poupart's ligament and discharges in the groin. In other cases, again, it makes its way backwards towards the loin, and upwards to the liver; or it opens into the rectum or bladder.*

The bowels sometimes communicate freely with an abscess of this kind, and much fæcal matter may be discharged with the pus. In one case for a considerable time before the patient's death almost all the fæces passed through an opening in the groin; in that instance the abscess was secondary to cancerous disease of the cæcum. The fact, however, that the pus discharged from a circumscribed abscess in the abdomen has a fæcal odour does not prove that there is a communication with the intestine; for matter collected in the neighbourhood of the bowels may acquire such an odour

* Such abscesses are sometimes difficult to distinguish from those caused by diseased bone, and the difficulty is increased by the fact that when the crest of the ilium lies in the way of the pus, part of it sometimes becomes denuded of its periosteum, so as to be within easy reach of a probe.—C. H. F.

from diffusion of the intestinal gases. Again, as an exception, the intestine may be perforated from outside by a suppurating gland.

A rare form of circumscribed abscess in the peritoneal cavity is limited to the sac of the lesser omentum. In one such case this cavity contained two or three pints of pus, the inflammation having started from disease of the pancreas.

Subphrenic abscess.—Suppuration limited to one or other hypochondrium is by no means uncommon. In the 'Guy's Hospital Reports' for 1873-4 Dr Fagge recorded several cases of this kind. In some of them the abscess started from an ulcer of the stomach, or other disease in the neighbourhood, but in others it resulted from some direct injury, particularly when seated in the right hypochondrium. In cases of the latter kind the suppuration is often preceded by a circumscribed effusion of blood, which may itself form a distinct swelling, analogous to the pelvic affection known as peri-uterine hæmatocele.

In one of the most interesting of these cases of subphrenic abscess, the patient had been kicked in the left side, and came in with a large rounded tumour in the hypochondrium. After a time we found that air had entered it, for curious musical sounds, synchronous with the heart's beat, were heard over it, and the percussion-note became tympanitic. Yet there were no symptoms indicative of constitutional disturbance, and the man left the hospital refusing to believe that anything serious was the matter with him. Some time afterwards he returned, saying that he had vomited a quantity of matter, and that the tumour had disappeared; on examination no trace of it could be discovered.

Cases of abscess in the hypochondrium do not often terminate so favourably.

When the subdiaphragmatic abscess, or hypophrenic empyema, to use its correct name, communicates with the stomach or intestine, there is naturally gas as well as pus in the cavity.*

Prof. Leyden, of Berlin, has since proposed the title of "subphrenic pyopneumothorax" for this important pathological condition ('Zeitschr. f. kl. Med.,' 1880); but pneumothorax is just what it is not, and there is no reason to alter the generally accepted name under which Dr. Fagge described it.

In the left hypochondrium, beside cases of traumatic origin, the most frequent are those which depend on a perforated gastric ulcer, but suppuration may also begin in or about the spleen.

In the right hypochondrium, beside traumatic cases and those which arise from caries of the ribs, a subphrenic abscess may be caused by suppurating hydatids, a perforated hepatic abscess, and particularly by the form of tropical abscess described by Dr Cantley as formed between the liver and the peritoneum, or by suppuration which has begun in the bile-passages and burrowed round to the posterior margin of the liver. In several cases it has been traced to a perforated appendix, which has caused pus to burrow upwards; in others it may be due to pyelitis from calculus or some other cause affecting the right kidney, and in one of Mr Mayo Robson's cases it was caused by a suppurating ovary.

The pus sometimes finds its way upwards through the diaphragm, causing a secondary true empyema or pyopneumothorax, or possibly opening into the pericardium, or along the aorta into the posterior mediastinum.

The symptoms of a subphrenic abscess are those of pus at the base of the chest mingled with gas if its origin is in perforation of the stomach.

* Perhaps the earliest case on record of circumscribed abscess of the hypochondrium containing pus and gas was that published by Dr G. H. Barlow in the 'Medical Gazette' for 1845, the report being furnished by Samuel Wilks, then a student of Guy's Hospital.

The difficulty therefore is to distinguish it from empyema or (on the left side) from pyopneumothorax. The physical signs are the same. The absence of preceding pleurisy or pneumonia or phthisis, and the presence of preceding symptoms of a gastric ulcer, of typhlitis, or of abdominal suppuration, are probable grounds for a diagnosis. Dr Lee Dickinson mentions the important facts in diagnosis of hypophrenic abscess from empyema, that the liver moves down with inspiration in the former condition, and that after the collection of pus has been punctured, the flow will be accelerated during inspiration in the former, and during expiration in empyema.

The treatment when the existence of the pus is suspected can only be surgical, by probing with a subcutaneous syringe; and when it is found, by incision and drainage.*

A woman of sixty-four was admitted into Miriam Ward under the writer's care, December 21st, 1897. There was abdominal pain and obstinate constipation, but the rectum contained fæces. The pain was chiefly in the left iliac and hypochondriac regions, with dulness in the latter part, and she was thought to have subacute peritonitis and consequent inaction of the bowels. She improved apparently; but then a rub, with dulness at the base of the left pleura, developed, and this was followed by similar signs on the right side, with evidence of acute hepatisation as well as pleurisy.

After death (Jan. 3rd, 1898) there was found a perforated gastric ulcer in the posterior wall of the stomach and suppuration around the spleen; recent perforation of the diaphragm and acute pleurisy on the left side. What was most remarkable was the presence of a secondary abscess on the right side, under the diaphragm, which was not perforated, and recent acute septic pleurisy with pneumonia and gangrene of the right lung.

Diagnosis of peritonitis.—The first question is to decide when a person, previously supposed to be well, is suddenly seized with pain in the abdomen. whether the attack is acute peritonitis, or irritant poisoning, or colic, or hysteria, and this is sometimes far from easy.

The chief distinction is found in manipulation of the abdomen. In *colic* pressure and friction give relief; the parietes are hard and contracted; the pain intermits from time to time, so that the patient has intervals of ease; and when his sufferings are at their worst he is restless, and tosses about in search of relief. In *hysteria*, on the other hand, there often appears to be the most extreme tenderness of the surface; but if the patient's attention be diverted no further complaints are made, and after a time considerable pressure is perfectly well borne, while the abdominal walls become soft and supple. The exaggerated sensitiveness to the lightest touch is in such cases the very symptom that shows the absence of serious disease. One must not, however, forget that acute peritonitis from perforating ulcer of the stomach occurs in anæmic young women who are very likely to have had hysterical symptoms. It is possible that the thermometer may sometimes show a rise of temperature in cases of mere hysteria; and, on the other hand, pyrexia may be absent in peritonitis.

The probability that the pain is due to colic is of course greater if the patient has lately eaten indigestible food, or if the gums present the dotted lead-line.

In all doubtful cases one must remember that to attribute to peritonitis a pain really due to colic or hysteria is an error free from serious consequences, whereas the converse mistake may be fatal to the patient; and a few hours' delay will always solve the question.

* Since the last edition of this text-book in 1891 numerous papers have appeared on the subject, of which Dr Clifford Allbutt's in the 'Clinical Journal' for June 7th, 1893, and Dr Lee Dickinson's in the third volume of Allbutt's 'System of Medicine,' may be particularly mentioned. The writer gave an account of it in the second edition of the present work (1888).

The rupture of a concealed aneurysm into the subperitoneal tissues is another possible cause of sudden severe pain that must not be overlooked, particularly if the patient fainted when the attack began, or was pulseless from the first. It may at first be impossible to distinguish this from perforation of the stomach or intestine, or from hæmorrhage in the broad ligament.

The characteristic symptoms of acute peritonitis when fully developed, as described above (pp. 478, 479), might seem to leave no room for doubt. But unhappily they are not so unmistakable as was once thought. In the first place they may be present from severe abdominal pain due to impaction of a gall-stone or a renal calculus, or to a twisted Fallopian tube or epididymis, apart from the peritonitis which may come on afterwards or not. And secondly, they may be absent in some of the most severe cases of suppurating peritonitis.

When it is clear that peritonitis is present, the diagnosis of its *cause* remains. Among the numerous affections that may produce peritonitis, few are likely to be latent. Hence, when a person supposed to be healthy is attacked, the range of probable causes is not extensive. Perforating ulcer of the stomach or duodenum, perforating typhoid ulcer of the ileum, perforation of the gall-bladder or of the cæcal appendix, and pelvic disease in the case of a woman, are the chief.

A perforating ulcer of the stomach or intestine is commonly fatal in a few hours, or in a day or two at the latest. Hence when peritonitis runs a longer course than this in a young male patient (or even in a woman, if the ovaries, Fallopian tubes, and uterus are healthy) there is a strong presumption that it started from the cæcal appendix.

There is also a negative side of the difficulty in the diagnosis of peritonitis; acute suppurative peritonitis, starting from sloughing or perforation of the vermiform appendix, may run its course without characteristic symptoms. In enteric fever, again, perforation of the intestine may have taken place some hours or days before death, without increased pain or tenderness, or any marked aggravation of the symptoms. The cause of this is not only that patients suffering from fever have their senses and intelligence stupefied, for peritonitis may remain latent in those whose minds are clear to the last. In making a *post-mortem* examination after an operation for hernia, ovariectomy, or the like, we have repeatedly found universal peritonitis when those who had watched the patient most closely had detected no evidence of it during life.

When symptoms of intestinal obstruction have been present for a few days, one can never assert positively that peritonitis has not already set in.

Again, in the majority of cases of acute peritonitis in Bright's disease, its presence is first discovered in the deadhouse, the patient having at most complained of slight pain in the abdomen.

In latent forms of peritonitis the effused fluid is generally pure pus, and the pain of peritonitis may subside when free suppuration takes place.

The most trustworthy guides to a prognosis in peritonitis are probably the aspect of the patient, the amount of distension of the abdomen, and the frequency of the pulse.

Treatment in acute peritonitis depends upon its cause. It was formerly held that nothing is to be gained by active interference, and the administration of opium was the only treatment in vogue.

The patient was kept in bed from the moment that peritonitis was

suspected, and the recumbent position scrupulously maintained. A pillow was placed beneath the knees to support the thighs in a flexed position. No purgatives of any kind were allowed to be given, even though the bowels had been closed for many days; in most cases not even an enema. The result was, however, almost invariably fatal.

Occasionally success followed this treatment, as is shown by the following case recorded by Hughes in the 'Guy's Hospital Reports' (2nd series, vol. iv, p. 332).

A young woman became collapsed, and was seized with severe pain in the stomach. The last food which she had taken was a little gruel, four hours before; for some days previously she had eaten almost nothing. She sent for the late Mr Ray, of Dulwich, who (instead of giving her brandy and castor-oil) administered twenty minims of tincture of opium in a little water. She rallied somewhat, and was carefully removed to the hospital. She was there ordered half a grain of opium in a pill every three hours, and to have nothing whatever to drink except two measured teaspoonfuls of toast and water. After two days she complained much of thirst. An enema of five ounces of strong, tepid beef-tea was therefore administered, with five minims of laudanum. This was afterwards repeated three times a day. She was also allowed to suck one teaspoonful of beef-tea jelly, instead of the toast and water. It was not until the ninth day that she was permitted to have two table-spoonfuls of strong mutton broth. She completely recovered, and was discharged from the hospital. Nearly four months afterwards, having been so foolish as to indulge freely in cherries and gooseberries, she was attacked with the same symptoms as before. She had brandy and water given to her, and died in nineteen hours. An ulcer in the stomach was found, which had become torn away from a thick layer of old lymph by which it had before been closed. In its neighbourhood there were old vascular adhesions. It seems almost certain that in this case perforation of the stomach occurred during the first attack as well as the second, and that she would have died on the former occasion under less skilful treatment.

The systematic treatment of peritonitis by opium was introduced by Graves, of Dublin, who, in 1822, ordered it in large doses, to relieve the agony experienced by a woman in whom inflammation had set in after the operation for tapping. Her case seemed hopeless; but to his great astonishment she recovered.

Two grains of opium may be given at first, and afterwards one grain every three hours, the action of the drug being carefully watched. There is great tolerance of this remedy in cases of peritonitis. A lad, who had probably never swallowed a dose of opium before, once took as much as twelve grains daily, without being made sleepy or having a furred tongue, and without his pupils being in any way affected by the drug. In another case the bowels began to act regularly every day, while the patient was still taking a grain of opium every two hours throughout the day and night.

In many cases the pain is much relieved by a few leeches applied to the most painful part of the abdomen. Warm fomentations or large poultices should be used constantly, and changed as often as they cool. When there is much meteorismus, relief is often afforded by a flannel, wrung out of boiling water, and sprinkled with turpentine. A long tube introduced into the rectum, and cautiously pushed upwards, has been known to give vent to a large quantity of gas from the colon; but it more often fails.

If tympanitic distension is excessive, it may be justifiable to puncture the intestine with a very small trocar through the parietes, but this is attended with more risk than in cases of early mechanical obstruction, because the coats of the bowel when inflamed lose their elasticity.

When the distended and immoveable abdomen, the small and rapid pulse, and the other symptoms above enumerated show that general and acute peritonitis is already present, the ill-success of all treatment, even

that by full doses of opium, to do more than procure an easy death, has led to another line of practice; and the experience of surgeons in performing ovariectomy and other operations involving the peritoneum has encouraged them to open the abdomen, wash out the products of inflammation, and put in a drainage-tube. This plan of treatment is now adopted as the rule, sometimes with only temporary relief, but sometimes undoubtedly saving an otherwise forfeited life.* In the group of cases of peritonitis from perforation, this treatment, combined with suture of a gastric ulcer, resection of a sloughing intestine, or removal of a gangrenous appendix, affords the only chance of success.

When we have reason to believe that the peritonitis is septic and purulent, but not due to perforation, it is always good practice to open the abdomen, wash out the cavity, and drain it.

The intensely dangerous character of acute peritonitis is manifest. Often death is inevitable, but even when peritonitis follows perforation of an ulcer in a stomach containing food, or when perforation occurs in enteric fever, instances of recovery are on record (vol. i, p. 147). That form of peritonitis which is set up by ulceration of the cæcal appendix is, when properly treated, far less dangerous, if only its nature is correctly diagnosed, and if it is treated early (p. 406).

CHRONIC IRRITATIVE PERITONITIS.—This is very different from the acute septic disease we have been studying in its causes, pathology, and results, and the one seldom or never ends in the other.

Anatomy.—The whole surface of the peritoneum is found after death to be thickened and opaque. Adhesions often have formed between different parts so that the liver, spleen, and stomach may be united into a single mass by firm connective tissue, and may be closely adherent to the diaphragm and abdominal parietes. The omentum is frequently drawn up, and its folds inextricably blended together; so that, with the fat which it contains, it forms a solid mass, binding the colon to the stomach, and capable of simulating a tumour during life. The intestines may be fixed to the front wall of the abdomen; indeed the entire peritoneal cavity may be obliterated by adhesions. More commonly, however, the small intestines are only adherent among themselves, and are collected in a more or less rounded mass in front of the spine. Sometimes the membrane which unites the several coils can be stripped off, leaving the intestines still covered with a serous coat. This formation of "false membranes," looking like thickening of the peritoneal covering of the viscera, but really laid over them, may be seen in all parts of the abdomen. Such a "reduplication" of the capsule of the liver is one of the most remarkable features of the affection known as perihepatitis; which, besides occurring independently, forms part of many cases of chronic peritonitis.

The false membranes may form adhesions among themselves, dividing the general cavity into separate chambers, each containing fluid.

A woman, aged forty-four, was sent to Guy's Hospital, supposed to be suffering from cystic disease of the ovaries. The physician under whose care she came doubted this, and

* Among the early literature of abdominal section for peritonitis, see the paper by Sir Thos. Smith in the 'St Bart.'s Hosp. Reports' for 1873 (vol. ix), one by Sir Jos. Lister in the 'Lancet' for 1881 (vol. ii, p. 863), and cases brought before the Royal Medical and Chirurgical Society in 1885, by Mr Howard Marsh and Mr Treves, and before the Clinical Society in 1887, by Dr Knaggs and Dr K. Clarke, of Huddersfield.

thought that there was fluid in the peritoneal cavity. After some weeks she died. At the autopsy it appeared at first as though the original diagnosis had been correct. Nothing could be seen but a mass of cysts covering the intestines, the stomach, and the liver. Presently, however, it was seen that these cysts had been formed, not in the ovary, but in the peritoneal cavity. Several of them lay between coils of intestine, and some contained a fluid of milky appearance, from the admixture of chyle.

Dr Fagge met with a similar case. The abdomen contained a considerable quantity of fluid; and this would have been regarded as passive ascites (caused by the heart disease for which the patient was admitted), but that the physical signs were in some respects anomalous. After death the peritoneal sac was found to be divided into distinct chambers by adhesions; one of them was above the transverse colon, another occupied the middle of the abdomen, and a third filled the right loin.

In the great majority of cases of chronic peritonitis, however, the small intestines are neither compressed nor adherent. Their coils are still capable of moving on one another, and their mesentery is fan-shaped. But the mesentery is remarkably shortened; it may measure only two inches from the spine to the attached edge of the bowel, which is thus closely tethered to the back of the abdomen, instead of floating freely. Moreover, the length of the bowel itself is greatly diminished. It may not be more than a few feet long from the duodenum to the cæcum; so that the mucous membrane of the ileum is thrown into folds, resembling the valvulæ conniventes of the jejunum. Its diameter is no less contracted, so that it may hardly admit the little finger. The muscular coat of the bowel is generally thin, but that of the stomach is sometimes much thickened, so that it resembles an india-rubber bottle (*cf.* p. 365).

In most cases of chronic peritonitis a straw-coloured fluid occupies the abdominal cavity. It is usually more or less opalescent, contrasting with the translucent serum of passive ascites; and sometimes, instead of being pale, it is darkened by the presence of blood. It may contain flakes of lymph or even pus; but the latter, when present, is generally the product of an acute inflammation, supervening upon the chronic peritonitis, and too often as the result of paracentesis. The surface of the peritoneum, besides being thickened, is opaque, and it sometimes is not white, but slate-coloured or even blackened from effused blood.

Age.—Such chronic peritonitis is far from being a rare disease. Dr Fagge estimated from *post-mortem* experience on an average one case of this kind to two of ascites from cirrhosis of the liver. Of his thirty-four cases, eighteen occurred in males, sixteen in females. Between twenty and thirty there were almost as many cases as between thirty and forty, or between forty or fifty; several patients were more than sixty years old, and one had passed the age of seventy.

Origin.—Sometimes we can trace chronic peritonitis to what may be called a subacute attack. The peritoneum fills with serum, with little or no pain or febrile symptoms. In these cases it may be removed by diuretics or by tapping, and not return again, although more often it goes on to the ordinary chronic ascites with thickened peritoneum. In other cases the process is insidious from the first.

In a remarkable case we had several years ago in Guy's Hospital, the writer saw the origin of the disease in a healthy country lad of fifteen, and its slow increase, until at last it proved fatal, after nearly two years. Here there was a similar chronic effusion into both pleuræ and into the pericardium, with enormous thickening of all the serous membranes, including the tunica vaginalis, with which an open inguinal canal communicated. There was no trace of tubercle found *post mortem*, and the viscera, includ-

ing the kidneys, were perfectly healthy. It was an example of a concomitant affection of the peritoneum, pleura, and pericardium, comparable to, but distinct from, those of tuberculosis of the serous membranes which will presently be described.

Chronic peritonitis is sometimes traceable to lesions of the subjacent viscera; in one case it was believed to have started from the cæcum; and in two from old pelvic cellulitis, which itself in one of them arose from morbus coxæ. Most frequently, perhaps, perihepatitis is its origin. Like that affection, it commonly occurs in patients who have Bright's disease, which may perhaps be regarded as its principal cause. Many patients affected with chronic peritonitis have been intemperate, and some have suffered from disease of the heart.

The principal *symptom* of chronic peritonitis is the presence of fluid in the abdominal cavity. This can most conveniently be discussed further on, together with its diagnosis, prognosis, and treatment, under the head of ascites (*infra*, p. 493).

TUBERCULOUS PERITONITIS.—This is a common subacute or chronic disease, to which children are particularly liable. It is always secondary to tubercle elsewhere.

Anatomy.—The peritoneum is covered with minute grains, which, however, are seldom uniformly distributed over its surface, but are more numerous in some parts than others, especially on the under surface of the diaphragm and in the flanks. The serous surface of the intestines is sometimes comparatively free. The omentum often contains a large quantity of yellow and caseous, or of recent grey tubercle; and it is drawn up into a flattened mass, two or more inches thick, lying below the stomach and across the colon. The abdomen is often found to contain turbid serum or pus; it is closed by adhesions, or with merely a few scattered collections of liquid here and there between the viscera.

Sometimes the intestines are firmly matted together, and tuberculous ulcers perforate, so as to form communications at several points. In one case of peritonitis in a child under the writer's care in 1890 an abscess appeared, which was opened and formed an intestino-cutaneous fistula.

Tuberculous affections of other parts are associated with tuberculous peritonitis. In women, the Fallopian tubes are almost always affected; they are much enlarged, lined with a thick caseous layer, and very often contain pus. Sometimes the same condition is present also in the cavity of the uterus. Moxon believed that the disease spreads into the open mouths of the tubes from the serous surface, since the tuberculous change is often limited to the ends furthest from the uterus. In men the epididymis or testis (on one side or both) is sometimes the source of tuberculosis; and when this can be made out during life, it affords great help in diagnosis.

Other serous membranes often become affected in the same way as the peritoneum. Thus one or both of the pleural cavities may contain a considerable quantity of fluid, or they may be covered with tubercles and closed by adhesions; and less frequently we find tuberculous pericarditis—sometimes with effusion of much turbid serum or pus.

In most cases the lungs and spleen contain tubercles, and frequently the liver or kidneys or the pia mater.

The intestines often show tuberculous ulcers, and the mesenteric glands

are usually enlarged and caseous. In 127 cases of abdominal tuberculosis in children, at Edinburgh, Dr Woodhead, in 1888, found the lymph-glands affected in 100, but in only 14 affected alone. The old term *Tabes mesenterica* may be now replaced by that of Tuberculosis abdominalis, including the probably primary enteritis, the secondary mesenteric, and the tertiary peritoneal infection.

Symptoms.—Tuberculous peritonitis is sometimes acute, but never so rapid or severe as that which follows perforation or septic poisoning; and it is sometimes chronic, but seldom so free from symptoms and insidious in its course as the “simple” chronic peritonitis above described. Subacute is the adjective which best qualifies its symptoms and its progress. The first signs are often vague and obscure. The patient feels out of health and loses flesh, and he complains of pains in different parts of the abdomen. There may be diarrhoea, and the abdomen may be tender and harder than natural. Occasionally it is rather retracted than enlarged, but, as a rule, it is swollen, with marked fluctuation, and other indications of the presence of fluid in considerable quantity. Clinically, ascites is more often detected than might be supposed from experience in the deadhouse, for at an early stage of the disease the peritoneum frequently contains fluid, which is absorbed in its further progress.

A well-marked case in a child is recognised at once. The swollen abdomen, more or less resonant but with patches of dulness, and sometimes with lumps to be felt, the diminutive thorax covered only with skin, the wasted limbs, pinched features, and fretful cry, make up a characteristic picture.

Age and sex.—Tubercle of the peritoneum is one of the common diseases of childhood; and when associated, as it often is, with tuberculous ulceration of the bowels and secondary tubercle of the mesenteric lymph-glands it assumes the familiar clinical aspect of *tabes mesenterica*. But it would be a mistake to suppose that tuberculous peritonitis is only a disease of early life. In twenty-eight successive fatal cases at Guy’s Hospital, two patients were under ten years of age, six between ten and twenty, eight between twenty and thirty, five between thirty and forty, three between forty and fifty, and four over fifty. The disease is more than twice as common in men as in women. Of the twenty-eight cases only eight were in females; and in all of these, with one exception in a child, there was co-existent disease of the Fallopian tubes.

Diagnosis.—In the account just given of the symptoms of tuberculous peritonitis there is little to distinguish it from other forms of chronic and subacute abdominal disease. Great assistance, therefore, is often afforded by the induration of the omentum, which may be felt as a rounded tumour running more or less obliquely across the abdomen above the umbilicus. It has been mistaken for the edge of the liver, depressed and rounded by thickening of its capsule; but a resonant percussion-note can be elicited *above* the mass, where, if it were an hepatic tumour, there must have been absolute dulness.

Another useful sign is thickening round the umbilicus. This may sometimes point to adhesion of the small intestine to the abdominal wall at this spot, for in two cases a fæcal fistula resulted. More often it is caused by spread of the inflammation of the parietal peritoneum along the track of the obliterated umbilical vessels and the urachus, as in cancerous disease. In some of those rare cases of tuberculous peritonitis in

which the abdomen becomes distended with pus, the umbilicus gives way, and allows the fluid to escape.

The diagnosis of tuberculous peritonitis may be confirmed by the discovery of coincident effusion into one of the pleural cavities, or into the pericardium.

Lastly, whenever we suspect tuberculous peritonitis in a woman, we must not forget how constantly this disease is associated with tuberculous disease of the pelvic organs. One patient suffered from amenorrhœa for eighteen months, another from menorrhagia, a third from a miscarriage a month before her abdomen began to enlarge; while a fourth, after one period which lasted a fortnight, and was excessive, missed her next period, and from that date her abdomen began to swell and her fatal illness commenced.

Prognosis.—The clinical recognition of tuberculous peritonitis is the more important, because the disease is by no means so hopeless as was formerly supposed. The writer has seen several instances in which, probably, recovery took place, and in one case the diagnosis was afterwards proved to be correct by a *post-mortem* examination; for the patient, a child, who had left the hospital apparently well, came in again some months afterwards, and died with tubercles in almost all parts of his body; it was then clear that the peritoneum had been the seat of the same disease before.

A remarkable instance of recovery from tuberculous peritonitis occurred to the late Sir Spencer Wells. The patient, aged twenty-two, was believed to have an ovarian tumour, and had twice been tapped. It was decided that ovariectomy should be performed. But, on the abdomen being opened, the peritoneum was found studded with tubercles. Some coils of small intestine were floating, but the great mass was bound down with the colon and omentum, all nodulated with tubercles, towards the back and upper part of the abdomen. The fluid was syringed out and the wound closed. The patient went through a sharp attack of peritonitis, but recovered, and she afterwards married; six years later she was stout and well.

Treatment.—It appears probable that in children tuberculous peritonitis may be cured by the local application of linimentum hydrargyri. This practice has long been carried out in our wards, the liniment being spread freely over the surface of a flannel belt, which is stitched tightly round the abdomen. We have more than once seen the greater part of the fluid removed within a few days under this treatment, and the patient has also improved in health and gained strength. After recovery there was naturally no direct proof of the tuberculous nature of the affection; but there is evidence that in children all tuberculous affections tend less uniformly to a fatal termination than in adults. It is no doubt advisable to give cod-liver oil, syrupus ferri iodidi, and the like; but in several cases these have failed, and the mercurial application has proved successful.

In obstinate cases there is good ground to hope for success, not by mere tapping—which is only useful to relieve tension or for diagnostic purposes—but by opening the abdomen, washing out the peritoneal cavity, and putting in a glass tube or flexible drainage-pipe. The result has been favourable beyond the writer's expectation. In one case recovery ensued even after a tuberculous ulcer in the ileum had opened externally through the wound.*

* Among many other cases recorded at home and abroad, three published by Mr

MALIGNANT PERITONITIS.—This—a third chronic disease to which the peritoneum is liable—is both common and fatal.

Anatomy.—Numerous round or flattened nodules are found studded over the peritoneal surface, sometimes isolated, sometimes aggregated together. Often each little tumour is distinctly umbilicated, and it may send out processes which are apt to pucker and drag the neighbouring parts of the serous membrane. In this way the calibre of one or more of the hollow viscera may be considerably diminished. By a somewhat similar process of contraction, the omentum is drawn up and converted into a solid mass, which lies transversely across the abdomen, below the stomach, just as was described above in the case of simple and of tuberculous peritonitis (pp. 487, 490). This induration of the omentum is present in most cases of malignant peritonitis, although in other respects the distribution of the nodules may vary widely. Sometimes the mesentery is covered with them; in other cases it is comparatively free. Often, as Moxon pointed out, the growth is far more abundant on the peritoneum lining the flanks and the diaphragm than elsewhere.

Histology.—The microscopical structure of the malignant nodules is not always the same. Virchow included a considerable number among the sarcomata, and Dr Fagge found that they generally yield but little juice when cut, and “often consist of fibres with a few spindle-cells,” without alveolar structure or epithelial elements.

In the revision of the Museum specimens by Drs Perry and Shaw for their new catalogue, they have found the most frequent elements of peritoneal cancer to be columnar or spheroidal cells. Sarcomatous nodules are usually secondary to ovarian growths, and alveolar epithelial nodules to those of the stomach and intestine. Colloid cancer of the peritoneum is not very uncommon, and usually secondary to growths of the stomach or of the broad ligament.

Origin and course.—Malignant disease of the peritoneum is seldom, if ever, a primary affection. As a rule, one of the subjacent viscera is the seat of a similar growth; and to this the peritoneal nodules are secondary. The most frequent is, as Virchow stated, in the stomach and ovaries.

Out of forty-five consecutive cases of extensive malignant disease of the peritoneum, Dr Fagge found only six with the abdominal viscera free. In nineteen the *ovaries* were affected, and were often converted into large tumours by the growth. In seventeen the *stomach* was diseased in the same way; seven times without, and ten times with malignant disease of the ovaries also. In three cases the peritoneal cancer started from the *uterus*, in two from the *rectum*, in three from the neighbourhood of the *pancreas*. In two there was a hard mass in front of the rectum; and in several cases (including some of those in which the disease seemed to have begun in the ovaries) the uterus and its appendages were matted together and fixed to the adjacent parts by a large diffused growth in the sub-peritoneal tissue.

In one instance the affection of the peritoneum seemed to have started from a cancerous growth in the ascending *colon*. The omentum formed a solid mass an inch thick, which was spread over the intestines, and reached down to the pubes.

The way in which malignant disease spreads from the stomach or Keetley in the ‘Lancet’ (Nov. 15th, 1890) may be referred to for the sake of the judicious remarks at the close of the paper.

ovaries over the whole peritoneal surface is a matter of much interest. When the growth reaches the serous surface of an organ, it is well known to be capable of infecting the surface opposed to it without the formation of adhesions between them. Dr Fagge recorded a good example of this. The body of the uterus was affected with cancer, which reached its outer surface. The omentum was long and hung down into the pelvis, so as to touch the uterus, and in its extreme lower end there was a hard mass, exactly resembling the uterine cancer in its character. There was no malignant growth in any other part of the peritoneum. It is probable that such local infection of the omentum is not uncommon, and forms the starting-point of the remarkable change in this structure already mentioned. The infection of the general surface of the serous membrane probably arises in the course of the movements of the contained organs. It is even possible, to use the words of Rindfleisch, that "the mutual friction of the viscera may detach fragments of the nodules, and carry them hither and thither over the smooth surface of the membrane, until they find their way into some fold or recess, when they give rise to the development of fresh nodules."

Age and sex.—Unlike tuberculous peritonitis, cancer of the peritoneum appears to occur more frequently in women than in men. Out of the forty-five fatal cases referred to above, only eleven occurred in males.

Under the age of thirty this disease is exceedingly rare. Between thirty and forty it is not very uncommon in women, but is seldom seen in men. In each sex the most numerous cases occur between the ages of fifty and sixty; it is also common between sixty and seventy, and Dr Fagge records a case in a man who died at the age of eighty-two.

Diagnosis.—Clinically, malignant disease of the peritoneum presents itself in different cases with very different symptoms.

The growth may, by the adhesions and puckerings which it causes, so narrow the intestine as to interfere with the passage of its contents, and to give rise to well-marked obstruction by contraction (*cf.* p. 414).

Most frequently the only marked symptom of the disease is ascites. The serum is usually stained more or less deeply with blood. Another character, first pointed out by Sir William Jenner, is the occasional presence of a hard mass in the skin and other tissues round the umbilicus. Probably the growth travels along the connective tissue in the path of the obliterated urachus or umbilical arteries or vein.

Occasionally the first symptom of malignant disease of the peritoneum is an increase in size of the abdomen, without any fluid being present. In these cases the growth is a true carcinoma, which has undergone colloid degeneration.* All the organs may be enveloped in thick gelatinous masses, many of which are attached only by the most delicate threads.

ASCITES.†—Apart from chronic inflammatory exudation (whether with or

* Many years ago, when I was a senior student at the hospital, a medical man in the country asked me, during the vacation, to look at a case in which he was about to tap for ascites. I found that although there was very great enlargement and dulness on percussion over the whole abdomen, yet no fluctuation could anywhere be discovered. I remembered hearing Dr Wilks describe colloid cancer of the peritoneum, and ventured to suggest that the case was of this kind, and that paracentesis would lead to no result. During a subsequent vacation I made the autopsy, and found that I had been right.—C. H. F.

† Ascites (*ἀσκήτης*), from *ἀσκός*, a wine-sack, was recognised by the Greek physicians, and distinguished from *lympanites* (*τύμπανον*, a drum), the false or "windy dropsy."

without tubercle or cancer) the peritoneum is also liable to passive dropsical effusion, which is known as ascites. This is sometimes accidentally discovered by the physician, or the patient finds it out for himself, by a fulness and sense of weight. But the same sensations may result from flatus in the bowels, or from obesity, or from the presence of a tumour; so that in every case of abdominal swelling, we must ask ourselves to which of these causes it is due.

The most common is deposit of fat in the subcutaneous fascia, the sub-peritoneal tissue, and the omentum. It is recognised by the thickness of the walls, the modified resonance over the entire abdomen, a deeply depressed umbilicus, and transverse horizontal folds in the skin. Such obesity is usually combined with excess of adipose tissue elsewhere—not unfrequently with flatulent distension, and only occasionally with ascites.

Tympanites is almost as common. The resonance is long, loud, sometimes high-pitched, and always with more or less pure tone.

Ascites is distinguished by fluctuation, and by a peculiar disposition of dulness and resonance, to be presently described.

Abdominal tumours do not fill the whole abdomen; and they have the special characters which point to the stomach, liver, spleen, or kidneys. Two kinds of tumour should always be thought of in cases of abdominal swelling occurring in woman: ovarian dropsy, and a pregnant uterus. The differential diagnosis will be given below, but the possibility of the latter should always be present to the mind of the physician.

Physical signs.—Palpation and percussion are both useful in revealing the presence of fluid in the peritoneal cavity.

Palpation may be employed in two ways. If a solid organ or tumour lies at a little distance from the anterior wall of the abdomen, and separated from it by fluid, one can often, by a sudden movement of the fingers, depress the abdominal wall, and push aside the fluid, so as to feel the solid mass beneath, as we could not if no fluid were present. Thus one may not only detect an enlarged liver, but also at the same time determine the presence of ascites. This procedure is sometimes spoken of as “dipping for the liver:” it requires a little dexterity, and should be carefully practised by the student.

The other method of discovering by palpation whether there is fluid in the peritoneal cavity is by observing whether *fluctuation* can be felt. This is not the peculiar elastic sensation felt in handling an abscess. “Medical fluctuation” is detected by an impulse given to the wall of the abdomen at one spot, and transmitted freely in the form of a wave. If, for example, the left hand be placed on one side of the patient’s abdomen, and a tap be then given to the other side with the right, the left hand receives a distinct shock. When the parietes are thin, and other conditions favourable, the slightest touch may cause a thrill that can be felt all over the belly. There is perhaps no other physical sign which the tyro recognises so easily as this. If, however, the parietes are massive, with œdema or fat, or the muscular walls are hard, or loaded with fat, the detection of fluctuation may be difficult. The two hands must then be placed near one another: and a smart tap must be given with one hand, while attention is closely directed to the reception of the impulse by the other. Sometimes the fat in the abdominal walls gives a sensation that might be supposed to be due to fluctuation. To avoid this error an assistant should hold a thick piece of

cardboard (a bed-letter in a ward, a thin volume in a house), with its edge pressed upon the median line of the abdomen while percussion is made.

In some few cases we may fail to obtain fluctuation, although a large quantity of fluid is present; probably the walls of the abdomen are too unyielding for a wave to be transmitted.

It is remarkable what small quantities of fluid can often be detected in the way just described. One might have expected that unless it were present in large amount it would all have gravitated into the loins or into the pelvis, according to the position of the patient; but in fact distinct fluctuation can frequently be felt over parts of the abdomen where the intestines lie in contact with the parietes.

Percussion is also of service in detecting the presence of ascites, and still more in distinguishing this from some other conditions which resemble it in causing abdominal enlargement. Whenever the amount of fluid is at all considerable, that part of the abdomen which contains it gives a dull note on percussion. But a small quantity, lying among the intestines in the way just described, may fail to affect the natural tympanitic note, and this although it gives distinct fluctuation.

Diagnosis.—For the determination of ascites, however, something more is required than the mere discovery of dulness on percussion, or even of fluctuation. The former might be caused by a solid tumour, and the latter might depend upon a collection of fluid within one of the hollow viscera, or in an adventitious cyst. Cystic disease of the ovary is by far the most important of all the affections that may be confounded with ascites. But there are several other conditions that have been mistaken for it. It is recorded that John Hunter once tapped the bladder in the belief that the patient had dropsy; and Murchison relates a case of ascites in which 480 ounces of urine were drawn off by a trocar introduced midway between the umbilicus and sternum, on the supposition that there was a hydatid tumour. A large and elastic tumour may yield physical signs more or less like those of ascites; and so may a renal cyst, or even a pregnant uterus.

In the great majority of cases one can readily distinguish an accumulation of fluid in the peritoneal cavity from all these conditions by noticing which parts of the abdomen are dull and which are resonant on percussion, when the patient assumes different positions in turn. In ascites the fluid tends to sink towards the more dependent part of the peritoneal cavity; while the intestines may be said (as Aretæus put it*) to float in it. Hence, when the patient lies upon the back, the small intestines fill the umbilical region, and there the percussion-note is tympanitic, whereas in the flanks it is dull. But if the patient is made to turn upon one side, the position of the intestines at once becomes altered; whichever side is uppermost is now resonant, while the dulness on the other side undergoes a corresponding increase. When the patient stands upright, the fluid gravitates towards the lower part of the abdomen, which, up to a certain level, becomes uniformly dull. Again, when in ascites the border of the dull region is percussed firmly, the left-hand finger being pressed backwards as much as possible, one can often detect a resonant note from the presence of intestine beneath.

All these characters are wanting when enlargement of the abdomen is

* "Ἡν δὲ ὕδωρ ἄλις ἐς τὸ περιτόνσιον ἐμπεριεχῆται, ἐμπλώη δὲ τῷ ὑγρῷ τὰ ἔντερα, καλέομεν ἐπικλυσιν ἀσκήτην. Aretæus: *De Morbis Chron.*, lib. II, cap. i.

due to cystic disease of the ovary, or to pregnancy, or to distension of the bladder. Moreover, each of these swellings rises from the pelvis into the front of the abdomen, pushing the intestines backwards; so that when the patient lies upon the back, the front of the abdomen yields a dull note on percussion.

Another sign of some value is prominence of the umbilicus, or occasionally the presence of a protrusion there, containing fluid. This contrasts with the deep depression of the navel in cases of obesity. Moreover, in the latter case two transverse lines can generally be traced—above the pubes and above the umbilicus.

It must be admitted that in some exceptional cases of ascites, the whole of the front of the abdomen is dull, in whatever position the patient lies. When the quantity of fluid is very large, the intestines may stretch the mesentery to its full extent, and yet, perhaps, be unable to reach the anterior abdominal wall. But, as a rule at least, it will be found in these cases that the mesentery has been shortened by chronic inflammation, or by cancer, so that it tethers the bowels and prevents their floating up.

It will be convenient to consider here the special indications which distinguish pregnancy and an ovarian cyst from ascites.

Pregnancy is distinguished by the shape of the tumour, its dulness on percussion, its gradual increase from below, and occasionally by feeling the rhythmical contractions of the uterus. The state of the breasts and the absence of menstruation confirm the diagnosis; and also the condition of the cervix, the movements of a living foetus, and auscultation of the beatings of its heart or the rush of blood in the placenta.

The positive signs of an *ovarian cyst* are often no less conclusive. The patient may be able to say that the swelling began on one side. A careful examination of the swelling will often lead to the detection of a solid nodule; or the outline of the cyst may be felt; or, when the patient draws a deep breath, the upper border of the tumour may be seen to descend. In ascites the greatest circumference of the abdomen is at the level of the umbilicus, in ovarian disease it is often some inches lower. In ascites the umbilicus usually retains its natural position, being about an inch nearer to the pubes than to the ensiform cartilage, while in ovarian disease its distance from the pubes is increased. To these signs may be added the fact that in cases of ascites the fluid expands the lower ribs, and so produces a uniform barrel-shape; while an ovarian cyst grows more forwards and laterally, and so gives an hour-glass shape to the abdomen when looked at in front, and a bulging like that of pregnancy when looked at sidewise.

In obscure cases an ovarian cyst is often distinguished from ascites by the character of the fluid drawn off by paracentesis. That which comes from the ovarian cyst is frequently viscid and of a dark greenish-brown colour, quite unlike the secretion of a serous membrane.* Ovarian fluid, however, may be pale yellow, and clear, and limpid, like the fluid of ascites; but it never deposits fibrin, as ascitic fluid almost always does.

The fluid drawn from the single cysts which form in the *parovarium*† from the remains of the Wolffian body is characteristic,—clear, transparent,

* Its viscosity is said to depend upon its containing a modification of albumen (par-albumen of Scherer) which does not coagulate when boiled with a small quantity of acetic acid. Paralbumen is said never to be present in ascitic fluid.

† Known as the organ of Rosenmüller, and answering to the epididymis in the male.

and consisting of nothing but water and salts, unaltered by heat. The only liquid like it is the contents of a hydatid cyst, but that contains hooklets.

In cases of ascites it is important to decide whether the effusion is *passive*, and due to portal obstruction or to general dropsy, or whether it is *active*, the result of inflammation of the peritoneum. Chronic peritonitis with effusion includes at least a third of all the cases of ascites which occur independently of heart disease or Bright's disease and are unattended with jaundice.

So long as the quantity of fluid in the abdomen is not very large, one can generally without much difficulty distinguish ascites caused by obstruction of the portal veins from active effusion. In the former case the area of dulness in the right hypochondrium is diminished, the intestines float freely towards the anterior wall of the abdomen; there is often a history and signs of intemperance, and the urine is high-coloured, depositing lithates stained with purpurine. In the latter case the front of the abdomen is often dull from retraction of the bowels, there may be no history or symptom of intemperance, the omentum may be felt hardened and nodulated; or, again, a tumour may give evidence of cancer.

When paracentesis has been performed in cases of cancerous disease of the peritoneum, the ascitic fluid is often of a brownish colour, or reddened, from the presence of blood. In cases of simple chronic peritonitis, and of passive ascites, the fluid is generally straw-coloured. By rare exception ascitic fluid may be as thick and gelatinous as that of an ovarian cyst.

The deposit of a thick, firm clot, or the presence of flakes of coagulated fibrin, and the fact of leucocytes being present in such amount as to make the fluid turbid and to form a visible precipitate, are conclusive evidence that the ascites is of inflammatory and not passive origin.

A careful physical examination of the abdomen should always be made after paracentesis; this often clears up a doubtful case by leading to the discovery of a solid tumour, or of some disease of the liver or intestines, that could not previously be detected.

Chylous ascites.—In certain rare cases the fluid removed by tapping is opaque white; its milky appearance is the result of admixture with chyle, and is in most cases due to a lacteal vessel having opened into the peritoneal cavity by ulceration.

At the end of 1878 the writer had a patient with this chylous ascites, a woman. The fluid was much like milk in appearance, opaque, yellowish white, alkaline, with sp. gr. 1016. There was no subsidence after twenty-four hours, but scanty flakes of fibrin had separated, probably due to the accidental admixture of a trace of blood. There was no precipitate on heating after the addition of acetic acid, but it formed abundantly when ferrocyanide of potassium was added, and also with nitric acid.

In 1896 a second case occurred in Philip Ward, when the thorax was the seat of the same chylous effusion. It is rare in the pleural cavities, or still more rare in the pericardium or the tunica vaginalis. In most cases there is pressure upon the thoracic duct which leads to rupture of the distended receptaculum or lacteals, or of lymphatic vessels of the thorax. The opacity is not always due to fat, but to proteid granules.*

* Similar opaque fluid sometimes flows from a lymphatic vessel in the upper arm or the groin. Just as the lacteals are only intestinal lymphatics and contain transparent lymph in a fasting animal, so the lymphatics of other parts from time to time absorb fatty or proteid matters so abundantly that their contents are more or less opaque like chyle.

Origin.—First, ascites may be due to subacute or chronic inflammation as above described, whether simple, tubercular, or cancerous; secondly, it may be part of general dropsy, usually cardiac or renal; thirdly, and most frequently, it depends on portal obstruction. To distinguish certainly between passive and chronic inflammatory effusion is often as difficult as in cases of hydrothorax, hydropericardium, hydrocephalus, and hydrocele.

The conditions which lead to passive or dropsical effusion of portal origin are the following:

1. *Cirrhosis of the liver* is the most common cause of passive ascites, or dropsy of the peritoneum. It will be fully described hereafter (p. 534).

2. *Chronic inflammation of the capsule of the liver*, or, as it is often termed, *perihepatitis*, is also a frequent cause of ascites, both passive and inflammatory. At Guy's Hospital there is about one fatal case for every five of dropsy from cirrhosis of the liver; and this proportion would be greatly increased if we were to take into account those cases in which thickening of the capsule of the liver is merely a part of a general chronic peritonitis. If in a case of ascites the urine be healthy, there is little likelihood that the cause is thickening of the capsule of the liver; but when in a case of renal dropsy the abdomen is filled with fluid to a disproportionate degree, this is probably due to perihepatitis rather than to cirrhosis.

3. *Simple chronic atrophy* is another affection of the liver which may very occasionally cause ascites. The 'Pathological Transactions' furnish two remarkable cases of this kind, in which the abdomen contained a large quantity of fluid (1867 and 1868).

4. *Syphilitic disease of the liver* sometimes causes effusion into the peritoneal cavity. A striking case, which appears to have been of this kind, was recorded by Grainger Stewart. A patient had ascites, for which she was tapped twenty-one times, the enormous quantity of 606 pints being removed in the course of these operations. At first the paracentesis had to be repeated every fortnight, but the intervals gradually became longer, until at length she regained tolerable health. In twenty years (1860-82) there occurred in Guy's Hospital six cases of fatal ascites due to this cause. In several the liver could be felt during life to be enlarged and adherent to the parietes, with an uneven and nodular surface, and these characters more than once enabled a correct diagnosis to be made.

5. *Carcinoma of the liver* is another disease that may give rise to effusion of fluid into the peritoneal cavity. The presence of malignant growths in the substance of the organ itself is to be distinguished from the cases in which cancer merely involves the structures in the portal fissure. Both affections will be discussed afterwards (*v. infra*, pp. 552, 555).

6. Occasionally ascites is found to depend on the trunk of the portal vein (not its branches within the liver) being obstructed by plastic inflammation—*pylephlebitis adhesiva*. This is a very rare condition. The only case of ascites from adhesive portal phlebitis which the writer has seen occurred many years ago (1863) in a boy of ten, at Guy's Hospital: no other lesion was found after death, but the whole liver was channelled by dilated branches of the portal trunk.

Although adhesive pylephlebitis is a very rare disease, thrombosis of the portal vein is not at all uncommon, and probably takes place towards the end of many cases of cirrhosis, perihepatitis, and other forms of hepatic obstruction leading to ascites.

The curious cases of ascites of portal origin in children, not due to

alcohol, which have been described of late years by several French physicians, will be again referred to under the section on Cirrhosis.

Prognosis.—In most cases of ascites this is unfavourable. Some of the diseases that give rise to it are, from the first, malignant; and others do not cause effusion of fluid into the abdomen until they have reached an advanced stage. The first remark applies to cancer of the liver or peritoneum; the second to renal, cardiac, and hepatic dropsy. In cirrhosis of the liver, especially, death occurs in the majority of cases in six months after the detection of ascites.* There are, however, exceptions to this rule. One patient who recovered from ascites and jaundice under medicinal treatment remained well for several months, after which the fluid reaccumulated, and he returned to the hospital to die. He was supposed to have syphilitic gummata of the liver, but the disease proved to be cirrhosis. A patient of the writer's in Philip Ward in 1886 had been four years previously in the hospital, with hæmatemesis, ascites, and other signs of cirrhosis, and they had all disappeared.

When, however, permanent recovery takes place from ascites (whether after paracentesis or otherwise) the probability is that the effusion was the result of either chronic peritonitis or perihepatitis.

In children and young persons ascites is not infrequently curable, even when it is the result of tuberculous peritonitis.

Treatment.—Diuretics may often be prescribed with advantage. Of these none appear to be more efficacious than copaiba; but it often disturbs the stomach, so that the patient cannot continue to take it. The resin is less likely to disagree, and is no less efficient than the oleo-resin. The acetate or the acid tartrate of potass, the spirit of nitrous ether, the compound spirit of juniper, the decoction of broom-tops, and the infusion of digitalis, are all remedies of approved value; and a favourite prescription of Addison's was a diuretic pill containing the grey oxide of mercury, powdered digitalis leaves, extract of henbane, and powdered squill (of each a grain).† A useful and pleasant remedy is "Imperial drink," an infusion of lemons with cream of tartar and sugar, of which the patient should drink as freely as possible.

Purgatives, also, are useful, especially the compound jalap powder. But caution is needed, for they may set up fatal colitis.

Paracentesis.—In most cases tapping the abdomen is sooner or later necessary. It should be performed as soon as the peritoneum is full of liquid, or earlier if the breathing becomes hurried and shallow, from the diaphragm being pressed upwards and the lower ribs stretched.

The trocar should be of small calibre, and should be introduced in the median line below the umbilicus, it having first been ascertained that the spot selected yields a dull note on percussion, and consequently that the intestines are not in the way. The bladder should have been previously emptied. The trocar is then fitted with a long piece of elastic tubing by which the fluid can be carried into a pail placed below the patient's bed, and the entrance of air prevented.

The operation is not quite unattended with risk, immediate and prospective. The patient has now and then fainted as the fluid escaped.

* In some rare cases the umbilicus gives way, and allows the ascitic fluid to escape. Jenner recorded a case in which this happened with a report loud enough to be heard at a distance from the patient's bed. The fluid may continue draining away for a time, but this scarcely even postpones the fatal issue.

† Matthew Bailie was probably the author of this combination.

Some prevent this by having a jack-towel folded round the abdomen before the operation is begun, and tightened as the fluid escapes; others depend on keeping the patient's head low or giving him brandy or ammonia. Syncope was more frequent when a large trocar was used than with the gradual evacuation by one of Southey's tubes. When the fluid ceases to flow, the operator removes the cannula with one hand, while with the other he grasps the surrounding integument, so as to prevent the entrance of air into the abdominal cavity. A pad of lint is then placed over the wound, and upon this a few broad strips of plaster.

It occasionally happens that this fails to close the opening into the peritoneal cavity, and the fluid keeps oozing out. The leakage may generally be stopped by a suture.

In other instances tapping is followed by peritonitis, which proves fatal in the course of two or three days. Cases of Bright's disease are particularly liable to such a result, even when the trocar and cannula have been well sterilised.

After tapping, as a rule, the fluid begins to reaccumulate at once, being, indeed, poured out much more quickly than before, in consequence of the absence of pressure upon the secreting surface. The operation has soon to be repeated; and although the risks are now even less than before, it is also more likely that the relief will be but temporary.

When paracentesis fails to prolong life, it is not therefore useless. It almost always affords great relief, and is sometimes followed by unexpected benefit even in advanced cases of cardiac or portal dropsy.

In some cases the operation is as successful as one could wish. There may be no return of the ascites at all; or it may return very slowly. Moreover, paracentesis, by relieving the kidneys and the affluents of the vena cava from pressure, may do much to help the action of diuretics and increase the flow of urine.

The most favourable cases are those of ascites from primary peritoneal effusion, described on p. 488. In one such case under the writer's care, the patient, a woman of about forty, recovered completely after a single tapping, and in two others recovery ensued after one repetition.

In cases of chronic peritonitis with thickening the number of times paracentesis can be borne is sometimes surprising. The boy whose case is mentioned at p. 488 was tapped nearly a dozen times. Another patient of the writer's, who first came to him in 1884 with ascites, due to rupture of a compound ovarian cyst, was, before her death in 1893, tapped 292 times (see 'Path. Trans.,' vol. xlv, p. 111).

Of late years it has been proposed to open the abdomen in cases of ascites due to portal obstruction, and to suture the omentum to the parietes, so as to secure a passage for the blood in addition to the natural collateral channels.

DISEASES OF THE LIVER

JAUNDICE AND GALL-STONES

“Lurida præterea fiunt quæcunque tuentur
Arquati: quia luroris de corpore eorum
Semina multa fluunt, simulacris obvia rerum;
Multaque sunt oculis in eorum denique mixta
Quæ contage sua palloribus omnia pingunt.”

LUCRETIVS, iv, 334.

So-called bilious or hepatic dyspepsia—disturbance of secretion or elimination of bile—bilious symptoms—treatment by diet, drugs, and exercise.

Icterus—symptoms—tests for bile-pigment and for bile-acids in the urine.

Idiopathic jaundice: its course, pathology, and prognosis—Pruritus—Slow pulse.

Symptomatic jaundice—cardiac, febrile, toxic, pneumonic, pyæmic—from portal sepsis, suppurating hydatid, and acute tubercle—Jaundice from cirrhosis—

Epidemic jaundice—Weil's disease—other causes of jaundice.

Effects of permanent jaundice on the liver and gall-bladder—fatty stools, etc.—

Pathological theories of jaundice.

Treatment of jaundice, primary and symptomatic—of permanent jaundice.

Gall-stones—Their structure and varieties—Symptoms and events of biliary colic—treatment—Suppuration of the ducts and gall-bladder.

THE functions of the liver are more various than those of any other gland in the body. For beside secreting bile, it also provides the portal part of the systemic circulation; it is concerned in two important metabolic processes, the formation of glycogen and the formation of urea; it contains a large amount of cytogenic or lymphatic tissue; it has an important share in blood-making; and it contains at different periods a varying amount of fatty material, which it almost certainly secretes. Not only are its glandular, vascular, hæmatogenic, and metabolic functions, each liable to derangement, but the liver is also, like other organs, prone to inflammation, both chronic and acute, to the varied kinds of degeneration and atrophy, to the invasion of new growths, the formation of calculi, and the immigration of parasites.

It is difficult to arrange the diseases of the liver satisfactorily, since some of its disorders are still unknown in their origin and pathology, and others are only secondary to precedent local or general diseases.

In the present chapter, we will consider the liver as a secreting gland, and describe the functional disorders of secretion, the graver obstructions

which produce jaundice, and the concretions which mechanically interfere with the excretion of the bile, as well as other affections of the excretory channels.

The two following chapters, dealing with the structural diseases of the liver, will follow an anatomical arrangement, as being convenient and not more artificial than any other which is at present possible.

BILIOUS CACHEXIA.—We have already spoken of a kind of dyspepsia probably associated with chronic gastric catarrh, which is characterised by a furred tongue, sluggish bowels, and a sallow complexion (p. 320).

There is often a feeling of oppression and weight, with lassitude or irresistible drowsiness coming on about an hour after a meal. Muscular "rheumatism," lumbago, sciatica, and cramps in the legs may also occur. This form of dyspepsia has long been supposed to indicate "a sluggish state of the liver" and is often described as a bilious attack.

Another symptom called bilious is headache. The difficulty is to be sure whether this is due to dyspepsia, or to constipation, or to deficiency of biliary secretion, or to its reabsorption. Whatever its pathology, it is a dull, heavy pain, seated in the forehead, or more rarely in the occiput. It generally comes on when the patient first wakes in the morning, and usually follows some indiscretion in diet the night before. It is not identical with the "bilious" or "sick headache," described among the diseases of the nervous system as migraine (vol. i, p. 898), although in some cases it is hard to draw the line.

An occasional symptom is giddiness. Wilks says that, if due to digestive disorder, swimming in the head is especially apt to come on when the patient stoops or lays his head upon the pillow, and that it often passes off when he assumes the erect posture.

Patients speak of these symptoms as indicative of a "torpid state" of the liver, and think that they are caused by a deficiency in the amount of bile secreted. This opinion was formerly held by all physicians, who pointed to the fact that the complaint is often removed, at least for a time, by mercurials, which bring away from the bowels a mass of fæces, apparently loaded with bile. But Rutherford's elaborate and conclusive experiments proved, at least in the case of dogs, that the additional bile passed by the bowels is due to increased peristalsis or possibly contraction of the gall-bladder, but not to increased secretion.

In his 'Croonian Lectures' of 1874, the late Dr Murchison argued in favour of the popular view that these varied symptoms are not to be referred to catarrh of the stomach, or to constipation, but to a disordered liver, to bilious cachexia. And he further argued that since there is good reason to place the formation of urea in the liver, we are justified in ascribing to the supposed "torpid" or "inactive" or "sluggish" state of the liver, not only deficient secretion of bile, but deficient formation of urea. Lastly, he supposed that the metabolism of proteid matters, which in a healthy liver ends in urea, stops short in a sluggish liver at the formation of uric acid—a supposition now proved to be unfounded. Since uric acid is only a new name for lithic acid, and gravel, stone, and gout are undoubtedly connected with the presence of lithic acid and sodium urate in the urine or the joints, it would follow that we are justified in referring all the symptoms in question to the liver. Thus two of the most wide-reaching conditions of popular pathology, "torpidity of the liver," and "a gouty diathesis"—

"goutiness" if not gout—are alike embraced by this comprehensive theory. Not only gastralgia, flatulence, and acidity, headache, migraine, and vertigo, but also grinding of the teeth, singing in the ears,* drowsiness and sleeplessness, palpitation and heartburn, bronchitis and asthma, have all been included as due to the hypothetical state called lithæmia. Moreover, lithæmia and the uric-acid diathesis would also bind gravel to gout under the name of lithiasis.

Now that there is sometimes an excess of lithic acid, or, rather, of sodium-quadrurate in the blood is certain. It is the condition which precedes the deposit of crystalline biurates in the cartilage of joints, and so provokes an attack of articular gout. But that all the symptoms seen in persons subject to attacks of gout are due to "lithæmia" in this strict and accurate sense of the term, is an assumption of which there is no proof, and which is opposed by what we know of the effects of rheumatism; syphilis, and plumbism. These diseases, like true lithæmia, cause definite and recognisable symptoms, and we do not ascribe dyspepsia, constipation, or other common disorders to them.

Moreover it is an obvious, though frequent, fallacy to assume that the appearance of uric acid or of urates in the urine means excess of uric acid in the blood. It is not yet finally ascertained whether uric acid is separated or manufactured by the kidneys; and urates are deposited in all concentrated urine, in all highly acid urine, and in many specimens of healthy urine when exposed to cold.

If, warned by the past history of medicine, we refuse to accept vague hypotheses without criticism, we shall regard Murchison's lithæmia, like biliousness and goutiness, and the dartrous diathesis, as a purely hypothetical condition.

What remains is that some persons when suffering from constipation, with the furred tongue, and headache, and anorexia which go with it, are for the time of a sallow complexion, and "the white of the eyes looks yellow." This condition is accompanied by pale motions, and sometimes by itching of the skin and a slow pulse. Lastly, such a state will now and then develop into well-marked jaundice before it passes away.

Treatment.—For such patients there is no doubt that three or four grains of blue pill, with as much of the compound colocynth pill, followed by the traditional haustus sennæ or some less saline nauseous aperient, is the most successful treatment. Podophyllin was much recommended for cases of this kind, but the slowness and uncertainty of its action, and the disagreeable griping it causes in some persons, are great objections to its use. A much better substitute for calomel or blue pill is euonymin or iridin, in doses of two or three grains.

The popular "cure" for torpid liver, suppressed gout, biliousness, or lithæmia, is a visit to certain German watering-places, particularly Carlsbad. All such purgative waters contain sulphate of soda and chloride of sodium, and many of them sulphate of magnesia also.

The proper dose is about five ounces of Püllna water, seven ounces of Friedrichshall water, eight or ten ounces of Carlsbad water. The necessary quantity should be mixed with hot water, and taken the first thing in the morning before breakfast. Many patients, however, perhaps chiefly women, find they do better with Hungarian bitter water, Hunyadi Janos; and many others have no reason to forsake the cheaper saline laxatives of Epsom, Cheltenham, Seidlitz, or Rochelle. Which-

ever is selected, it should be taken before breakfast in not less than a tumbler of warm water. Most laxatives are better in combination. This is probably one advantage of natural waters over the sulphate of magnesia or of soda, or the tartrate of potash and soda, alone; but it is easy to imitate the waters of spas by adding a little carbonate of soda and common salt to the combined sulphates. What is sold as citrate of magnesia often consists of bicarbonate of soda and tartaric acid. The combination known as "Lamplough's Pyretic (or Antipyretic?) Saline" contains the same ingredients with about two per cent. of chlorate of potash; "Eno's (so-called) Fruit Salt" adds to these sulphate of magnesia and sugar. Such saline aperients are probably less injurious than patent purgative pills or lozenges or Indian "tamar," which often contain aloes, jalapin, or gamboge.

The benefit of "taking the waters" is due partly to purging, partly to a stricter regimen and more exercise. If bilious patients would rise early, walk or ride every day, eat and drink less, and once or twice a week take a blue pill overnight and Epsom Salts next morning, they would be cured as well at home.

The diet of the patient should be regulated by excluding rich dishes, and all melted butter, sauces, and condiments. Champagne and the stronger wines and malt liquors should be prohibited; and the patient should be limited to a very moderate allowance of light but sound Bordeaux or Rhine wine with his principal meals. Brandy or whisky diluted with water is a popular substitute for wine and beer, but in these cases is far less desirable in the writer's judgment than hock or chablis with seltzer water. The best beverage of all is the pure element, taken either hot or cold as the patient prefers.

The patient should also be made to take exercise. Of all kinds horse-exercise is the best. Rowing is also excellent, and any exertion which produces deep breathing and free sweating. Walking, however good in other ways, is perhaps least useful for this hepatic form of dyspepsia, and bicycling is not active enough for most patients otherwise in robust health. A quarter of an hour's game at rackets, for instance, is far more beneficial to most persons than an hour's walk. This advice applies particularly to persons much engaged in business which keeps them on their legs all day. A long walk only makes them more tired, and unable to digest the heavy meal which their sense of exhaustion prompts them to eat; whereas a much shorter time spent in riding or rowing or tennis exercises the whole body, and after (not before) a short rest, or, if needful, a quarter of an hour's sleep, they can enjoy dinner with a zest "that after no repenting draws."

ICTERUS.*—When the bile cannot escape by the natural passage, it is reabsorbed by the lymphatics of the liver and carried into the systemic veins, and to the heart. The bile-pigment is excreted in the urine, and deposited in the skin and other parts, so as to give the patient a yellow colour. This condition is jaundice or *icterus*.

* *ἰκτερος*, *galbula*, the golden oriole, which, by the doctrine of similars, was believed to cure a jaundiced person who caught sight of the bird. *Icterus* was applied both to the disease and the patient.—The Latin term for a person affected with jaundice was *arquatus*, and for the disorder *morbus arquatus*, i. e. *arcuatus*, from *arcus*, the rainbow. It is so used by Celsus and by Lucretius in the passage from his fourth book which stands at the head of this chapter. Other synonyms were *aurugo* (i. e. the golden disease) and *morbus regius* (Cels., iii, 24).—*Fr.* Ictère, Jaunisse.—*Germ.* Gelbsucht. In English the disease is popularly, by a natural tautology, called the yellow jaundice.

Many of the diseases of the liver are attended with jaundice, but by no means all. Moreover, it is sometimes seen without other evidence of hepatic disorder: not only in yellow fever and certain remittents, but also occasionally in typhus, in relapsing fever, and in pneumonia.

Jaundice is no *disease*, due to a single cause, and capable of being treated with drugs without further investigation of its origin. On the contrary, it is one of the best examples of a condition, which appears a disease to the laity, as it once appeared to physicians, but which we now regard as only a *symptom*, to be traced to its cause whenever we can do so.

General Characters.—The parts that best display the yellow colour are those which are naturally pale. The redder parts of the skin are less decidedly altered in appearance, but in the superficial mucous membranes the difference is more striking. Whereas even in deep jaundice the lips show no change, the yellow hue is well marked in the lining of the cheeks, and we always look to the conjunctivæ for the first signs of icterus.

The internal parts are affected as well as the surface of the body; the connective tissue generally, the lining of the heart and blood-vessels, the serous membranes and also any serous effusions.

Of the *viscera*, the lungs and the kidneys, and other organs which are not so red as to conceal their yellow tint, are evidently jaundiced. The liver displays the same colour, often in an extreme degree. Later observations in the deadhouse at Guy's Hospital confirm those of Moxon, who stated that the *brain* remains unstained with bile even in advanced jaundice.

The transparent humours of the *eye* are sometimes yellow, and sometimes not. It is generally supposed that (as Pope, following Lucretius, said) "all looks yellow to a jaundiced eye;" and by actual trial this may be demonstrated in many patients. But sometimes they are able to discriminate yellow or buff objects from white without difficulty.

The colour of the *skin* in jaundice varies according to its intensity and its duration. If the natural escape of the bile be suddenly and completely arrested, the body may quickly assume a deep orange hue. But in many cases this is more gradually developed, and the tint at first is a pale sulphur yellow. The whole cutaneous surface is not equally discoloured; the jaundice is generally more marked in the face, arms, and abdomen than in the legs.

In very chronic cases the colour becomes greenish, and at last passes into a dark olive tint. This change corresponds with the alteration which bile undergoes when exposed to the air, by the conversion of its bilirubin into biliverdin, and of this again into choletelin or bilifuscin. Persons in whom the skin assumes the dark green colour above referred to were formerly said to have "black jaundice,"* and it was supposed to be an indication that the hepatic disease was of a malignant nature. We now know that it means only that the jaundice has lasted for a long time, but it is true that such cases are often cancerous.

There is seldom difficulty in telling whether a patient is jaundiced if we see him by daylight. The hue is very different from the greenish-

* In the pathology of Galen, while the yellow bile was secreted by the gall-bladder, the black bile was formed by the spleen. Hence the *dyscrasia* or ill temperament due to excess of yellow bile was called choleric or bilious; that due to excess of black bile, melancholic or atrabilious.

"Quatuor humores in humano corpore constant:

Sanguis cum cholerâ, phlegma, melancholia.

Terra, melan: aqua, phleg: et aer, sanguis: coler, ignis."

Regimen Sanitatis Salernitanum, v, 257.

yellow hue of chlorosis, or the yellowish waxen tint often seen in cancerous disease of the abdomen, or the dusky sallow look of malaria. Nor is the olive-green tint of inveterate jaundice like the bronze colour associated with disease of the adrenals, although before Addison's discovery these patients were supposed to be jaundiced. In all the conditions just mentioned, particularly in the last, the conjunctivæ retain their natural pearly-white appearance.

Cases are, however, sometimes met with in which it is difficult to distinguish jaundice from idiopathic (so-called pernicious) anæmia, when the skin has a clear yellowish tint, and occasionally the conjunctivæ also. In elderly persons the presence of a little fat in the submucous tissue causes a partial yellow colour, but here the shape of the lobules can be seen, and the vessels which supply them.

One must bear in mind that the yellow tint of jaundice is invisible by gas- or candle-light, and almost so by electric light.

Several of the *secretions* of the body contain biliary colouring matter in jaundice. The sweat is yellow, so that the patient's linen is often much stained under the armpits; and the lachrymal secretion is discoloured. The milk may be bile-stained, as observed by Bright;* but the saliva is colourless, and the secretions of the muciparous glands remain unaffected. The gall-bladder and the ducts of the liver itself secrete a colourless mucus. That the intestinal mucus and succus entericus contain no bile-pigment is evident from the fact that the fæces are of a greyish-white colour, or (to use the common expression) "clay-coloured." Dr Fenwick pointed out that in cases of jaundice the sulphocyanide of potassium normally present in the saliva is not to be found, and the writer has confirmed this observation. Dropsical serum is deeply stained with bilirubin in jaundiced patients.

The colour of the *urine* in jaundice may vary from a yellow, scarcely deeper than natural, to a dark brown, a greenish brown, or a black so intense that one can recognise its colour only by looking at the margin of the fluid, or pouring it out in a thin layer, or making it froth. The presence of bile-pigment in the urine is constant in jaundice, as we should expect from the diffusibility of bilirubin. Indeed, we sometimes obtain more delicate indications of slight or early icterus from the urine than from the conjunctiva. There is only one condition in which the urine may for a short time have its natural appearance, although the patient's skin is still yellow. This is when the cause of the jaundice has been suddenly removed, particularly if it has lasted for a long time. The bile-pigment then ceases to circulate in the blood, and the kidneys no longer excrete it; but the skin does not at once give up all the colouring matter that had been deposited in its tissues, and remains for a few days yellow.

Test for bilirubin.—The presence of bile-pigment in the urine is confirmed by a chemical test, known as Gmelin's, which is capable of detecting it in minute proportion. It consists in the addition of fuming nitric acid (containing nitrous acid) to a small quantity of the urine. This causes a beautiful play of colour if bile-pigment be present. A good way of employing the reagent is to pour a drop or two of the urine on the flat

* Heberden affirms the contrary, and an error of observation is incredible in either case. The writer has never seen jaundice during lactation, and has inquired in vain of physicians more likely to observe the coincidence. More recently, however, Dr H. W. Beach has obligingly informed him that in a case he saw in 1895 there was deep yellow staining of the milk, which continued slightly stained when the urine and the skin were gradually recovering their healthy colour.

surface of a white plate, and then carefully to add to it a single drop of the nitric acid. Around the drop a series of colours is developed, rapidly passing through the shades of green, blue, and violet, into red, and finally becoming a dirty yellow. Neubauer and Vogel recommended that nitric acid should be poured into a conical glass, and that the urine should then be carefully spread over its surface by means of a pipette. The play of colour begins where the fluids come into contact with a beautiful green ring, which gradually extends upwards, and at its under surface exhibits a blue, violet, red, and lastly a yellow ring.

Mere darkening of the urine by oxidation of its natural yellow pigment into a reddish-brown colour is a familiar effect of nitric acid quite distinct from that just described. When, however, nitric acid is added to urine, red and violet rings may be produced by another substance, of which a small quantity is present in healthy urine, and which is increased under various pathological conditions. This is *indican*, once called uroxanthin, the same principle which, when obtained from the indigo plant, has long been known as the mother-substance of the indigo pigments.

The most delicate of all methods of applying Gmelin's test for bile-pigment is to shake a large quantity of the urine with chloroform. This extracts any bilirubin that may be present, and when nitric acid is afterwards added the reaction is apparent.

Tests for bile-acids.—It has been disputed whether the sodium glyco- and tauro-cholates of the bile are excreted by the kidneys in jaundice, and whether this is the case in some forms of jaundice and not in others. It is now certain they are present, but only in traces, and that their presence or absence affords no clue as to the origin of the icterus.

The ordinary chemical test for the biliary acids, or rather for the cholic acid which they both contain, is known as Pettenkofer's. It consists in the admixture of a few drops of syrup or a few grains of sugar (either sucrose or glyucose) with the liquid suspected to contain biliary acids, and the subsequent addition of strong sulphuric acid, the test-tube being kept cool in water. A beautiful violet colour appears if the acids of the bile are present. There is no difficulty in thus demonstrating the presence of cholic acid when bile has been purposely added to urine, but it is doubtful if the presence of the bile-acids can be thus observed in urine without their having been so added. Moreover the test may be due to other possible constituents of the urine beside cholic acid, and when the latter is proved to be present by more elaborate methods than Pettenkofer's we learn little or nothing more.

Only small amounts of those acids are discharged from the body in health. According to Bischoff not more than a quarter of the amount of biliary acids poured into the intestine by the liver passes away in the fæces, and even this has undergone chemical changes. The greater part is reabsorbed into the blood; but it is very improbable that this is further transformed, as Frerichs supposed, into bilirubin.

IDIOPATHIC JAUNDICE.*—In many cases of jaundice there is scarcely any other symptom than the facts that the skin and conjunctivæ are of a deep yellow colour, that the urine contains bile-pigment, and that the stools are clay-coloured. There is not, nor has there been, any pain or uneasiness in the region of the liver. Often the patient says that he feels perfectly well,

* *Syn.*—Simple jaundice—Catarrhal jaundice.

and would not know that anything was wrong with him but for seeing his yellow face in the glass. The temperature is normal and the appetite good.

After a variable period—sometimes a fortnight or less, sometimes six weeks or more—the jaundice subsides. The first sign of improvement is generally that the motions regain their natural colour; the urine soon ceases to contain bile-pigment, and a few days later the skin and conjunctivæ regain a healthy appearance. This favourable change often takes place about the twenty-first day. At no period of the complaint can one generally make out that the liver is enlarged; from beginning to end there is nothing to throw light on its cause. For jaundice of this kind “primary,” “idiopathic,” and “benignant” seem at present to be suitable adjectives.

Pruritus.—Jaundice is sometimes attended with itching of the skin. In certain persons papules develop themselves whenever the skin is scratched; hence when they are jaundiced they often present an eruption of pimples, and the marks of their finger-nails—a condition sometimes mistaken for scabies. Graves observed urticaria develop; and he also noticed that itching of the skin sometimes precedes jaundice by a considerable interval, in one of his cases a period of ten days. Addison used to teach the same fact, and in one case predicted an attack of jaundice when a patient complained of itching for which no explanation could be found, and was justified by the result.

Slow pulse.—Another symptom sometimes observed in jaundice is infrequency of the beats of the heart. The pulse occasionally falls to 50, 40, or even 20 in the minute. It has been found in experiments upon animals that the pulsations of the heart are much reduced in frequency by the injection of the salts of the biliary acids into the circulation.* It was, therefore, supposed that in such cases of jaundice the blood contains these acids. But chemists have hitherto failed to discover them, except in traces, in the urine, whether in this or in other forms of jaundice; and, since they are readily diffusible, they can scarcely accumulate in the blood so as to affect the heart while freely excreted by the kidneys. The prognostic significance of a slow pulse in jaundice is doubtful; it is certainly better than the quick pulse of inflammatory jaundice. Like xanthopsia and pruritus, this symptom is more often seen in idiopathic than in secondary jaundice, and though frequent is by no means constant.

Pathology.—The complete absence of bile from the fæces in this form of jaundice affords a presumption that there is some obstacle to its flow; and the common theory is that simple jaundice depends upon catarrh of the larger bile-ducts. It is believed that their lining membrane is swollen, and that mucus is secreted, which obstructs the channel. One difficulty is that patients never die from, and rarely during, this idiopathic and benignant form of jaundice, so that we have little or no knowledge of its morbid anatomy. Another is that we have no corresponding instances of spontaneous catarrh of a duct, with obstruction and reabsorption of the secretion, in the case of the ureter, or other ducts of glands.

A more probable suggestion is that catarrh of the duodenum obstructs the oblique and narrow passage of the duct through the walls of the gut; but here the difficulty is that the jaundice does not more constantly follow what is probably a frequent disorder. Moreover we should have expected

* Röhrig, ‘Ueber d. Einfluss d. Galle auf d. Herzthätigkeit,’ 1863. See also a paper by Dr Legg (‘Proc. Royal Soc.,’ 1876) with references to Traube’s observations, and a fuller account with tracings in his work on ‘The Bile, Jaundice, and Bilious Diseases,’ p. 204.

that chronic catarrh would have produced permanent jaundice, as chronic obstruction in the nasal duct produces permanent epiphora.

In acute yellow atrophy of the liver the ducts are not obviously obstructed (cf. *infra*, p. 548); and there is no evidence or likelihood of catarrh of the gall-ducts when icterus accompanies pneumonia. That the low pressure in the portal veins may be readily overcome is true; but at present the source of the obstacle in "simple" jaundice is not clear.

It has long been taught that jaundice sometimes follows directly upon some mental shock. Sir Thomas Watson mentions the case of a young medical friend of his who became jaundiced from anxiety before an examination at the College of Physicians; and he refers to another case in which an unmarried woman, on its being accidentally disclosed that she had borne children, became in a very short time yellow. Similar instances are on record, some of them more marvellous than credible. Except the fact that they are associated with mental emotion, there is no difference between them and other cases of simple icterus in symptoms or course. They have been ascribed to "the effect of the mind on the body," to sudden fall of blood-pressure, to sudden spasm of the gall-duct, and to rapid toxæmia.

That cerebral disturbance due to mental emotion may arrest the secretion of bile, is no more wonderful than that it excites that of the lacrymal glands, of the intestines or of the kidneys; but in the case of jaundice there is no over-secretion like that of sorrow, of terror or compassion, or of hysteria, to explain the yellow colour.

An affection having the characters of simple jaundice has occasionally prevailed *epidemically*. Several instances of this were collected by Frerichs. Murchison mentions a remarkable outbreak of the same kind which occurred at Rotherham. In 1862 this town was visited by enteric fever, which proved very fatal. Early in the following year jaundice became epidemic. It is said that in February no fewer than one hundred and fifty persons were suffering from it; and it was curious that none of those who were attacked had passed through the fever. Other examples are recorded among our troops in India by Morehead and Goodeve (cf. *infra*, p. 511).

Diagnosis.—The characters which distinguish idiopathic from secondary jaundice are chiefly negative; and one might expect that the diagnosis would be uncertain, since in some cases of cirrhosis, of cancer of the biliary passages, and even of gall-stones, jaundice may be the first symptom. But whereas these diseases seldom occur in young and hitherto healthy subjects, "simple" jaundice usually attacks the young and robust. Practically the only serious difficulty to be considered is the possible, though happily very rare, supervention of acute atrophy of the liver, on an attack of apparently—and perhaps really at first—benignant jaundice.

SYMPTOMATIC JAUNDICE.—Jaundice is often seen in cases of advanced *heart disease* with an enlarged and congested liver. The naturally low pressure in the portal vein appears to be overbalanced by the increased pressure in the right auricle and hepatic veins, and the stasis thus produced prevents secretion of bile. It is usually an icteric tinge, seldom a deep discoloration.

As a complication in some of the *specific fevers* jaundice is not infrequent. It is always present in "yellow fever," and is a frequent and not always an unfavourable symptom in "relapsing fever" and in malarial remittents. It sometimes, though very seldom, occurs in typhus, and in

this disease almost every patient who becomes jaundiced dies. In enteric fever and scarlatina jaundice is extremely rare.

The icterus which frequently follows the bites of venomous *snakes* may be mentioned in this connection.

Another specific febrile disease in which jaundice may occur is *pneumonia*. This has been supposed to be due to an extension of inflammation through the diaphragm to the upper surface of the liver—an almost absurd notion, for there is no sign of inflammation of the diaphragm, pleura, or peritoneum between liver and lung; pneumonia cannot “extend” to anything but pulmonary tissue; and even if “inflammation” does attack the liver, it does not in itself cause jaundice. At least one case of this kind has been observed at Guy’s Hospital in which jaundice occurred as a complication of pneumonia of the *left* side. The prognosis of cases accompanied by icterus does not appear to be affected.

General pyæmia is often accompanied with jaundice; indeed, a slight yellowness of the skin is one of its most frequent symptoms. Wilks investigated the question whether those cases of pyæmia in which abscesses occur in the liver are or are not particularly liable to be accompanied by jaundice, and he came to the conclusion that the local disease had nothing to do with the production of this symptom.

Portal pyæmia.—Besides these general maladies, there are certain diseases of the liver itself, attended with fever, and often with rigors, local pain and tenderness, which also give rise to jaundice. Icterus is a rare and accidental symptom in cases of *tropical hepatic abscess*; but it often accompanies a purulent *pylephlebitis*, or inflammation of the branches of the portal vein with their sheath of connective tissue. The following is a striking case of this kind.

A man aged thirty-seven was admitted into Guy’s Hospital under the care of the late Dr Barlow, exceedingly ill with jaundice, fever, and delirium; and he died in two days. He had been apparently well a week before, but he suffered from stricture of the rectum. The liver was very large, and its tissue was suppurating throughout. The branches of the portal vein were all distended with soft thrombi of a brownish colour. The main trunk of the vein contained a dirty-looking fluid. The suppuration of the liver was evidently due to septic absorption by the veins of the rectum, which was extensively ulcerated.

Other causes of septic or suppurative pylephlebitis are—ulceration of the stomach or bowels, abscess of the spleen, suppuration of the mesenteric glands, and the penetration of one of the veins which go to form the portal trunk by foreign bodies; in a case quoted by Frerichs, a fish-bone had entered the inferior mesenteric vein.

Another cause of jaundice with pyrexia is suppuration excited by the presence of *gall-stones* in the gall-bladder or hepatic ducts. This possibility must never be forgotten in any case of biliary colic in which febrile symptoms show themselves.

A suppurating *hydatid* cyst in the liver often communicates with a large bile-duct, so that portions of the hydatid, or of its capsule, detached by sloughing, may enter the bile-duct and obstruct it by a kind of septic embolism. The writer saw an interesting case of this kind, which occurred in Dr Barlow’s practice at Guy’s Hospital, a few days later than the case of pylephlebitis above mentioned. The liver was found after death to contain a suppurating hydatid cavity which held three pints of fluid. The bile-ducts throughout the organ were suppurating, and the main canal was obstructed by a piece of detached membrane rolled up into a cylinder.

A very rare cause of febrile jaundice is *acute tuberculosis* of the

liver. An instance of this was recorded by Murchison in a woman forty years of age. Another case was observed some years ago at Guy's Hospital. A man, aged thirty-seven, died with jaundice and symptoms like typhoid fever; the history was imperfect, but he was stated to have had jaundice only five days. After death the liver was found to be full of tubercles, there being as many as fifty to the square inch; but there was also pneumonia of the left lung, which may possibly have caused the icterus.

Jaundice from cirrhosis of the liver.—In the majority of cases cirrhosis runs its chronic course without marked icterus. There is, nevertheless, a considerable minority in which jaundice is present, and sometimes it is the most obvious symptom of the disease. Among one hundred and thirty cases occurring in the *post-mortem* room of Guy's Hospital, in which the liver was found after death to be cirrhotic, Dr Fagge found jaundice in thirty-four, and in ten it was deep. During this period there were only some sixty other cases in which jaundice was present. The fact is that the frequency with which cirrhosis of the liver occurs is far in excess of that of the other organic diseases that give rise to jaundice; and consequently, although marked jaundice is not a very common result of cirrhosis, cirrhosis is by no means an uncommon cause of jaundice.

Jaundice due to this disease has some peculiar characters. It is frequently gradual in its onset, it is often slight in degree, there is seldom complete absence of bile from the *fæces*—and it is unattended with pain, in contrast to jaundice from gall-stones or cancer. On the other hand, ascites is frequently present. In fact, the concurrence of jaundice with ascites is scarcely seen except in cirrhosis and cancer.

Weil's disease.—Some cases of icterus are apparently due to poisoning with ptomaines from diseased meat, sausages, or tinned fish. To this group seem also to belong certain cases, described by Weil in 1886. Jaundice with enlarged and tender liver and with pyrexia had occurred epidemically in certain towns of Germany, and also in Servia and in Egypt. It differed therefore from the epidemic but uncomplicated cases referred to on p. 509. Weil found it occur among male adults, and usually among butchers, but this was probably an accident. It runs an acute febrile course for six or eight days, is not contagious, is apt to relapse, and has a favourable issue. Beside an enlarged liver and spleen, the jaundice and pyrexia, there is a slow pulse and occasionally albuminuria. The pathology is unknown, but probably some cases depend on impacted gall-stones with inflammation of the bile-ducts, and this would agree with the fact that sometimes the result is permanent jaundice. Other cases of epidemic febrile jaundice referred to the same category appear rather to belong to remittent fever, and some, with vomiting and coma which ended fatally, with hæmorrhage in various organs, are more like acute yellow atrophy. (See '*Wiener med. Wochenschrift*,' No. 26, 1890, and Nos. 6, 7, 8, 1898; '*Lancet*,' 1889, vol. ii, p. 1109.) In the '*British Medical Journal*' for June 22nd, 1898 (p. 212), will be found a case of febrile jaundice, and a summary of forty-five cases more or less similar, mostly in children, and with only one death.

Dr Weil's original title was "A peculiar acute infective disease, characterised by enlarged spleen, jaundice, and nephritis," and most of his cases may be regarded as febrile epidemic jaundice of infectious or toxic origin. They are certainly not "*Typhus biliosus*" (Relapsing fever) nor enteric fever. The acute onset, febrile symptoms with albuminuria, short

course and favourable event, are, with the icterus, the leading characters. In the few cases which have proved fatal, the liver has been found large, soft and fatty, with exudation of leucocytes, and there have been numerous ecchymoses in the internal organs.*

Recurrent icterus.—Cases of jaundice have been described in France by Hayem and Levy (1898) which are paroxysmal in course and are associated with a large spleen and marked anæmia: they last for several years, and do not seem to be dangerous. Their origin is supposed to be in intestinal toxins absorbed by the portal vein. The full title of this group of cases is *Ictère infectieux chronique spleno-mégalique à poussées paroxysmiques*.

Icterus gravis.—Jaundice is an early and constant symptom of the remarkable disease known as *acute yellow atrophy*. This will be fully treated in the next chapter (p. 543), as will be the effect of poisoning by *phosphorus* in producing jaundice (v. *infra*, p. 548).

Malignant jaundice.—Icterus is often due to *cancer*, either of the liver itself or of the biliary passages, and will come under notice as a symptom of malignant growths in and about the liver.

Icterus neonatorum.—Deep and permanent jaundice may be caused in infants by a congenital obliteration of the common bile-duct, apparently resulting from intra-uterine perihepatitis; several instances of this have been recorded. The jaundice appears a few days after birth, and is attended with purpura and hæmorrhage from the bowels and from the umbilicus. If this is not fatal, death is due to progressive atrophy, with vomiting and diarrhoea; in two instances the child lived as long as six months.

Jaundice in infants is also said to arise from plugging of the common duct by inspissated bile, as in a case quoted by Murchison. But in many cases of supposed jaundice in new-born children the yellow tint is the result of changes of the blood in the over-congested skin, "the vivid redness of the new-born baby" (to use Murchison's expression) "fading, as bruises fade, through shades of yellow into the genuine flesh-colour."

Stricture.—Another very rare cause of permanent jaundice is stricture of the common duct, resembling stricture of the urethra. This condition is generally caused by cicatrization of an ulcer, itself probably set up by a gall-stone; or the submucous tissue may be converted into an indurated mass of new growth like annular stricture of the rectum.

External pressure.—Permanent jaundice may also be caused by an external tumour, of any kind, pressing upon the hepatic or common duct. Tuberculous glands in the portal fissure or an aneurysm of the hepatic artery have occasionally obstructed the flow of bile. Such cases have generally been attended with severe paroxysmal pain—like that produced by impacted gall-stones.

In the case of a boy under the writer's care some years ago, jaundice was caused by the pressure of a hydatid cyst upon the common bile-duct in the portal fissure. Ascites was also present from obstruction to the flow of blood in the portal vein.

Anatomical effects of persistent jaundice.—Whatever its cause, jaundice from obstruction of the biliary ducts leads to a definite series of further changes. The gall-bladder and all the biliary passages become dilated at first with bile, and afterwards with mucus of a greenish tinge, or perfectly

* The outbreak described by L. Klein and Schütz, in 1898, occurred in an Austrian regiment and was attributed to the men swallowing dirty water; at least no more cases occurred after bathing was prohibited.

colourless. The gall-bladder may thus come to contain many ounces, and may be felt as a smooth rounded mass below the liver.

Very often there are gall-stones in it as well as mucus, and their presence has been sometimes detected by palpation. They yield a peculiar crackling sensation, which has been compared to that produced by grasping a bag of hazel-nuts or rolling pebbles about in the mouth.

If the bile-ducts become infected by septic organisms, the gall-bladder suppurates and may point and open externally. The result is the production of a fistulous opening, through which after a time green bile free from pus is discharged—from eight ounces to two pints daily. The jaundice may then subside; but the patient continues to lose flesh and strength, and before long dies exhausted.

The hepatic ducts become dilated in cases of permanent jaundice; they may be larger than the branches of the portal vein with which they run, and may be visible on the surface of the liver.

In 1895 a patient under the writer's care, sixty-nine years old, had suffered from jaundice twenty-five years before and recovered. Jaundice returned a week before death, with ascites, loss of flesh, and a palpably distended gall-bladder. After death, cancer of the head of the pancreas was found, and also of the common bile-duct, and the whole liver was in the condition of a cirrhotic lung with saccular bronchiectasis; the bile-ducts dilated into large sacculi, separated by little but fibrous tissue.

A more frequent condition is uniform tubular dilatation, like bronchiectasis in children (vol. i, p. 1069). The enlargement of the ducts appears to be the cause that the liver as a whole is larger than natural in the early stages of this form of jaundice. But after a time the organ begins to shrink, and at length becomes much smaller than in health.

Another change in the liver consists in its assuming a dark olive-green colour, probably due to conversion of the bilirubin in the hepatic cells into biliverdin.

The connective tissue in the portal canals becomes thickened when the common bile-duct is obstructed. Dr Wickham Legg showed that in the lower animals the operation of ligaturing the bile-duct is followed by an overgrowth of connective tissue as great as in intense cirrhosis, and his observations have been since confirmed. Dr Fagge believed that a similar change occurs very frequently in cases of obstructive jaundice in man, although not to the same degree.

The liver-cells appear not to undergo any change beyond being somewhat reduced in size.*

Fatty stools.—In 1832 Bright recorded some cases of jaundice in which a substance like fat was either passed separately from the bowels, or soon divided itself from the rest of a stool and lay upon the surface; "sometimes forming a thick crust, particularly about the edges of the vessel, if the fæces were of a semi-fluid consistence; sometimes floating like globules of tallow which had been melted and become cold; and sometimes assuming the form of a thin, fatty pellicle over the whole, or over the fluid parts in

* In 1843 Dr Thomas Williams recorded a case of obstructive jaundice, caused by malignant disease of the head of the pancreas, in which almost all the cells of the liver were found to be broken down into fatty globules and granular matter ('Guy's Hosp. Reports,' 2nd ser., vol. i, p. 444). But no one has since recorded a similar observation, except when cerebral symptoms had been present, such as to justify the opinion that the disease was acute yellow atrophy. This occurred in a case which Murchison placed on record in the 'Pathological Transactions' (vol. xxii, p. 159).—C. H. F.

which the more solid fæces were deposited." This state of the fæces was so marked that it was noticed by the patient before Bright saw the case. The oily matter had generally a slight yellow tinge, and a most foetid odour. Bright was himself disposed to regard this peculiar condition of the evacuations as due to disease of the head of the pancreas and of the duodenum, and he seems to have thought that the presence of jaundice in his cases was accidental. Later writers also have generally attributed the symptom to obstruction of the pancreatic duct preventing the fatty matters of the food from being digested and absorbed. It is, however, possible that the presence of fat in the fæces in Bright's cases was due to obstruction of the common bile-duct rather than of the duct of the pancreas.*

Dr Walker, of Peterborough, brought before the Royal Medical and Chirurgical Society, in 1889, two cases in which the fæces were colourless though the patient was not jaundiced, and after death the pancreatic duct was found occluded, but not the bile-duct.

Emaciation.—Dogs in which a biliary fistula has been formed require a larger quantity of food to maintain their nutrition than before. This corresponds with the fact that in most protracted cases of jaundice the patient becomes emaciated, although free from fever or malignant disease. Nevertheless in a patient of Mr Mayo Robson, in whom all the bile escaped for many months by a biliary fistula, there was no loss, but a gain of weight.

Flatulence, &c.—Another result of the prolonged absence of bile from the bowels is that their contents undergo putrefactive changes, the occurrence of which is prevented, under normal conditions, by the antiseptic properties of bile. Hence the evacuations often have a foetid odour, and gases are generated which cause tympanitic distension of the abdomen. The contents of the intestines may probably in this way acquire irritant properties, and so set up diarrhoea. More often obstinate constipation is present in jaundice, from absence of the bile as the "natural purgative" or stimulant to the peristaltic action of the bowels.

Xanthelasma.—Persistent jaundice, from whatever cause, is apt to lead to a remarkable affection of the eyelids and other regions called originally "vigililigoidea" by Addison and Gull ('Guy's Hospital Reports,' 1851 and 1887), but now known as xanthoma or xanthelasma. It will be described in a future chapter among diseases of the skin.

Theory of jaundice.—It has been long supposed that there are two distinct forms of jaundice, in one of which the bile-pigment is secreted by the liver as usual, but, owing to *obstruction*, is afterwards reabsorbed into the blood; while in the other the secreting action of the liver is *suppressed*, so that the bile-pigment in the skin and urine must have been formed by some other organ or in the blood itself. Undoubtedly in certain cases of jaundice the ducts are mechanically obstructed, while in other cases they are apparently patent. Is the jaundice essentially different in its origin in these two classes of cases? Must we admit two kinds of icterus—the one hepatogenous, the other hæmatogenous?

One distinction between these two forms of jaundice has been supposed to be that bile-acids are present in the urine in cases of obstructive jaundice,

* Bidder and Schmidt found that in dogs, after the bile-duct was ligatured, the amount of fat that could be absorbed from the intestines was reduced to less than one half, and sometimes even to one fifth or one seventh of the quantity that the animal could digest before.

and are absent when the bile-ducts are free ; but we have seen this distinction to be untenable (p. 507).

After death in cases of jaundice due to whatever cause, the biliary passages are found to contain, not bile, but an almost colourless mucus. As Moxon remarked, the contrast is at first sight astonishing between the deep yellow fluid that fills distant serous cavities in such cases and the clear liquid in the ducts of the liver itself. The absence of bile from the biliary passages is, however, easily explained. The secretion of bile takes place under very low blood-pressure ; hence, when the common duct is obstructed, the entrance of bile into the biliary passages is at once arrested. Then mucus secreted by the walls of these passages and by the gall-bladder soon displaces the last trace of bile ; and, like all mucous fluids, it is itself unstained by bile-pigment.*

The theory of a hæmatogenous form of jaundice is inconsistent with the physiological doctrine that the bile-pigment is in health formed by the liver, and does not pre-exist in the blood. No bile-pigment can be detected in the blood in health. The probability seems to be, as Dr Gamgee ably states the case, that what is true of animals is true in man ; that the bile-pigment is formed in the liver—not in the general circulation ; and, hence, that without a liver there can be no icterus. Dr William Hunter believes that the formation of bilirubin takes place in the intestinal and splenic, as well as the hepatic districts of the portal circulation ; but he agrees that all jaundice is due to obstruction and none to suppression.

Kühne's theory of a hæmatogenous jaundice due to disintegration of hæmoglobin in the blood has been now disproved.

From experiments performed on geese and pigeons by Minkowski and Naunyn and by Stern, it appears certain that, when the stream of portal blood is diverted from the liver, no bile is manufactured and no icterus appears. This is far better evidence than the older experiments on frogs by Moleschott, in which the entire liver was excised without causing jaundice.

If then we admit as the best working hypothesis that all jaundice is obstructive or "hepatogenous," we must suppose that in cases where no obvious mechanical obstruction is found, there is either "catarrh of the duct," or temporary "blocking by inspissated mucus," or so viscid a condition of the bile that it will no longer flow. The last condition has been actually observed by Afanassiew in dogs which have been jaundiced by the administration of toluylen-diamine. There may perhaps be, in some cases, compression of the smaller ducts by vascular turgescence ; but Dr Wm. Hunter regards catarrh of the bile-passages, which is markedly present, as the most important cause of this artificially produced toxic icterus (Brit. Med. Asso., Aug. 20th, 1892 ; 'Path. Trans.,' 1890 ; see also Dr Auld's chapter in his 'Selected Researches in Pathology,' 1901, pp. 62—73).

Method of absorption of bilirubin.—It was formerly assumed that the bile was absorbed after secretion by the veins of the ducts. But experiments on dogs by Kufferath in 1880, followed up by a careful inquiry directed

* If this explanation be true, a colourless mucus should be found in the bile-ducts in cases of obstructive jaundice only when the obstruction is complete. I searched through the pathological records of Guy's Hospital for twenty years without discovering any exception either to this rule or to that from which it is derived : on the one hand, there was no case of long-continued complete obstruction in which the ducts contained a liquid highly charged with bile-pigment ; on the other hand, there was no case of partial obstruction in which they contained a colourless mucus.—C. H. F.

to this point by Dr Vaughan Harley under Prof. Ludwig's guidance in 1891, prove that if the thoracic duct is tied after ligature of the common bile-duct, no jaundice ensues. This appears to demonstrate that the lymphatics only, not the blood-vessels, absorb the bile.

Whether in all cases of jaundice the bile is first secreted and then reabsorbed, to pass by the hepatic lymphatic vessels and the thoracic duct into the blood, is still doubtful. It is possible that in some cases the bilirubin is never excreted at all, but after being formed in the portal capillaries, passes on directly into the hepatic veins. The probability, however, is that it is in all cases first manufactured by the liver-cells, and then passed into the ducts to be taken up by the lymphatics.

Icterus by obstruction, and reabsorption of the secreted bile from the bile-ducts into the lymph-passages of the liver and so into the general circulation, is now a well-ascertained process. That slight degrees of obstruction may produce jaundice is shown by the familiar case of nutmeg liver in chronic heart disease.

Frerichs's supposition, adopted by Murchison, of excessive secretion of bile and of reabsorption of the excess from the intestine, is unsupported by evidence and contrary to analogy.

Treatment of jaundice.—The *idiopathic* form is so uncertain in its course that it might well appear a hopeless task to determine whether remedies are capable of abridging its duration. On the Continent the most efficacious remedy is believed to be taking the waters at Vichy, Ems, Kissingen, Marienbad, or Carlsbad. Those of Vichy are strongly recommended by French physicians, while German writers speak no less highly of Carlsbad. All these springs contain a considerable quantity of the salts of soda, especially the sulphate and carbonate; and the same salts are believed in this country to be useful in the treatment of jaundice; according to the tradition that the salts of potassium act best upon the kidney, those of ammonium on the skin, and those of sodium on the liver.*

Carbonate of soda with or without taraxacum, and saline laxatives, to which many physicians add an occasional blue pill, is the traditional treatment for "simple" jaundice; but our ignorance of its pathology and the uncertainty of its duration make it difficult to measure the efficacy of these drugs. Dilute nitro-hydrochloric acid has a scarcely less established reputa-

* That it is possible to bring an attack of jaundice to an end I feel confident, and the following two cases go far to prove it. (1) A man aged fifty-nine came to me on January 9th, 1874, suffering from jaundice, which was not very deep, but had already been of two months' duration. There was some tenderness and fulness over the liver. I ordered him to take half a drachm of spiritus ammoniæ aromaticus in a mixture of rhubarb, soda, and calumba. He came again on the 16th, and said that for five days his motions had continued to be clay-coloured, but that on the 14th they began to contain bile, and that they were now quite dark-coloured. On testing his urine I found that it contained very much less bile-pigment than before. His jaundice was much diminished, and in the course of another week it entirely disappeared. (2) A bargeman came to me on February 23rd, 1872. Ten months before he had become suddenly jaundiced without pain. A month afterwards he had been seized with excruciating pain over the liver, lasting some hours, and he had since had three similar attacks. He had been under treatment both in Guy's Hospital and at King's College, but without result; he remained jaundiced the whole time. I ordered him to take ten grains of carbonate of soda, with a scruple of extract of taraxacum, and half a drachm of tincture of hop three times a day, and a grain of opium at night. On March 1st he came to me again, and assured me that he was very much better, having lost the pain and sickness. On March 4th he noticed that his motions resumed their natural appearance, and before long he was well.—C. H. F.

tion in cases of jaundice, and certainly patients may quickly lose their yellow colour while taking this medicine.

With regard to the general management of the disease, the patient need not be kept in bed, nor even within doors. He should be allowed to take moderate exercise, and to have his usual diet.

When the cause of the jaundice has been removed, the fading of the yellow colour of the skin may, according to Murchison, be hastened by warm baths, by giving the patient diuretics and diaphoretics, and by the administration of benzoic acid in four-grain doses three times a day.

With regard to the treatment of the various diseases which may give rise to *jaundice with pyrexia*, there is but little to be said: the only treatment is surgical (*infra*, p. 523).

When *permanent* jaundice has once declared itself, and the obstruction of the common duct is complete and irremediable, it is no longer advisable to prescribe carbonate of soda, or dilute nitro-hydrochloric acid, or taraxacum. The more faith we have in the efficacy of those remedies in simple jaundice, the more we shall fear that they may now do harm. Regulation of the diet is perhaps the most important part of the treatment. It has been shown experimentally by Bidder and Schmidt in Germany, and by Bennett and Rutherford in this country, that dogs with artificial biliary fistula may live for years provided they are supplied with and will take a sufficiently large quantity of food. At the same time its quality should be carefully attended to. In dogs whose common duct has been tied the daily quantity of fat that can be absorbed from the food is greatly diminished. Oleaginous diet should therefore be taken very sparingly by persons with permanent jaundice.

Something may be done to counteract the absence of bile in the intestines by the administration of the purified bile of the ox or pig. Murchison recommends that this should be given in doses of from three to six grains, about two hours after meals, in capsules or pills coated with a solution of tolu in ether, so that they may pass through the stomach unaltered. A glycerine extract of the pancreas may possibly do good in such cases.

Ox-gall has the further advantage of taking up the antiseptic function of the natural bile. With this object, as well as that of relieving the flatulence, creosote, thymol, or turpentine may likewise be prescribed with benefit. Occasional laxatives are generally required.

The *itching* caused by jaundice is sometimes so troublesome as to require special treatment. Warm alkaline baths have sometimes proved serviceable. Bicarbonate and bromide of potassium or of sodium, in full doses, are sometimes effectual. Belladonna internally and a hydrocyanic acid lotion externally are, in the writer's experience, the most often successful in giving relief. Opiates not infrequently aggravate the irritation. Dr Goodhart has found the injection of pilocarpine useful.

GALL-STONES.—One of the most frequent and important causes of jaundice is obstruction of the common bile-duct by calculi; they are probably always formed in the gall-bladder, not in the duct.

Gall-stones are of two kinds. Some consist almost entirely of bile-pigment, others mainly of cholesterine.

The former are small, dark reddish, olive-brown, or almost black in colour, irregular in outline, and so soft that on pressure they break down into a coarse gritty powder. They are small and multiple, often counted

by scores or hundreds. Baillie records more than 1000 taken from a single gall-bladder, and preserved in Dr William Hunter's museum.

The latter kind are firm and smooth on the surface, with a soapy feel. In size and colour they are very variable. Some are three and a half to four inches in circumference; a gall-stone of this size is generally single, and fills the whole gall-bladder, so that it has one rounded end answering to the fundus, and another tapering, which corresponds with the cystic duct. Others are small, the size of marbles, or peas, or scarcely larger than pins' heads. Several are often found in the same gall-bladder, and sometimes an enormous number; but this is not so frequent as with pigment calculi. Their surface is usually white or stone-coloured, and the superficial layers are also pale—often separated by a well-marked dark shell (seen as a band in section) from the deeper layers, which have generally a more or less deep-yellow or brown hue from bile-pigment.

On section cholesterine calculi are seen to be made up of concentric layers, which show radiating glistening lines, and break with a crystalline fracture.

The formation of the smaller pigment calculi seems to depend on the presence of a minute earthy concretion or a clot of blood or fragment of thick mucus, for the bilirubin to be deposited upon. The larger stones differ only in the presence of cholesterine, which crystallises out when the bile is, possibly from some toxic change in its chemistry, no longer capable of retaining this sparingly soluble secretion in solution.

Age and sex.—The liability to the formation of gall-stones increases with advancing years. They scarcely ever occur in children, and are rare under fifty years in men and under forty in women.

Women are much more liable to gall-stones than men, possibly because they more often lead sedentary lives. The proportion is about 4 to 1. Dr Brockbank found in 1347 autopsies at the Royal Infirmary, Manchester, gall-stones in 4 per cent. of the men, and in 15 per cent. of the women ('Edin. Med. Journ.,' July, 1898). He also noticed that patients suffering from chronic disease of the heart, and particularly from mitral stenosis, are very liable to gall-stones.

Symptoms.—Calculi are frequently latent, and are discovered in the dead bodies of those who during life had not been aware of their presence. In these cases, however, they are never found in the bile-ducts, but in the gall-bladder.

When the cystic duct is blocked by a calculus, the walls of the gall-bladder go on secreting mucus. It may then become distended and form a tumour in the abdomen below the liver, which may be mistaken for a pendulous hydatid cyst—for in such cases there is, of course, no jaundice.

When the calculus is impacted in the common bile-duct, severe symptoms follow, which are known as biliary colic, or a fit of the gall-stone. First comes sudden and agonising *pain*, sometimes so excruciating that the patient is bent double and rolls upon the floor, with loud cries. When the intensity of suffering abates, it is replaced by a constant dull aching, which continues until the more acute pain returns. The seat to which these agonising sensations are principally referred is the right hypochondrium, but generally they also shoot into the right scapular region and back, and downwards to the navel, or they spread over a large part of the abdomen.

Another marked symptom is *shivering*, with the face pale, the skin cool, and the whole body often covered with a cold sweat. The *temperature* is,

as a rule, normal. Sir Dyce Duckworth has, however, seen several cases of biliary colic with pyrexia; in one of them the temperature rose nearly to 104° , and remained high for several days. In these cases there is probably suppuration, or at least acute inflammation of the gall-bladder or duct. Murchison also mentions that pyrexia is not uncommon.

The *pulse* is much reduced in force and volume; sometimes it is slow, sometimes quickened, but generally of normal frequency. There is great exhaustion; the patient may swoon away, and it is said that fatal collapse has been known to set in.

Vomiting is a very frequent and characteristic symptom, and hiccough is not uncommon.

Jaundice is not one of the earliest symptoms of an "attack of gall-stones." It is evident that until the calculus has passed from the cystic into the common duct no jaundice will arise. Generally speaking, however, after a few hours, or at the longest a couple of days, the patient's urine contains bile-pigment, and a little later the eyes and the skin become yellow.

It is often supposed that an attack of biliary colic is invariably painful. But some years ago a case occurred at Guy's Hospital in which a man died of hernia who had previously had jaundice, which (it is expressly noted) was unattended with pain. The gall-bladder contained numerous gall-stones, and the common duct was dilated so as to admit the finger.

An attack of gall-stones may terminate in several different ways. Most commonly the jaundice sooner or later subsides; sometimes it passes off in three or four days, sometimes it lasts several months. Indeed, even the shorter period exceeds the limit within which an attack of gall-stones may occasionally run its course, for it may end in twenty-four or thirty-six hours; but in that case it is unattended with jaundice, which, as we have seen, seldom appears until the pain has lasted for some time. A good example of protracted jaundice from gall-stone is given by Murchison, that of a man who was jaundiced continuously for more than six months. Even in his case, however, the pain was not constant, but repeatedly went away for a week at a time. At last the jaundice disappeared, and he returned to work.

When an attack of jaundice from gall-stones subsides, it is usually because the calculus has passed into the duodenum. It is soon afterwards voided in the fæces, where it may sometimes be found without difficulty. It used to be said that if water is poured over a stool containing a gall-stone, the latter will rise to the surface, from being lighter than the liquid; but it is now known that this is a mistake. When first voided, gall-stones have a higher specific gravity than water; it is only when they have been dried that they float. The best way to detect a gall-stone in the fæces is to mix them with water and pass the whole through a sieve. In some cases, however, after the subsidence of an attack, no gall-stone can be found, in spite of careful search. Possibly it is retained for a time in the bowels, or it may possibly have undergone disintegration; or it may never have escaped into the duodenum, but have slipped back into the gall-bladder. When a patient has numerous attacks of biliary colic in quick succession, and when yet no calculus can be found in the evacuations, one naturally supposes that all the attacks are caused by a single gall-stone slipping to and fro in the duct. But it must not be forgotten that a large number of calculi are sometimes present in the same gall-bladder, and that many have been found in succession in the fæces.

Watson relates the case of a patient who collected fifty-five calculi from his stools within a space of five weeks. The discovery of the stone after an attack of jaundice is not only important as verifying the diagnosis, but it may also help to answer the question whether the complaint is likely to recur. If the gall-stone was alone in the gall-bladder its form is rounded; but if it was one of several, it will generally show flat facets where it touched the stones in contact with it.

Results.—As above stated, attacks of gall-stones have been reported so severe as to prove directly fatal. But it is doubtful whether, in any well-authenticated case, an autopsy has shown that death was really due to this cause, and not to concomitant inflammation of the ducts. Sometimes, although happily rarely, a gall-stone sets up ulceration, which reaches the peritoneal surface of the gall-bladder, allows bile to escape into the serous cavity, and causes fatal peritonitis.

Thus, some years ago a woman in Guy's Hospital, who for some years had repeated attacks of jaundice, for four or five weeks suffered continuously from this symptom and from pain in the abdomen, which became more severe until she died. At the autopsy acute peritonitis was discovered, which had been caused by the escape of bile through an ulcerated opening in the hepatic duct. The common bile-duct was obstructed by a gall-stone.

In other cases the ulceration of the gall-bladder or biliary passages caused by gall-stones sets up a local pyæmia, with secondary abscesses in the liver; and thus leads within two or three weeks to a fatal result.

Biliary colic frequently occurs over and over again, and sometimes, but not always, the first attack is the most severe. When a concretion of some size has once passed through the common duct into the duodenum, it is easier for another calculus of the same size to perform the same journey. Most of the cases in which biliary colic ends within a few hours, and even without jaundice, are those which have been preceded by several attacks of the same complaint. These repeatedly recurring attacks of biliary colic are often very trying to the patient; but in the majority of cases they sooner or later cease to return.

Sometimes, however, the jaundice persists until death. This termination may be due to an accidental complication; but the natural end of a case of impacted gall-stone with jaundice is suppuration, or perforation, or the gradual supervention of cholæmia, ending in coma.

In chronic cases the distended gall-bladder can be felt as a smooth, globular, painless tumour projecting below the edge of the enlarged liver. It is sometimes described as "pear-shaped," but this can scarcely be, for the narrow end is covered by the liver, and what is felt is more like an apple or a billiard ball.

It is very rare for death to occur by mere gradual wasting, as the result of obstruction of the common bile-duct by a calculus.

One such case was recorded by Murchison: the patient, who had for years been subject to attacks of gall-stones, died after six months of jaundice, having suffered during the last three weeks from greatly increased pain and vomiting, with hæmorrhage from mucous membranes; the common bile-duct was obstructed by a large cylindrical gall-stone, which was ulcerating into the bowel by the side of the orifice of the duct. Another case was observed by Dr Wale Hicks (afterwards Bishop of Bloemfontein); the patient died seven months after the attack began, but the jaundice, instead of being persistent, gradually faded, and at last entirely disappeared, and the hepatic tissue was found to have broken down into granular matter and oil; the obstruction in this case was not complete.

A remarkable and fatal complication of gall-stones is the development

of cancer of the gall-bladder or the bile-ducts. Frequently the clinical history seems to show that the jaundice was originally due to an attack of biliary colic, and that the development of the cancer was secondary; indeed, the following case seems to admit of no other interpretation.

A man aged forty-five died of jaundice which had lasted four months. Dr Moxon found that the gall-bladder was very large, containing hundreds of gall-stones; the common bile-duct at its commencement was greatly narrowed, and its walls were thickened by a cancerous growth; *below* the narrowed spot it contained three or four faceted gall-stones, just like those in the gall-bladder. This part of the duct was also dilated, and had evidently been accustomed to the passage of gall-stones before the cancer had begun to form.

In the great majority of such cases no gall-stone has been discovered impacted in the duct at the seat of the cancer; the concretions have been found in the gall-bladder itself, which has often been found contracted round them, and empty, or containing only a little purulent mucus. Thus it appears probable that if the malignant growth had not supervened, all the symptoms would have subsided, and the health of the patient would have been restored.

Probably cancer of the bile-ducts in association with gall-stones arises as the result of their irritation; whereas, when gall-stones are discovered in the bodies of those who have died from cancer of the breast or of other organs, this is probably due to the fact that both cancer and gall-stones are apt to occur in elderly persons.

Treatment of gall-stones.—In an attack of “biliary colic” the patient should first be placed in a hot bath; and afterwards fomentations or poultices should be applied to the abdomen. If there be much tenderness on pressure in the right hypochondrium, a few leeches often give great relief; but to alleviate the agonising pain we must, in most cases, administer opium or morphia, in full doses and frequently repeated. In a patient previously in good health two grains of opium are not too much to begin with, followed by a grain every two or three hours until ease or sleep is obtained, it being of course understood that the effects are carefully watched, and that he has no albuminuria. Very often the stomach is too irritable to retain the anodyne, and then the subcutaneous injection of a quarter of a grain of morphia at once will prove of signal benefit. Another valuable antispasmodic is belladonna, prescribed in full doses. The inhalation of chloroform has sometimes proved still more effectual, and is certainly the speediest way of relieving the pain when it is at its worst.

Prout used to recommend the administration of large draughts of hot water, containing one or two drachms of the bicarbonate of soda to the pint; and even when the stomach rejected the first portion of the fluid, he still persevered, believing that it diminished the severity of the retching. If the vomiting be very violent, it should be checked by dilute hydrocyanic acid. Many of the older physicians, and even Bright, prescribed antimony in the treatment of biliary colic, with the hope of relaxing spasm, and so facilitating the expulsion of the calculus; but this remedy is now justly discarded on account of its tendency to aggravate the vomiting. A mixture of turpentine and ether was once a famous remedy for biliary colic, but it is usually rejected by the stomach of modern patients.

There are no means at present known of *preventing* the formation of gall-stones, or dissolving them before they become impacted. Active exercise, spare diet, and occasional cholagogues are believed to be of some service, and the sulphate or phosphate of soda is often prescribed.

Chloroform is a chemical solvent of cholesterine and of bilirubin; hence it has been given when gall-stones were suspected; five or ten up to thirty drops may be taken in spirits and water ('Brit. Med. Journ.,' 1890, i, p. 50). Perhaps a better plan is to give chloral hydrate in doses of ten or fifteen grains. The succinate of the peroxide of iron has been recommended as a solvent, but without even theoretical probability of its being useful. Olive oil in half-ounce doses has often been prescribed, and some physicians believe that it has more than a laxative action.

It is a question whether the treatment of the effects of biliary colic by mercurials is wisely abandoned in the present day. Sir Walter Scott had a most severe attack of this kind, as the pain, vomiting, and jaundice prove, though it was treated as "cramp of the stomach," and writes, July, 1819, "No less a deity descended to my aid than the potent Mercury himself, in the shape of calomel, which I have been obliged to take daily, though in small quantities, for these two months past. Notwithstanding the inconveniences of this remedy, I thrive on it most marvellously, having recovered both sleep and appetite." (Lockhart's 'Life,' vol. vi, p. 113.)

Operative treatment (cholelithotomy) has of late years been sometimes followed by brilliant success, and is certainly justified in severe and inveterate cases when the patient's condition allows of probable success.

Suppuration of gall-bladder and biliary passages.—The catarrhal inflammation of the common bile-duct, which has been supposed to be the cause of "simple" jaundice, is, as we have seen (p. 508), a purely hypothetical affection. But we occasionally find the ducts in a state of suppuration, as the result of the presence of a gall-stone, and this may lead to ulceration of the mucous membrane, and to a cicatricial stricture.

Still more frequently the gall-bladder, when full of calculi, suppurates, and may burst into the peritoneum or the colon, or may open externally. In a case recently in the writer's knowledge, an elderly gentleman died from what could only be described as idiopathic suppuration of the gall-bladder with consequent portal pyæmia, for there had been no history of jaundice or biliary colic. When, as in such cases, ulceration and suppuration are not due to calculi, their pathology is quite unknown.

When the gall-bladder is distended by an impacted calculus, it occasionally becomes adherent to the abdominal parietes; an abscess may then be developed which points externally, and when it breaks or is opened by a surgeon, gall-stones are discharged along with the pus. The site of the external opening is not always directly over the gall-bladder; it may be at the umbilicus, or even in the left side of the abdomen; nay, a case has been recorded in which two biliary calculi made their way into the connective tissue of the vagina. It is important to note that in cases of this kind there are (or may be) no symptoms of the presence of gall-stones until they are found in the discharge. The common bile-duct is often quite free, while the cystic duct is completely closed; and thus neither is there any jaundice, nor does any bile enter the gall-bladder and mix with the pus. Hence the abscess is often supposed to be seated in the abdominal walls, or in the substance of the liver, and months or even years may pass before the real nature of the case declares itself. In the meantime the patient has a fistulous opening in the side, which, however, need not seriously affect his health; and when all the gall-stones have come away it may at length heal up.

The *treatment* of gall-stones and of a suppurating gall-bladder by operation has been followed in recent times by some remarkable successes. (See Dr Marion Sims' case, 'Brit. Med. Journ.,' June 8th, 1878, p. 811; and Mr

Lawson Tait's, 'Med.-Chir. Trans.,' 1880, and subsequently; also Mr Mayo Robson's, *ibid.*, 1890, and 'Clin. Trans.,' Oct. 25th, 1889; also Sir Spencer Wells' remarks in his "Bradshaw Lecture," 'Lancet,' Dec., 1890. Reference to earlier proposals of the operation, and isolated cases of its execution, will be found in the 'London Med. Recorder,' April 15th, 1881, p. 153.) During the last few years cholecystotomy has become a recognised operation, and it has undoubtedly saved many lives ('Allbutt's System,' vol. iv, p. 246).

Gall-stones in the intestines.—When a gall-stone escapes by ulceration into the intestine, it is, as a rule, safely voided *per rectum*; but since a concretion which takes this course is often very large, its passage through the anal orifice may be attended with severe pain and violent straining, the cause of which cannot be explained until the gall-stone is discovered. Probably when a large stone thus makes its way out of the body it has passed from the gall-bladder directly into the hepatic flexure of the colon; but a stone of moderate size may pass into the duodenum.

As mentioned in a previous chapter (p. 412), the gall-stone may become impacted in the jejunum or the ileum, and produce obstruction of the bowel. The recognition of this condition is, as we saw, difficult, and rests usually on probability derived from the age and sex of the patient and previous accounts of jaundice and pain.

The treatment is by opium, so as to relax the grip of the bowel on the gall-stone. When, after abdominal section for obstruction, the cause has been found to be an impacted calculus, it has sometimes been possible to manipulate it through the ileo-colic valve, and safely leave it there. In other cases the gut has been opened and the stones extracted with complete success. Crushing the calculus by means of padded forceps without wounding the intestine, and breaking it up by means of a needle, are methods which have been suggested, but not as yet carried out.

INFLAMMATORY DISEASES OF THE LIVER

“And let my liver rather heat with wine
Than my heart cool with mortifying groans.”
Merchant of Venice.

ACUTE SUPPURATIVE HEPATITIS—ABSCCESS OF THE LIVER—*Geographical distribution—Ætiology—Relation to dysenteric ulceration—Statistics—Morbidity—Anatomy—Characters of the pus—Direction of rupture—Symptoms—Physical signs—Diagnosis from acute perihepatitis—from abscess between the liver and diaphragm—from suppurating hydatid or gall-bladder—Prognosis—Treatment of acute hepatitis—Paracentesis of hepatic abscess.*

CHRONIC INTERSTITIAL HEPATITIS—CIRRHOSIS OF THE LIVER—*History—Anatomy—Ætiology—Non-alcoholic cirrhosis in children—Effects upon the portal circulation—Early symptoms—Hæmorrhage—Jaundice—Ascites—Cerebral symptoms—Hypertrophic cirrhosis—Prognosis and treatment. Perihepatitis—Hypertrophy of the liver—Simple chronic atrophy—Syphilitic hepatitis and gummata of the liver.*

ACUTE ATROPHIC HEPATITIS—ACUTE YELLOW ATROPHY OF THE LIVER—*History—Symptoms—jaundice—diminished percussion-dulness—the urine—cerebral symptoms—hæmorrhage—Course and event—Anatomy—Ætiology and pathology—Diagnosis—Prognosis—Statistics—Treatment.*

THE liver is not liable to ordinary “simple,” primary, or idiopathic inflammation as the result of irritation, like the bronchi and the pleura; nor to acute inflammation secondary to a definite preceding cause, as endocarditis to rheumatism. The substance of the liver, consisting of secreting epithelial cells (parenchyma) and connective tissue with blood-vessels and lymphatics (stroma), is subject to the following three forms of hepatitis, different in their pathology, and no less different in their clinical features:

1. Acute suppurative inflammation, septic in origin, producing either a single abscess or multiple pyæmic abscesses like those of septic lobular pneumonia. This we may compare with abscess of the brain.

2. Chronic interstitial inflammation, with subsequent fibrous change, contraction, and hardening, leading to atrophy of the glandular parenchyma—“cirrhosis,” a disease closely analogous in its pathology to sclerosis of the spinal cord, to chronic interstitial pneumonia, and to the most chronic form of Bright’s disease. It is from this analogy that we often speak of cirrhosis of the lung and cirrhosis of the kidneys, while some pathologists name the corresponding hepatic disease, not cirrhosis, but sclerosis of the liver.

3. There remains the most rare, most obscure, and most fatal disease

of all that affect the liver—an acute affection, which leads to rapid atrophy, and is wanting in many of the characteristics of inflammation. It is open to question whether it should be regarded as hepatitis at all. But it appears to bear some analogy to acute pneumonia on the one hand and to acute Bright's disease on the other; and unless we admit it to be a parenchymatous inflammation, peculiar to the liver as pneumonia is to the lung, it is altogether unique, in pathology no less than in its clinical aspect, and we must either treat of it apart, or name it by one of its most striking features, Icterus gravis, and group it (as was done in the first edition of this work) with jaundice from obstruction. On the whole it appears best to place it near hepatic abscess and cirrhosis; but the arrangement is mainly one of convenience, and each of the three sections of the present chapter will be treated independently.

ACUTE SUPPURATIVE HEPATITIS—ABSCESS OF THE LIVER.—In the liver, as in the other solid viscera (kidneys, spleen, lungs, testes, brain), ordinary inflammation does not end in suppuration. With the important but still doubtful exception of tropical abscess, hepatic suppuration only occurs as the result of septic, bacterial, infective inflammation.

Abscesses of the liver are frequent in cases of pyæmia, general or portal, and in the latter form pus fills all the portal canals.

Tuberculous and typhoid ulceration of the bowels do not produce hepatic pyæmic abscesses, while dysentery does.

Primary hepatic abscess in India, China, and other hot climates is very common, and indeed takes an important place in the European death-rate. It is therefore not without reason called tropical abscess of the liver. In the West Indies it is said to be comparatively rare.

In England, apart from pyæmia, abscess of the liver is very seldom met with, and some writers have stated that it is never seen except in those who have lived in a hot climate. This statement, however, is too exclusive. Dr Fagge collected in Guy's Hospital (1860—69) fifteen cases in which death was caused by the formation of a single large abscess (or in one instance two large abscesses) in the liver. Five of these cases occurred in patients who had come from China, or India, or the West Coast of Africa; but in ten there was no such history, and several patients positively stated that they had never been out of England.

Dr H. J. Campbell extracted for the third edition of this work sixty-nine cases of hepatic suppuration from the *post-mortem* records of Guy's Hospital during the twenty years 1870—1889. Forty-three of the patients were men and twenty-six women; the ages varied from childhood to old age. In forty-six the abscesses were multiple and in twenty-three single.

The causes were very similar. There was no instance of an idiopathic tropical abscess. General, surgical or internal, pyæmia accounted for fourteen multiple and three single abscesses. Portal pyæmia from ulceration, in several cases dysenteric, accounted for eleven cases of multiple and eleven of single abscess. In some of these cases the dysentery had been contracted in the tropics. Pyæmia from the female pelvic organs produced four cases of multiple and one of single abscess, and prostatic pyæmia one case of single abscess. A suppurating hydatid cyst furnished seven cases; injury to the ribs one. Extension of inflammation from a perforating ulcer of the stomach or duodenum, or from suppuration around a cancerous growth, accounted for seven cases. Suppuration of the bile-ducts, usually from an

impacted calculus, is a frequent cause of multiple hepatic abscesses, and led to six cases of this series. Lastly, one case was due to actinomycosis.

The following is the complete tabular statement :

	Single.	Multiple.	Total.
Injury to ribs	1 .	0 .	1
Extension from a neighbouring organ	2 .	5 .	7
Suppurating hydatid	4 .	3 .	7
Suppuration of bile-ducts	0 .	6 .	6
General pyæmia	3 .	16 .	19
Intestinal pyæmia	11 .	11 .	22
Pelvic pyæmia	2 .	4 .	6
Actinomycosis	0 .	1 .	1
	—	—	—
	23	46	69

All the above sixty-nine cases were fatal, but several have occurred in which the abscess has been opened with a good result.

The following twenty cases, which have been under the writer's care during the last ten years, 1890—1900, are not included in the above list.

Three were tropical; one traumatic; several from general or portal pyæmia (mostly dysenteric or pelvic in origin); one from perforation of a gastric ulcer; one, beside a second among the sixty-nine, was due to inflammation of the appendix; and one to a basal vomica in the lung perforating the diaphragm. Two others turned out to be suppurating hydatid cysts, and one to be due to actinomycosis.

It has long been known that tropical abscess in the liver is often associated with dysentery, and various opinions have been held as to the connection between these two diseases. Annesley supposed that sometimes, as the result of an hepatic abscess, the bile acquires peculiarly irritating properties, and thus sets up inflammation and ulceration of the intestine. Dr George Budd in 1842 first taught that dysentery is the earlier of the two diseases, and that abscess of the liver is the result of absorption from one of the intestinal ulcers; in other words, that the pathology of the so-called tropical or single abscess of the liver is essentially the same as that of the multiple abscesses of portal pyæmia. Dr Budd's view for some time received general acceptance, but it was afterwards rejected by many writers of experience in tropical diseases.

It is argued that if dysenteric ulcers in the colon were the cause of abscess of the liver, the same result ought to follow other forms of intestinal ulceration, such as occur in phthisis or in enteric fever.

Another argument is that many cases of hepatic abscess, in which recovery takes place, are not preceded by symptoms of dysentery. It is, however, possible for dysentery to begin insidiously, or to remain altogether latent (cf. p. 396). In England this is often the case, and it probably is so in India likewise.

Again, it is said that abscess of the liver does not occur in some epidemics of dysentery, and is comparatively uncommon in certain countries where dysentery prevails—in China, for instance, as compared with India.*

But the strongest argument that abscess of the liver is not secondary to dysentery, is the fact that many cases have been recorded in which, after death from tropical abscess, the intestines have been found to present no sign of past or present inflammation. Murchison met with a case of this

* That Baly did not meet with it in the epidemic of dysentery at Millbank (p. 392) may perhaps be explained by a certain length of time being required for the development of suppuration in the liver, which thus may be wanting in rapidly fatal epidemics.—C. H. F.

kind in a European soldier in Burmah. The man had never had dysentery, although while under observation he was suffering from persistent diarrhoea. He died, and an enormous abscess, holding four quarts of pus, was found in the liver, but neither the small nor the large intestines nor the stomach presented any cicatrices or trace of recent ulceration. Again, Waring collected 204 cases of abscess of the liver, in exactly one fourth of which the intestine is said to have been perfectly healthy; and Morehead mentions that he has notes of twenty-one similar cases.

Of Fagge's fifteen fatal cases of large abscess of the liver at Guy's Hospital there were three in which no sign of ulceration was found in the intestines; and one of these was a tropical case. In eleven others it is expressly stated that the bowels were or had been diseased. In one case which came under Dr Moxon's observation there was only a cicatrix in the gut, so small that it might easily have been overlooked ('Path. Trans.,' vol. xxiv). Among Campbell's sixty-nine cases, there was disease of the bowels in twenty-two.

In this country dysentery and hepatic abscess are each so rare that their frequent association would be most extraordinary unless they are more than casually connected; and it is the rule that the ætiology of a disease can best be studied in countries where it is not endemic. Even in Murchison's cases dysentery was present in three fourths.

In India the current opinion seemed recently to be that dysentery and abscess of the liver are common results of the same causes; or that inflammation extends from the mucous membrane of the intestine to the liver as it does from the urethra to the testes in cases of gonorrhœa. But if so, why does it not also cause abscess of the pancreas? Moreover the duodenum is seldom, if ever, the seat of dysenteric ulceration.

The connection between dysentery and hepatic abscess is far more probably by the veins of the portal system.

Within the last few years the reality of this channel of infection has been proved, and the conveyers of infection have been identified as leucocytes in the blood of the mesenteric vein.

After the researches of Laveran on the pathology of Malaria had been confirmed by other observers, it was soon ascertained that in many cases of malarial dysentery, the characteristic amœbic parasites can be found in the intestinal ulcers, whence the term "amœbic dysentery" (Councilman, Stockton, Laffeur, Osler). And next it was found that they are usually, though not constantly, present in the pus-corpuscles of tropical abscess. (See Dr Windsor's article in the 'Lancet,' Dec. 11th, 1897.) The exceptional cases in which these Protozoa are absent are probably due to streptococcic infection from other sources; but it seems to be well ascertained that the largest and most typical solitary abscesses of the liver in India and elsewhere are due to the presence of these malarial amœbæ, and therefore of the same essential pathology as tropical dysentery.

Apart from dysenteric ulceration and pyæmia, no other cause of hepatic abscess can be assigned. It appears from the statistics of the European General Hospital in Bombay that the admissions of patients with hepatitis are more numerous during the months which follow the cold season, and during the cold season itself, than during the hot months. Heat, therefore, is apparently not even a predisposing cause. Nor is there reason to suppose that intemperance has anything to do with the disease.

Local injury has sometimes been supposed to set up suppuration in the

liver. A case in point occurred at Guy's Hospital in 1876. A drunken woman was run over, but the ribs were not fractured; she died with symptoms of pneumonia of the right base, and after death there was found in addition a large abscess in the right lobe of the liver, which had not perforated the diaphragm. A similar case under the writer's care was that of a little boy who in like manner had his ribs injured but not broken.

In a remarkable case, which happened in 1881, an abscess in the liver was caused by the perforation of a large phthisical vomica in the base of the right lung, which opened into the liver.

Excluding cases of general pyæmia, suppurating hydatid cysts, and tropical cases in soldiers and sailors from abroad—hepatic abscesses in Guy's Hospital have been, with few exceptions, due to portal pyæmia; from dysentery, ulcerative colitis, fistula *in ano*, gastric ulcer, or typhlitis.

In a case which occurred under the writer's care in the same year, two acute abscesses of the liver in a boy of ten were found after death to have been caused by a pin lodged in the appendix cæci; a similar example was recorded by Dr Payne in the 'Pathological Transactions' for 1870 (vol. xxi. p. 232).

Anatomy.—Rindfleisch distinguished a "thrombotic" from an "embolic" origin of hepatic abscess. In the former the inflammation occupies the walls of the interlobular branches of the portal vein. These, and the sheath of connective tissue round them, are swollen by an infiltration of leucocytes until the columns of hepatic cells become compressed and perish. The adjacent infiltrated foci come into contact, and form small white nodules about the size of lobules. As they melt away they form an abscess cavity, while fresh suppurative foci are constantly making their appearance at its periphery, and give a ragged character to its inner surface. In the "embolic" variety the portion of hepatic tissue which corresponds to the distribution of the plugged vessel becomes intensely congested, the circulation in it is entirely arrested, and it sloughs *en masse*. The lobules round it become enlarged, and those nearest the sloughing part are permeated by numerous pus-corpuscles, which lie between the secreting cells and the capillaries. The hepatic cells appear to take no active share in the formation of pus, which begins in the connective tissue.*

In most cases of tropical abscess, and in multiple suppuration after dysentery, the amoeboid organisms above mentioned are found present in the blood and pus. In cases of abscess secondary to portal suppuration and infective thrombosis or embolism, there are present, as a rule, streptococci, and occasionally staphylococci.

Multiple abscesses of recent formation and rapid growth often have no limiting membrane whatever, the pus lying in an irregular cavity of softened hepatic tissue. If of somewhat longer standing, they are lined with a layer of opaque yellowish material, the formation of which has been described above. Very old abscesses have a thick fibrous wall, which may be so hard as to feel like cartilage, or it may even be partly calcified.

The pus of an hepatic abscess is, as a rule, yellowish and free from odour. But we have sometimes found it greenish, gelatinous, or curdy, and sometimes reddish or reddish brown in colour. In a case of hepatic abscess that came under Dr Fagge's care in Guy's Hospital, the fluid removed by the trocar was of a brick-dust colour, looking not unlike anchovy sauce; and it had a most peculiar and nauseous odour.

* The observations on which these statements rest appear to have been made in cases of the small multiple abscesses of pyæmia.

An abscess of the liver may sometimes attain an extraordinary size. The largest we have recorded held six pints; Murchison's, above quoted, a gallon; but Maclean mentions one which contained altogether nineteen pints.

When the pus has reached the surface of the liver, it may point externally in the right hypochondriac or epigastric region; or it may rupture into the peritoneum; or it may discharge itself into the stomach, duodenum, or colon. In these last events the patient may vomit a considerable quantity of matter, or pass it *per rectum*, but in many cases the pus cannot be traced. Sometimes, again, it burrows towards the lumbar region, or it may perforate the diaphragm into the pericardium or the pleura, or if adhesions have previously formed, into the lung, and be evacuated through the trachea.*

Symptoms.—In many cases abscess of the liver remains entirely *latent*, and is found after death in the bodies of those who have never been known to suffer from any symptoms of the disease. Cases of this kind are mentioned by all writers on tropical diseases, for it is chiefly in persons who have been in hot countries that latent abscess of the liver has been met with. Dr Fagge once examined the body of a gentleman who died of protracted diarrhoea a few years after his return from China. There was an abscess the size of a walnut in the back of the liver, but careful inquiry during the patient's life had failed to elicit any evidence of it.

These latent abscesses of the liver may suddenly give rise to the most serious symptoms. Maclean mentions the case (occurring in the Mauritius) of a man, apparently in good health, who had walked seven or eight miles in search of employment, when he complained of pain at the pit of the stomach, and in a few hours died; an abscess of the liver, lined with a dense fibrous membrane, had burst into the pericardium. Another case is that of a man who had been invalided from India on account of "chronic hepatitis," but who when he arrived at Fort Pitt had apparently recovered so completely that he was sent to the dépôt for duty. Some weeks afterwards, while he was straining at stool, he felt "something give way," and died very shortly afterwards. An hepatic abscess, as in the last case, had ruptured into the pericardium.

In most cases, however, abscess of the liver gives rise to symptoms which are strongly indicative of its presence; and the results of physical examination of the abdomen are often conclusive. The symptoms are a sense of fulness and weight in the right hypochondrium, pain there or in the right shoulder, inability to lie on the right side, fever, rigors, and cough.

The *pain* which attends abscess of the liver is exceedingly variable in degree. It is often more intense in the right shoulder than in the hypochondriac region. Budd mentions a case in which the pain in the shoulder was for a long time most severe; and when the abscess was opened the pain was relieved. The pain over the liver itself is often increased by pressure, and also by the patient drawing a deep breath, or turning over on to the right side.

In cases of hepatic abscess *cough* is often present. It is generally short and dry, and is no doubt due to reflex irritation.

Pyrexia is usually a marked symptom. Maclean found that in every

* Dr Morehead saw three cases in which a patient suffering from hepatic abscess expectorated pus, and yet no perforation could be discovered after death.

case of suppuration of the liver at Netley the thermometer showed a rise of from one to three degrees. In the case above mentioned, of hepatic abscess from a pin in the appendix cæci, the temperature rose to 106° ; and this, with the great increase of hepatic dulness, local tenderness, and absence of other causes of fever, led to the diagnosis of suppuration of the liver.

Morehead found from his experience in India that *rigors* are not of much value as indicative of suppuration of the liver; they may be present when there is no abscess; and, on the other hand, they are often absent when an abscess is forming.

Jaundice appears not to be a symptom of single tropical abscess, unless by a rare accident, from pressure upon one of the main ducts. With multiple pyæmic abscesses it is very frequent (cf. p. 522).*

The *physical signs* of hepatic abscess depend mainly upon its position and its size. Even a deep-seated abscess often indicates its presence by producing a peculiar rigidity of the upper part of the rectus abdominis muscle on the right side; but though this is an important sign of disease in that region, it does not point especially to hepatic abscess. Enlargement of the liver is of much diagnostic value, particularly if the outlines of the organ are altered, if there is bulging in one particular direction, and if any spot can be detected which is soft and fluctuating. Where an enlargement of the liver can be clearly made out, there is generally much tenderness on pressure in the hepatic region; and round any fluctuating point there is almost always inflammatory œdema of the abdominal parietes.

These last decided indications of hepatic abscess are of course absent when the back part of the liver is the seat of the disease. Even then, however, there may be an increase in the area of hepatic dulness, which, if other symptoms be present, may make the case sufficiently clear.

Diagnosis.—That the detection of hepatic abscess is often difficult may be inferred from the statements already made.

According to Maclean, there is another affection of the liver which is common in India, and apt to be mistaken for suppurative hepatitis, viz. acute inflammation of the capsule of the liver, or *acute perihepatitis*. The symptoms of this disease resemble those of pleurisy. The pain is sharper than in cases of abscess of the liver, and it is more decidedly aggravated by pressure or by a full inspiration, or by movement of the patient's body. Acute perihepatitis is probably not uncommon in this country, for the liver is often found after death to be fixed to the diaphragm by adhesions, which appear to correspond to an acute rather than a chronic inflammation, and sometimes a peritoneal rub may be heard over the surface of an enlarged liver. Dr Cantlie has found the same condition in China, an acute perihepatitis arising from chill like pleurisy ('Clin. Journ.,' June 22nd, 1898, p. 163).

Even where there are distinct signs of the existence of an abscess in the right hypochondrium, this is not always seated in the liver. In the 'Guy's Hospital Reports' for 1874, Dr. Fagge published six cases of a collection of pus in the *right hypochondrium* between the upper surface of the liver and the diaphragm. This affection is most frequently caused by direct injury to the part, or by extension of inflammation from disease of one of the

* See on this and other symptoms of multiple hepatic abscesses the late Dr Carrington's account of thirteen cases of multiple small abscesses of the liver ('Guy's Hosp. Rep.,' 1883).

other abdominal viscera. In these respects it differs from abscess of the liver itself, but clinically it is scarcely distinguishable by physical examination. Such a hypophrenic or subdiaphragmatic abscess can be more readily cured by evacuation of its contents than one embedded in the liver (*cf. supra*, p. 483).

Again, a *suppurating hydatid* of the liver may easily be mistaken for simple abscess if the case be first seen when inflammation has already been set up within the capsule of the cyst, and if the existence of a painless tumour previously should have escaped the observation of the patient. Indeed, in such cases it is rarely possible to determine the nature of the disease until the contents of the abscess are discharged and submitted to microscopical examination. The treatment is the same as for a single large hepatic abscess, and the result is more often successful.

Lastly, it is said that a *suppurating gall-bladder* has been mistaken for an abscess situated in the right lobe of the liver (*cf. p. 522*).

Prognosis and treatment.—Writers of experience in India believe that in the early stage of the disease ipecacuanha is a potent remedy. Maclean says that this drug is nearly as efficacious in suppuration of the liver as in tropical dysentery, and that it should be given in the same manner, viz. in doses of from fifteen to twenty grains repeated at intervals of five, six, or eight hours. Antimony is also used by some surgeons in India, either in addition to the ipecacuanha or separately. They relieve pain and give sleep by the hypodermic injection of morphia; and fomentations or poultices are kept applied to the hepatic region.

It appears to be established that under such treatment patients often quickly recover after having had symptoms exactly like those which are recognised as the early symptoms of hepatic abscess; but can one be certain that suppuration would have occurred in these cases if left to themselves?

When there are grounds for believing that an abscess has actually been formed in the liver, the only question is when and how the pus should be evacuated by the surgeon. No doubt hepatic abscesses in rare cases subside spontaneously, for in making *post-mortem* examinations dried-up abscesses have sometimes been found with cheesy contents. This, however, occurs far less often than was formerly supposed; for not only have dead hydatids been mistaken for cured abscesses, but the same view was taken of syphilitic growths in the liver before their real nature was understood. Certainly the spontaneous subsidence of an hepatic abscess cannot be counted on as at all likely.

Cases in which an hepatic abscess has discharged its contents spontaneously not infrequently end in recovery, the cavity after a time closing and pus ceasing to be formed. It would appear that the prospect of such a termination has hitherto been greatest in those cases in which the abscess has made its way through the lung. Stovell is said to have reported nine recoveries out of sixteen cases of this kind; nor is recovery infrequent when an abscess discharges into the stomach or intestine. Morehead had five cases which took this course, and three of them ended in recovery. Rupture into the peritoneum or pericardium is quickly fatal; but fortunately abscess of the liver seldom takes either of these directions.

When an abscess points towards the exterior of the body, and presently breaks and discharges its contents through the skin, the patient sometimes recovers. Maclean confirms Morehead in the statement that this is much

more likely to occur when the point at which the abscess reaches the surface is near the ensiform cartilage than when it is in the right hypochondrium or in an intercostal space. This distinction apparently depends upon the collection of pus being in the former case seated in the left lobe of the liver, and therefore smaller.

The whole aspect of treatment has been changed—first, by the introduction of the aspirator of Dieulafoy; and subsequently by the antiseptic and aseptic method in abdominal surgery.

No better illustration of the advantage of treating cases of hepatic abscess with the aspirator could be given than the following case, which is reported in the 'Medical Times and Gazette' for April, 1874, from the Madras Hospital.

An Englishman was admitted with a tender swelling extending from the hepatic region downwards to the level of the umbilicus. He had daily accessions of fever, and other symptoms indicative of deep-seated suppuration. As there was fluctuation in the centre of the swelling, this was tapped with the aspirator, and four ounces of pus were withdrawn. The relief, however, was but partial; and when the operation was twice repeated, only a small quantity of matter came away each time. The patient's condition kept getting worse. The existence of a second abscess was therefore suspected; and as there seemed to be a slight bulging of the right lower ribs, it was determined to make an exploratory puncture in this position. The needle of the aspirator was introduced, and the syringe was soon seen to fill with pus. Forty ounces were withdrawn, and in a few weeks the patient left the hospital well.

Such a case as this contrasts strikingly with those which are given by the older writers on tropical diseases as showing the danger of opening hepatic abscesses.

With antiseptic precautions and better means of diagnosis, surgeons in India and China now deal boldly and successfully with hepatic abscesses. Usually, after once tapping, an incision is made and a drainage-tube inserted. Some surgeons provide for safe adhesions being formed by previously stitching the liver to the abdominal walls. If the aspirator should not be at hand, an ordinary trocar might be used, provided that asepsis was secured; for it is now well established that the introduction of an instrument into the substance of the liver is not of itself attended with any risk.

There is, however, a practical objection to puncturing an abscess without an aspirator, for the pus may refuse to flow through the cannula.

This occurred in a case under Dr Fagge in 1875; and it was not until suction was made that it began to escape. Eleven ounces of viscid reddish pus were then drawn off; the man had not the slightest elevation of temperature afterwards, and was kept in bed only as a matter of precaution. The tumour altogether disappeared. He had been in foreign service as a mariner off the Indian and West African coasts; but he had bought his discharge four years before he came into the hospital, and since then had been working as a carman. He said that he had suffered severely from diarrhœa, but had not had dysentery.

In India the practice of thrusting trocars into the liver is of ancient origin, and even when no pus is thus obtained it appears seldom to be dangerous and often beneficial—possibly by relieving congestion and preventing the formation of an abscess. Of this the following case is an example.

In a patient under the late Dr Ralfe, at the Dreadnought Hospital ('Lancet,' ii, 1876), hepatitis came on in the course of an attack of dysentery. On attempting to tap a presumed abscess, only a small quantity of blood flowed into the aspirator; but the patient declared that he felt in-

stantly relieved, and did in fact experience no more pain in the right hypochondrium, where it was before severe. His temperature, which had ranged from 99.4° to 102.2° , fell in two days to normal, and he made a good recovery.*

CHRONIC INTERSTITIAL HEPATITIS, OR CIRRHOSIS OF THE LIVER.—In strong contrast to the acute suppuration of the liver just described, there is an insidious and very chronic process of hepatic inflammation, which is unattended by fever, and never leads to suppuration. It ends in the production of fibrous tissue, which shrinks and thus strangles the secreting parenchyma. The condition in question is that known as *cirrhosis*, or more familiarly as “gin-drinker’s” or “hobnailed” liver.

The earliest representation of a liver in this condition was discovered in the ‘Philosophical Transactions’ for 1685 by Dr Payne; here the hobnail character is quite obvious. He also quotes an excellent clinical account of the case of a drunken German nobleman who died of ascites and jaundice, with a hard liver, epistaxis, and thrombosis of the portal vein, admirably recorded in ‘Bonet’s Sepulcretum,’ 1679. (See Dr Payne’s interesting paper and figure in the ‘Pathological Transactions’ for 1889.)

Matthew Baillie gave an excellent account of the anatomy of cirrhosis in his ‘Morbidity Anatomy,’ 1793—1818 (p. 228 of 5th ed.), under the title “Common Tubercle of the Liver.” He notes that it is hardly ever met with in very young persons, that it is more common in men than in women, and that it is most apt to occur in those who drink spirituous liquors. The “tubercles” (or “hobnails”) which give its characteristic appearance, when cut into, consist of a brownish or yellowish-white solid matter. The liver thus “tuberculated” feels hard, but “is generally not larger than in a healthy state, and (I think) it is often smaller.” The liver is of a yellow colour, arising from bile accumulated in its substance, and there is also water in the cavity of the abdomen. He adds that this condition is generally called a scirrhus liver (*i. e. hepar durum*), but says it is unlike scirrhus, and considers it a peculiar disease.

Laennec, in 1819, gave the name “cirrhosis,” and described the cirrhotic liver as *ratatiné* (shrivelled) *et offrait un grand nombre de très petites bosselures*. On section he describes these little nodules as pale yellow, separated by the ordinary parenchyma of the organ, and regarded them as *petits corps étrangers* (‘Ausc. Med.,’ tom. ii, p. 61).

Andral, in 1829, corrected Laennec’s mistake, and showed that these *granulations remarquables par leur couleur, assez semblable à celle d’une cire un peu jaune*, were nothing but liver substance. He quotes Bouillaud who shortly before had described them as hypertrophied acini (*i. e. lobules*); but he had no clear notion of what the intermediate substance was, and repeated what Laennec, Bichat, and other French pathologists had said of the atrophy of the cirrhotic liver.

Rokitansky explained the inflammatory nature of the interstitial substance and its gradual contraction (1842-51, 3rd ed., ii, p. 256), and described *Cirrhosis hepatis* as *granulirte Leber*.

Todd, in 1857, remarked that small cirrhotic livers are usually accompanied by ascites and large ones by jaundice.

Anatomy.—A well-marked cirrhotic liver presents very striking charac-

* See the papers by Dr Harley and Messrs Willett, Marsh, Thornton, and Lawson Tait in the ‘Brit. Med. Journ.’ for Nov. 13th, 1886.

ters. Instead of being red, it is pale, and mottled with grey and yellow. It is exceedingly tough, so as to resist the pressure of the finger. Its external surface is not smooth and even, as in health, but presents numberless round elevations of all sizes, from pins' heads up to peas, or even larger. On section similar islets are found closely packed throughout its substance. They are often of a bright yellow colour, and on this account Laennec invented the name of *cirrhosis* for the disease (*κίρρōς*, yellow).

The microscope shows that these "hob-nails" consist of liver substance, with lobules and epithelial cells, while the material which is really morbid is that which lies around and between the yellow islets. This is a greyish, somewhat translucent substance, and under the microscope is found to consist of fibrillated tissue, with nuclei, and more or less numerous leucocytes. If they make up the tissue without any fully formed fibres, this embryonic condition marks the earliest stage of the disease, when death rarely occurs. In most cases we find well-developed fibrous tissue, with small aggregations of corpuscles; and the presence of these exudation-cells is a proof that the disease was still advancing at the time of death.

The question has often been raised whether the primary process is, as generally believed, an interstitial inflammation, followed by secondary destruction of the secreting cells of the lobules, or a primary toxic atrophy of the epithelial cells to which the fibrous overgrowth is secondary. The same question has been discussed concerning the allied pathological conditions that result in the small contracted kidney, in cirrhosis of the lung, and in grey degeneration or sclerosis of the cord. In the case of the liver the writer believes the balance of evidence to be in favour of the atrophy being secondary, and the interstitial inflammation and cicatrization primary.

It is the fibrous material which gives the cirrhotic liver its peculiar characters. The "embryonic" or granulation tissue lies between the hepatic lobules, around the terminal branches of the portal vein. As it undergoes development, its separate portions coalesce, so as gradually to form fibrous rings, surrounding groups of hepatic lobules. But (like all young connective tissue) the new material has a strong tendency to contract, and thus compresses the secreting cells contained in the lobules. Some of them undergo absorption, and thus extensive tracts of a whitish-grey colour are formed, consisting of fibrous tissue with only a few isolated groups of hepatic cells. Other lobules, again, become squeezed up into the rounded yellow granules or nodules above described. The reality of the compression is evident from the fact that, when a section is made through a cirrhotic liver, the yellow nodules at once rise and project above the cut surface. The secreting cells no longer lie in definite columns, but are disarranged, so that it is impossible to determine how many original lobules each mass contains; but there are usually more than one.

Distinction has been made by some pathologists between unilobular or biliary, and multilobular or portal, cirrhosis, the latter being an interstitial inflammation which begins and runs its course in the "portal canals" between the lobules as above described; and the former, a precisely similar inflammation (exudation of serum, and leucocytes, ending in formation of fibrous tissue) which begins within the lobules among the secreting epithelial cells, and transforms irregular patches of the liver into fibrous tissue. The distinction, however, is not an absolute one: well-marked extremes may be readily found of the regularly "hob-nailed" liver and of the diffuse

fibrous change, but all gradations are to be found between them, and the process is essentially the same (p. 540).

The condition of the blood-vessels in a cirrhotic liver is very remarkable. If the hepatic artery be injected, one finds that the new fibrous tissue, which looks quite bloodless, is abundantly supplied with vessels from this source. But the branches of the portal vein often appear to be almost obliterated. Rindfleisch says that in one case he found it impossible to force an injection beyond the three or four main divisions of its trunk. He therefore supposes that the bile is secreted from arterial blood.

As a rule, the hepatic ducts are but little obstructed in cirrhosis. But isolated nodules are often found to be of a dark yellow or green colour, which is evidently due to local jaundice, caused by obstruction of the corresponding ducts.

Size of the cirrhotic liver.—In cases of cirrhosis, it is only occasionally during life that the dulness of the liver on percussion is diminished. As a rule it is increased, and the liver can be felt below the ribs. But this does not always mean that the liver is enlarged; and after death it is often found much contracted. Particularly in advanced cases the liver is often much smaller than natural, and often it is greatly reduced in weight. The records of Guy's Hospital contain several instances of its weighing as little as thirty-two or thirty-four ounces, and cases have been recorded in which it has been still smaller. But often a cirrhotic liver is found after death to be above the natural size. When considerable enlargement is present, the organ is almost always loaded with fat; thus Dr Fagge met with one remarkable instance in which a liver, which was of gristly hardness, weighed nine pounds; and it contained so much fat that it actually floated in water.

Dr Price, now of Reading, collected the cases of cirrhosis from our *post-mortem* records from 1875 to 1883 inclusive ('Guy's Hosp. Rep.,' xlii, p. 295). They were 142 in number—108 men to 34 women. There was admitted intemperance in half the cases. In only 9 was there evidence of syphilis, and in 6 of these there was also evidence of drink. The weight of the liver was less than fifty ounces in 29 cases only, and in two of these the patients were below adult age (16 and 14). Excluding accidental causes of enlargement, there were 63 cases above sixty ounces. In 33 cases the weight varied between fifty and sixty ounces. Ascites was present in 58 out of 72 cases, and more often when the liver was below than when it was above the normal weight. Jaundice was present in 30 out of the 72, and more often when the liver was above than when it was below the normal weight. When death was preceded by coma, jaundice was almost invariably present. Granular kidneys were more often found in association with hypertrophic than with atrophic cirrhosis.

Dr Pitt's subsequent observations continue and confirm those of Dr Price ('Path. Trans.,' 1889, p. 348). He found that of 89 cirrhotic livers with a marked alcoholic history, 28 weighed under fifty ounces, 18 between fifty and sixty, and 43 over sixty.

It is a separate question whether a minor degree of enlargement of the liver constantly occurs at an early stage of cirrhosis. That this should be the case is quite consistent with the pathology of the disease; for it begins as a subacute hepatitis with exudation of leucocytes, and atrophy of the lobules only comes on later. Bright long ago stated that he had noticed the enlargement of the liver when cirrhosis was beginning, and had traced

its gradual diminution in the later stages. But the physician has rarely an opportunity of observing this; and it has not yet been proved that an appreciable enlargement of the liver is constantly present at first. In any case this early inflammatory or hyperæmic condition is very different from the late enlargement due to increase of fat and fibrous tissue.

Hypertrophic cirrhosis as a supposed separate disease will be discussed below (p. 540).

Etiology.—The main cause of cirrhosis of the liver is the abuse of alcoholic liquors. The owner of the first cirrhotic liver of which a drawing remains was a life-guardsmen in 1685 (cf. p. 533), and though his death was ascribed to drinking too much water, we may assume that the water was not the pure element. In Hogarth's engraving, an occupant of Gin Lane is seen with the swollen belly of ascites,—indeed, the terms "hob-nailed liver" and "gin-drinker's liver" have long been accepted in this country as synonymous. Dr Dickinson found that cirrhosis was present in 22 out of 149 persons whose trade it had been to make or sell or carry stimulants, and who died in St George's Hospital; while it occurred in only 8 out of 149 other persons who had been unconnected with the liquor traffic, and who therefore may be presumed to have included a smaller number of intemperate persons. It has been objected that the disease is sometimes seen in children; but many observations appear to show that this may really be an argument in favour, rather than against, its alcoholic origin. Dr Wilks some time since had a little girl, eight years old, under his care at Guy's Hospital suffering from what proved to be a very small hob-nailed liver; she had been fed on gin, and had taken as much as half a pint daily. Niemeyer quotes from Wunderlich two very similar cases which occurred in sisters, aged eleven and twelve years respectively, who had each drunk spirits to excess. Dr Taylor's case in a boy who died at eight, was also alcoholic in origin ('Path. Trans.,' 1880, p. 119).

Gin and whisky are believed to be far more efficient in producing cirrhosis than wine or beer, and it has been supposed that other ingesta besides alcohol may set up the disease; the excessive use of spices and other condiments has been charged with causing it in India.

Cirrhosis has been attributed to the prolonged presence of gall-stones in the hepatic ducts; and persistent jaundice from obstruction of the biliary passages appears more or less constantly to lead to an overgrowth of the connective tissue in the portal canals—in fact, to a slight degree of cirrhosis. H. Mayer, Wickham Legg, Charcot, and Simmonds have produced this *biliary* cirrhosis in rabbits and cats by ligature of the common bile-duct. Clinically, however, such cases are very rare, and may mostly be ascribed to the more common and certain causation (see Prof. Potain, 'La Semaine médicale,' 1896; and Dr P. Weber, 'Brit. Med. Journ.,' 1896, vol. i, pp. 10—27; and Dr Taylor's comments, 'Guy's Hosp. Rep.,' lii, pp. 74-5).

Chronic heart disease, again, is believed by some pathologists to be a cause of cirrhosis; but this is exceedingly doubtful. By retarding the circulation through the liver, it may perhaps enable comparatively moderate quantities of alcohol to produce the disease.

Repeated intermittent fever is stated to be another cause of cirrhosis of the liver, but of this malarial form we see little in England. In one case of a large liver with jaundice in a patient of the writer who had lived in India, there was also a history of spirit-drinking.

Syphilis produces effects on the liver which resemble cirrhosis, but are really quite different (cf. p. 542).

In concluding that excessive indulgence in alcoholic liquors is the principal cause of cirrhosis, one cannot deny that well-marked cases are occasionally met with in children and others, who were certainly not intemperate. Such cases have been ascribed to scarlet fever (Barlow, 'Path. Trans.,' 1877, vol. xxviii, p. 355) or to rickets. One of the most conclusive cases of typical cirrhosis which was not alcoholic in origin was recorded by Mr D'Arcy Power, in an elderly clergyman who had suffered for many years from biliary colic and had passed gall-stones ('Path. Trans.,' 1890, p. 152). Another case in a girl of twelve was reported by Dr S. Mackenzie (*ibid.*, 1889, p. 339).

Two French physicians, MM Gilbert et Fournier, in the 'Revue des Mal. de l'Enfance' for 1895, have described marked cirrhosis of the liver in children, where the possibility of an alcoholic origin could be excluded, associated with an unusually large spleen, and with other more or less peculiar features. Dr F. Taylor has drawn attention to this form of disease in England in a paper in the 'Guy's Hosp. Rep.' for 1895 (vol. lii, p. 45), where he described three cases. The writer saw the autopsy on the third of these, and was struck by the enormous size of the spleen, and the characteristic cirrhotic condition of the liver. There had been repeated paracentesis for ascites and long-continued jaundice.

Some writers have ascribed a tuberculous origin to certain cases of cirrhosis; but this must be extremely rare, if a real cause at all, for whether we look at the autopsies on cases of chronic phthisis in adults, or on those of acute tuberculosis in children, it is very rarely that a cirrhotic state of the liver is found; and when present it is probably due to the intemperate habits which are unhappily as common as tubercle. (Cf. 'Path. Trans.,' vol. xxxiii, p. 172.)

Microbic infection.—A bacterial origin for cirrhosis has been sought for. It is said that in the Pictou disease of cattle in Nova Scotia, there is cirrhosis of the liver, with ulcers of the fourth stomach, and that diplococci are found in the hepatic cells. Dr Adami (now of Montreal) investigated human cirrhosis from this point of view, and has discovered similar diplococci in the epithelial cells of the liver. (See his interesting papers in the 'Montreal Med. Journ.,' July, 1898, in the 'Lancet' for August 13th, 1898, p. 396, and 'Brit. Med. Journ.,' October 22nd, 1898.)

Sex and age.—Of patients who die from the effects of cirrhosis of the liver, it appears from the records at Guy's Hospital that there is a larger number between forty and fifty years of age than in any other decennial period. Kelynack gives from forty-two to forty-six, and Rolleston and Fenton from fifty to sixty. The proportion of males to females at Guy's Hospital was 102 to 26; in 114 cases (also fatal cases) at St George's Hospital the numbers were 82 to 32 (Rolleston and Fenton, 'Birm. Med. Rev.,' Oct., 1896). In 121 fatal cases at Manchester they were 82 to 39 (Kelynack, *ibid.*, Feb., 1897).

Matthew Baillie remarked a hundred years ago that this disease is hardly ever met with in very young persons, but is frequent in persons of middle or advanced age; and more common in men than women. "It is more apt to occur in those who have been accustomed to drink spirituous liquors" ('Morbid Anatomy,' p. 228). So also Heberden's opinion, given somewhat earlier, was that men are more commonly affected with scir-

rhous (*i. e.* cirrhotic) livers than women, because they are more given to intemperate drinking" ('Commentaries,' p. 253).

Effects.—Latent cirrhosis is not very rare,* and the early symptoms are exceedingly indefinite. They are chiefly those which have already been described as "bilious dyspepsia" (p. 502), together with other symptoms of chronic alcoholism, restlessness at night, tremor of the tongue and hands, irritability of the bowels, and nausea. The chief cause of the more characteristic symptoms is the congestion due to pressure of the new fibrous tissue on the portal capillaries. This pressure is so great in advanced cirrhosis that we wonder how the blood returns from the chylipoietic viscera. Some of it no doubt escapes through the anastomoses which exist at the upper and lower limits of the distribution of the rootlets of the portal vein. Thus the œsophagus is often found to be surrounded by a plexus of dilated vessels, which had carried upwards a part of the blood from the stomach, and hæmorrhoids are very frequently present, which may be taken as an indication that some of the blood from the rectum had passed away by adjacent branches of the iliac veins. But these communications seem insufficient to make up for the great obstruction that must exist in many cases of cirrhosis.

Rindfleisch found in one extreme instance that the portal blood passed directly into the inferior cava through numerous dilated anastomoses between the mesenteric and spermatic veins. Frerichs laid stress on the existence of vessels in the newly formed adhesions between the liver and the diaphragm and abdominal wall. Sappey drew attention to accessory branches of the portal vein, which run along the round ligament of the liver to reach the under surface of the diaphragm, and in two cases of cirrhosis he found this vessel distended to the size of the little finger. Dr Fagge observed a large vein in this position when attempting to inject the portal vein in the body of a patient whose liver was hobnailed.

There seems to be no doubt that much of the portal blood passes into the epigastric and circumflex ilii veins, which anastomose with the internal mammary veins; for the superficial vessels of the abdomen become greatly distended in cases of cirrhosis, when there is no evidence that the trunk of the inferior cava is compressed.

The congestion of the portal system of vessels which results from cirrhosis of the liver accounts for the fact that after death from this disease the stomach is generally found to be reddened and lined with thick mucus. The spleen is often enlarged, according to some writers almost invariably; but Frerichs found the splenic enlargement in only half his cases. Its size is, no doubt, variable, but few autopsies on cases of cirrhosis take place in which it is small, and sometimes it is very large. Excluding the remarkable cases referred to above of non-alcoholic cirrhosis in children, the spleen is larger than it is in enteric fever, but not nearly so large as in septic embolism or anæmia splenica, and it never approaches the bulk of the malarial or leucæmic spleen.

A frequent and valuable symptom of cirrhosis of the liver is the presence on the patient's cheeks of minute red lines and points, consisting of cutaneous venules that have become varicose but are termed "stigmata."

* At Guy's Hospital the liver is accidentally found cirrhotic (in persons who have died of injury or of some other disease) once for every two cases in which cirrhosis has been the cause of death. In many of these cases the organ is indurated in an extreme degree, yet the patient certainly suffered from none of the marked symptoms of cirrhosis.—C. H. F.

They are not confined to the face, and may often be found on the chest and abdomen.

Hæmorrhage from the nose, as well as from the stomach and bowels and the skin, is a frequent symptom of cirrhosis of the liver.

The urine in cirrhosis is usually bile-stained, and deposits pink lithates. It is also stated that the excretion of ammonium salts is increased as that of urea is diminished, owing, it is supposed, to interference with the formation of urea from ammonium carbamide in the liver. In the later stage urea is very much diminished.

Dr Carrington observed pyrexia in eighteen out of forty-four cases of uncomplicated cirrhosis ('Guy's Hosp. Rep.,' 1883).

There remain the most constant effects of cirrhosis: hæmatemesis, ascites, jaundice, and cerebral symptoms.

Hæmatemesis, which has been described in the chapter on gastric ulcer, is one of the early symptoms. It is often profuse, the blood is dark, sometimes clotted, and it is not accompanied by the pain of gastric ulcer or carcinoma. *Melæna* is a usual result: if unaltered blood is passed from the bowels, it means hæmorrhage from the intestinal veins.

Ascites, a condition which has been fully described already, is also an almost constant effect of cirrhosis of the liver. It is usually abundant, and gives all the physical signs described in a preceding chapter (p. 494). It is probably a purely passive effusion, and often an occluding thrombus is found in the portal veins.

Jaundice, as a symptom of cirrhosis of the liver, has already been mentioned (p. 511). Among 130 cases in which the liver was found after death to be hobnailed, there were thirty-four marked by more or less jaundice, and nineteen in which it was intense. This statement, however, hardly does justice to the frequency of icterus, in comparison with the other clinical symptoms of cirrhosis; for in more than forty of the 130 cases the cirrhotic state of the liver was accidentally discovered in the *post-mortem* room. This would leave less than ninety cases in which the cirrhosis produced marked effects during life; and among these the proportion of cases in which some jaundice was present would be more than one in every three. The liver is generally enlarged in these cases; in nearly half the instances which occurred in Guy's Hospital with intense jaundice the organ weighed more than seventy ounces, once as much as 131 ounces. With few exceptions it contained much fat.

Cerebral symptoms, especially drowsiness and coma, frequently usher in the fatal termination in cases of cirrhosis with jaundice; but they are also common in cases which give rise, not to jaundice, but to ascites. Even when diuretics and purgatives have cleared the peritoneum of its fluid, this often avails the patient little, for he presently becomes dull and dies unconscious, although his abdomen may be perfectly flat. Indeed, it is a question whether the removal of the fluid by tapping does not sometimes hasten the fatal issue.

In addition to the drowsiness and coma observed in cases of this kind, Frerichs mentions noisy delirium. We had one patient who, although he could be partially roused, seemed to be quite unaware of being in the hospital, and, when asked where he was, always named some street in the city where he had previously lived. This man lay for two or three weeks in a semi-comatose condition.

The cause of these cerebral symptoms is obscure. It has been supposed

to result from a disintegration of the secreting cells of the liver, as in acute yellow atrophy. But after carefully examining the tissue of the organ in several such cases, Dr. Fagge always found numerous liver-cells in an apparently unaltered state; the microscopical characters were, in fact, indistinguishable from those of other cases of cirrhosis. The condition in question has been named "acholia" or "cholæmia;" but, as we have seen, it is far from being confined to cases of cirrhosis with jaundice. If any poison is retained, it is not bilirubin nor cholesterine,* but some other constituent excreted by the liver.†

Hypertrophic cirrhosis.—Charcot, in accordance with the statements of Hayem, Hanot, and other French pathologists, taught that when cerebral symptoms are present, with jaundice and without ascites, the liver will be always found enlarged; and that the cirrhotic change has then begun, not around the lobules in the portal canals, but within the lobules. He also believed that this intra-lobular or "monolobular" hypertrophic cirrhosis is not, like the ordinary hobnailed liver, due to drink.

It is no doubt a true clinical observation that cases of cirrhosis with marked jaundice run, as a rule, a more rapid course than those without—although one such patient in Guy's Hospital had persistent jaundice for seven years, and died at last of hæmatemesis, and not of cholæmia. It is also true that early and extreme ascites accompanies the more atrophied and contracted condition of the cirrhotic liver, while early and marked icterus is more common with a large liver. But certainly we meet with livers which are above normal weight in persons who have been intemperate, and who suffer from ascites with little or no jaundice.

Charcot described the formation of new biliary ducts within the lobules in cases of hypertrophic or "biliary" cirrhosis. It is difficult to prove that these are not the remains of hepatic tissue which have survived the disease, and Dr Saundby has shown that these new-formed ducts may be present when there is no jaundice, and absent when jaundice is well marked.

There seems therefore no ground for regarding "Hypertrophic cirrhosis" (more properly cirrhosis with a large liver) as a different disease from atrophic or common cirrhosis. The liver varies in size, but the causes, symptoms, prognosis, and course do not vary according to the size of the liver. This opinion is generally held in this country, and is temperately but decidedly defended by Dr Cheadle in his Lumleian Lectures (1900, pp. 21—35).

Prognosis of cirrhosis.—This is always very grave, though we have evidence that the process may become quiescent, and possibly may be arrested.‡ The frequency with which cirrhosis of the liver is latent is a remarkable feature of the disease. On casting up the ages of persons in whose bodies cirrhosis of the liver was discovered without there having been marked symptoms during life, Dr Fagge found that the average age was higher by about five years than that of those who died from the ordinary symptoms of the disease. This fact would prove that cirrhosis is not always (as we have been inclined to suppose) a progressive process, but rather that after having reached a certain point it may become stationary, and remain so

* A theory propounded by Dr Austin Flint, jun., was that the cerebral symptoms in these cases, and in acute yellow atrophy, are due to accumulation of cholesterine in the blood, for which he invented the name "*cholesteræmia*."

† See the observations of Weintraud, quoted by Dr V. Harley in the 'Clinical Journal' for July 1st, 1897, p. 195.

‡ See Dr Cheadle's remarks on prognosis in cirrhosis (Lumleian Lectures, pp. 57—75).

for the rest of the patient's life. The writer once had a man under his care for bronchitis, who had also a hard liver, which projected below the ribs. He had been in the hospital before with ascites and other symptoms of cirrhosis, but had been free from them for some years and so remained.

Before ascites appears, the progress of cirrhosis is marked by years, afterwards by months or weeks. After tapping, the fluid, as a rule, re-accumulates; and at last death occurs from coma or from dyspnoea.

Its *treatment* is first that of hepatic dyspepsia (p. 502), and afterwards that of ascites (p. 500). Paracentesis should be performed as soon as there is serious difficulty of breathing; but before this point is reached, such diuretics as digitalis and squill with mercury, acetate and iodide of potash, or resin of copaiba should be pushed. In more than one case the writer has seen ascites disappear and the patients apparently recover after free diuresis without being tapped.

In one patient of the writer, a robust labourer of about 35, ascites from cirrhosis was treated by acid tartrate of potash in lemonade, and, as he put it, he "passed all the water from his belly into the pot." He went out apparently in good health and vowing to live soberly.

In another case of cirrhosis, which he saw with Dr H—, for ascites and other serious symptoms, he advised tapping, which was performed twice with marked relief. Several months later he was sent to see the patient's wife, who was suffering from the same disease, and her husband was going about, apparently well. She also was tapped, but her case ended fatally.

The patient must, at any stage except perhaps the latest, be induced, if possible, to give up all intoxicating liquors.

Chronic inflammation of the capsule of the liver, or perihepatitis, is pathologically different from true cirrhosis, but closely resembles its clinical aspect. In this affection the organ is remarkably deformed; it no longer has a sharp edge, but is converted into a rounded mass. Its capsule is opaque, and often forms a separable layer, which, when stripped off, leaves a smooth surface. The alteration in the form of the liver is in part caused by the contraction of this thickened capsule; but sometimes its anterior edge is also folded over on to the dorsum.

In a case of this kind the margin of the liver touched a part of the convex surface that should have been four and a half inches distant in a direction from before backwards, and when the capsule was removed the organ returned to its natural shape.—C. H. F.

The weight of a liver affected with perihepatitis is generally about the same as that of the healthy organ. Its tissue is commonly soft, and is very often loaded with fat. It is seldom cirrhotic, but there may be a slight excess of fibrous tissue in the course of the larger portal vessels.

Perihepatitis is a not infrequent cause of ascites. It does not cause jaundice nor cholæmic symptoms; and it seems to be found less often than cirrhosis in persons who die of other diseases or are killed by accident; whence it would appear that it does not remain latent, but always advances until it causes ascites. In most cases of perihepatitis the kidneys are diseased, and often it is only a locally exaggerated form of general chronic peritonitis in the course of Bright's disease. It is sometimes found associated with chronic hypertrophic pleurisy and pericarditis.

Dr Hale White believes that most of the cases of portal ascites which do well after repeated paracentesis are not due to cirrhosis, but to perihepatitis; and no doubt the prognosis in the latter case is better than in the former.

Simple hypertrophy of the liver, distinct from hypertrophic cirrhosis, and from the vascular turgescence caused by heart disease, is not very uncommon. It is found in beer-drinkers, and in cases of diabetes. Wilks and Moxon record a liver of the former kind which weighed 80 oz., and was healthy in texture. They also describe compensatory hypertrophy when part of the organ has become atrophied from pressure or from syphilitic growths, and note a case in which the whole right lobe was atrophied, and the left weighed 56 oz., thus making up the loss.

Local circumscribed outgrowths of hepatic tissue are curiosities only, and are probably referable to a form of innocent glandular tumour, adenoma.

Simple chronic atrophy is a rare affection of the liver. In a case recorded by Dr Cayley ('Path. Trans.,' 1868) the liver weighed only twenty-two ounces; and the left lobe had almost disappeared, being only an inch wide. In another case, one of Murchison's (*ibid.*, 1867), the organ weighed twenty-five ounces; its margin was thin and flat, forming a kind of rim, which consisted only of connective tissue and vessels enclosed between the two layers of the capsule. This rim measured in one place an inch across. In both cases the substance of the liver was of a dark colour, and quite free from induration.

Minor degrees of atrophy of the liver are by no means uncommon, particularly in old people and in those who die of wasting diseases, such as cancer of the stomach or œsophagus. It does not, however, accompany phthisis, for when the rest of the body is emaciated the liver is usually enlarged by decomposition of fat. Atrophy of the liver may also be the result of chronic perihepatitis.

Syphilitic hepatitis.—Chronic interstitial hepatitis, with scarring and gummata, is sometimes called syphilitic cirrhosis, but this is an undesirable use of the word—like calling a chronic tuberculosis of the lung cirrhosis.

Sometimes gummata are scattered through the hepatic tissue, which is in other respects healthy. The condition is generally unattended with any symptoms; but it may happen that one of the gummata is so placed as to obstruct the circulation through the organ, and thus cause ascites. A case of this kind occurred at Guy's Hospital, in which one of the hepatic veins, close to the inferior vena cava, was so narrowed that it would only just admit a probe.

More often the gummata, instead of being embedded in the hepatic tissue, lie in the midst of broad fibrous bands, which traverse the liver from one surface to the other, forming deep notches or depressions, or cutting off large masses from their continuity with the rest. The deformity thus produced is obvious; and even when the syphilitic interstitial hepatitis has been more general, the fibrous bands are coarser and the islets of healthy tissue larger and less regular than those of true cirrhosis. The process is one of cicatrisation and puckering, and firm or caseous gummata are found as the foci of the star-like depressions. Perihepatitis often accompanies the interstitial hepatitis of syphilis.

Beside containing gummata and fibrous bands, the syphilitic liver is often lardaceous, and then it may reach a very great size, weighing from six to seven pounds, while the capsule is generally thickened and adherent to adjacent parts. In such cases ascites is most apt to occur.

Grainger Stewart recorded the following case, which appears to have been of this kind: a patient had ascites, for which she was tapped twenty-one times, the enormous quantity of 606 pints being removed in the course of these operations; at first the paracentesis had to be repeated every fortnight, but the intervals gradually became longer, until at length she regained tolerable health.

In twenty years (1862-82) Dr Fagge noted in Guy's Hospital only six cases of fatal ascites from gunnatus and lardaceous disease. In several of them the liver could be felt during life to be enlarged and adherent to the parietes, with an uneven and nodular surface, and by these characters a correct diagnosis was more than once made.

A very different congenital effect of syphilis on the liver has been found in the case of infants—a diffuse interstitial hepatitis leading to a uniform pale and very firm texture. It was first described by Gubler and Lancereaux in France in 1852, and afterwards by Wilks in this country (cf. 'Path. Trans.,' xvii, p. 167).*

ACUTE YELLOW ATROPHY OF THE LIVER.†—This is the name given to one of the most remarkable diseases known, rare in its occurrence, obscure in its pathology, fatal in its results, and unique in its anatomy.

History.—For many years it had been known that cases of apparently idiopathic jaundice occasionally do not run a favourable course, but become complicated by hæmorrhage and cerebral symptoms, and end in death by coma; they were called *icterus gravis*, or malignant jaundice. But the first clear and complete cases were published by Richard Bright in the first volume of the 'Guy's Hospital Reports' (1836), under the title "Intense Jaundice without Mechanical Obstruction, apparently depending upon Inflammatory Action in the Liver." The following is his account of these two typical cases of acute yellow atrophy (Nos. 5 and 6, pp. 624—630).

(1) A woman aged twenty-eight, of dissolute habits, while taking mercury, was attacked with abdominal pain, and jaundice quickly followed. Dr Bright saw her on the third day; the urine was then bile-stained and the fæces clay-coloured; on the twenty-first there was drowsiness, followed by delirium. She died comatose on the twenty-third day of the jaundice.

At the autopsy there was intense staining of every tissue with bile; the contents of the head, chest, and abdomen were healthy, except the liver, which weighed only thirty-seven ounces, and was "soft or flaccid to the touch," with no trace of peritoneal inflammation.

(2) A German girl, aged eighteen, was admitted into Miriam Ward January 11th, 1832, labouring under *icterus*. "The skin was of a brilliant yellow, and the cheeks, which were flushed, were the colour of a very ripe apricot." She had probably been ill for nearly four weeks, and the jaundice had gradually deepened to its present tint. She had lately sat by the fire in a kind of doze. That evening she vomited, and "lay in a perfectly torpid state the whole night, apparently suffering no pain; but towards the morning became delirious, so that it was with difficulty she could be restrained in her bed." Dr Bright ordered "two grains of calomel every two hours, and the ammonia julep (Mist. Ammoniae Co. of the present Guy's Pharmacopœia) every four hours, besides wine if she became more depressed." Purging,

* See Dr Adami's remarks on the liver of congenital syphilis ('Montreal Med. Journ.,' June, 1898).

† *Synonyms.*—*Icterus gravis*—*Icterus typhoides* (Lebert)—Irish yellow fever—*Ictère malin*—*Ictère hémorragique essentiel*—*Die acute Leberschmelzung* (Rokitansky)—*Atrophia hepatis flava sive acuta* (Frerichs)—*Hepatitis parenchymatosa diffusa acuta*—*Hépatite épithéliale de la fièvre ictérique* (Lancereaux).

enemata and a blister over the liver were also ordered; the head was shaved and mustard was applied to the feet. She continued, however, completely comatose all the following night, and died at ten in the evening of January 13th.

At the autopsy, excepting a universal and deeply jaundiced tint, the brain, heart and lungs, stomach and intestines, pancreas, and kidneys were found normal. The spleen was soft. "The liver was unusually small, and for the most part of a brightish yellow colour, with portions marked with purple or deep brown."

Dr Bright remarks, "In this case, as in the last, no obstruction could be discovered in the ducts which could have prevented the flow of bile from the liver." "The immediate cause of death in this case, as in the last, was the poisoning influence of the bile on the system." "The bile must have been rapidly absorbed into the system almost at the moment of its formation, and its profuse mixture with the blood seems to have acted as a poison, and hence the immediate cause of death. I am inclined to consider this as the result of a decidedly inflammatory state of the organ." Again, he remarks that, in these severe cases of jaundice with nervous symptoms, "the tendency to hæmorrhage comes on very early and is excessive."

Seven years later, in 1843, Graves, of Dublin, published in his famous 'Clinical Lectures' (2nd ed., 1848, vol. ii, p. 255) three cases of jaundice in sisters. One, a girl of seventeen, was attacked in July, 1840, with vomiting and icterus, followed by hæmatemesis, violent delirium with convulsions, coma, and death on the seventh day. No autopsy was permitted. The second sister, aged eleven, died in March, 1841, with similar symptoms on the fifth day. After death the liver was of "natural size," dull yellow with dark spots, and bile in the gall-bladder; the brain (examined first) was much more vascular than usual; the thorax was not examined. The third sister, aged eight, was taken ill with jaundice and vomiting the following June. She was actively treated by bleeding, leeches, and calomel, with James's (the compound antimonial) powder; the alarming symptoms disappeared after three days, and she recovered from her jaundice in about three weeks more.

Graves's cases were quoted as examples of acute atrophy by Budd and by Trousseau, and also by Frerichs in his well-known treatise. But there is no evidence that the last was anything more than ordinary icterus; the first is incomplete, and in the second there was no atrophy of the liver discovered.*

Rarity.—Acute yellow atrophy of the liver is a rare disease. Murchison says that although delirium and a brown tongue were a certain passport into the London Fever Hospital, only one case occurred among 3000 patients admitted in a period of six years. In Guy's Hospital there are notes of the inspection of only eleven cases in twenty-seven years, 1864-90.

Symptoms: Icterus.—The jaundice of acute yellow atrophy is very deep, but not otherwise remarkable. The conjunctiva and urine are bile-stained, and at least in most cases the stools are clay-coloured. Icterus may precede the graver characteristic symptoms not only by a day or two, but by weeks.

Nervous system.—The most distinctive symptoms are cerebral. Head-ache and intolerance of light are often first complained of, and before long

* Isolated cases of rapid and fatal jaundice, which may with more or less probability be referred to yellow atrophy, have been quoted by Frerichs and Trousseau from Rubæus (1660), Boerhaave, and Morgagni. More recent cases were described by Cheyne and Marsh in Dublin, and by Abercrombie in Edinburgh, between 1820 and 1830.

consciousness is more or less impaired. The patient becomes restless, screaming and tossing about, and sometimes fiercely maniacal; or violent convulsions may seize him. This irritable stage, whether more or less severe, always ends in complete coma; the pupils become widely dilated and insensible to light; the urine and fæces are passed involuntarily; and the breathing is stertorous until death ensues.

When acute yellow atrophy occurs in a pregnant woman, abortion or miscarriage almost always follows.

Sometimes instead of being maniacal, the patient falls into a "typhoid state;" his tongue is dry and brown, and his lips and teeth are encrusted with sordes. According to some writers, there is considerable pyrexia; but this is only an occasional symptom, for in several of the cases at Guy's Hospital the temperature was normal. In one patient it was below the average four days before death, but began to rise two days later, and shortly before his death it was 101.6° . Sir Dyce Duckworth noted the absence of pyrexia in three cases at St. Bartholomew's Hospital. Frerichs says that in his cases the skin was usually cool, dry, and inactive, and he quotes Bright and Addison to the same effect. The pulse is, as a rule, accelerated, but in one of Duckworth's cases it was on two days about 50. Towards the end it becomes very small and intermittent.

The death of the patient almost always takes place within five days from the onset of the characteristic symptoms. It is said that acute yellow atrophy has sometimes destroyed life in twenty-four hours, but Niemeyer's statement that the majority of cases end fatally on the second day is certainly not correct.

Physical signs.—We can by means of percussion trace from day to day the gradual diminution of the liver. From a normal measurement of four inches vertically in the right mammary line—reaching from the fifth intercostal space downwards to the costal margin—the hepatic dulness may be watched as it undergoes reduction to three inches, two inches, and one inch, until at last it disappears entirely.*

State of the urine.—This is always, or nearly always, albuminous—a symptom of concomitant nephritis; for in many cases, perhaps in all, there is more or less tubal inflammation. The urine does not generally look black when in bulk, nor in a thin layer has it an intense saffron-yellow colour.

Besides the icteric discoloration and presence of albumin, another change in the urine is constant and characteristic. The urea and lithic acid, and also the chlorides, sulphates, and earthy phosphates, are greatly diminished in quantity or nearly absent; and in their place are found two new substances—*leucine* (amido-caproic acid) and *tyrosine* (amido-sulphoperuvic acid), both products of albuminous decomposition. There is generally no difficulty in detecting them. They sometimes form a distinct deposit when the urine is left to stand for a time, or, if this is not the case, they may be made evident by evaporating a few drops with a drop of acetic acid on a glass slide. Tyrosine is easily recognised by its taking the greenish-yellow colour of the urine; it occurs in bundles or globular masses of needle-shaped crystals. Leucine appears as rounded, pearly discs, generally marked with concentric rings.

* In all forms of jaundice the bowels are apt to become inflated with gas, and the consequent enlargement of the abdomen may cause the right hypochondrium to become more and more tympanitic, and the area of hepatic dulness to diminish from day to day. This has led to an erroneous diagnosis of acute yellow atrophy.—C. H. F.

Hæmorrhage.—This symptom is nearly constant in acute yellow atrophy. Very frequently the patient vomits a dark fluid resembling coffee grounds, or otherwise altered blood. Petechiæ are often developed in the skin, and almost invariably, towards the end of the case, the evacuations are dark brown or a tarry black as the result of hæmorrhage.*

The stools.—With regard to the colour of the fæces in acute yellow atrophy, writers differ. Murchison said "the jaundice appears to be due to a poisoned condition of the blood, and consequently bile is still found in the stools." But it is certain that towards the end of a case of acute atrophy no bile enters the intestines, for after death the ducts and gall-bladder are found to contain an almost colourless mucus. Moreover, Frerichs says that in this form of jaundice the stools are dry and clay-coloured; and more than one case is recorded at Guy's Hospital which supports this statement. The question is not so easy of determination as might at first sight appear, on account of the great frequency of intestinal hæmorrhage in this disease. When the stools have been supposed to contain bile in acute atrophy of the liver, their dark appearance may have been due to altered blood. We must, however, remember that the disease does not always affect the whole liver uniformly, but attacks some parts earlier than others; so that at first bile very possibly continues to enter the intestines from those portions which have not yet been attacked.

When what seems ordinary icterus precedes the special symptoms of the disease, it is probable that such more chronic cases are examples of "red atrophy" (see the cases recorded by Dr Moxon, 'Path. Trans.,' 1872).

Anatomy.—The first thing that strikes one, in making an inspection, is the diminution in the size of the liver. It forms a thin flaccid mass, which lies at the back of the abdomen, hidden by the ribs and by the distended intestines. Instead of weighing from fifty to sixty ounces—the usual weight of an adult liver—it weighs perhaps thirty-two ounces, or not above twenty-three.

When cut into, the tissue looks as though it were softened; but the finger is found not to penetrate it more readily than usual, for although it is so flabby, it is not friable. Its colour is altered to a bright orange-yellow tint, with some parts dark red or purple. Portions having this red or purple hue are scattered through the substance of the organ; sometimes one part, generally the left lobe, is almost entirely red, while the rest of the liver is mainly of a gamboge-yellow hue. To the naked eye it appears as though the red parts were less altered than the yellow, but the microscope shows that this is not the case. In both parts the hepatic cells have undergone destruction, and are replaced by a mass of granules and oil-globules; but in the red parts the destruction is complete ("red atrophy"), whereas in the yellow parts some of the secreting cells still remain visible, and towards the centres of the lobules may even retain their columnar arrangement ("yellow atrophy"). Among the remnants of the hepatic tissue are often to be seen crystals of leucine and tyrosine.

A pellucid nucleated material has been detected along with the detritus of the cells, and Dr Fagge observed this in two cases.

Waldeyer and Klebs described, in the reddened parts, cells resembling those of the epithelial linings of the biliary ducts, arranged in regularly branching lines, and tubes which seemed to have cæcal terminations (cf. *supra*, p. 540).

* Dr A. H. Carter, of Birmingham, has drawn attention to a red pigment in the fæces which resembles unaltered blood in tint but is not hæmoglobin ('Lancet,' Nov. 25th, 1899, p. 1432).

The larger bile-ducts are found empty; their mucous membrane is unstained by bile-pigment. The gall-bladder is either empty or contains a few drachms of grey mucus or of a pale yellow or greenish fluid.

The kidneys can generally be shown by the microscope to have undergone morbid changes. The epithelium of the tubules is granular and may be very fatty. In fact, the condition is one of subacute tubal nephritis.

Ætiology.—Among the exciting causes of this disease, excessive mental emotions have been reckoned (p. 509). More than one instance has been recorded in which acute atrophy has followed directly upon a drunken debauch; and occasionally it has set in during the secondary stage of syphilis. Of 22 female patients referred to by Frerichs one half were pregnant, and of 88 collected by Thierfelder (quoted by Eichhorst) 30. Of Dr Wickham Legg's 69 cases in women, 24 occurred in pregnancy; and of Dr Wm. Hunter's 24, 12 were attacked during pregnancy or lactation.

Sex and age.—Apart from pregnancy, acute yellow atrophy of the liver is more common in women than in men—88 out of Thierfelder's 143 collected cases, 69 out of Legg's 100, and 24 out of Hunter's 42 being females.

It occurs principally in early adult life. Five times out of six the patient is between twenty and thirty years of age, and in nearly all between fifteen and forty.

It is rare in childhood, although Dr Goodhart recorded a typical case in a boy only two and a half years old ('Path. Trans.,' 1882). Dr Tuckwell had previously reported in 1874 two cases in boys, one seven years old, the other between four and five; and another occurred in a girl aged four and a half, a patient of the late Dr Charles West's in 1859 ('St Bart.'s Hosp. Rep.,' vol. x).

Pathology.—Some of the earlier writers on acute yellow atrophy of the liver, having found after death that the larger bile-ducts were free from obstruction, imagined that the minute channels which issue from the secreting lobules of the organ might have undergone compression from swelling of the secreting cells.

More recently the occurrence of jaundice from acute atrophy was held to prove the existence of a hæmatogenous icterus from suppression; but it is now probable that there is obstruction, although it is high up, in the interlobular bile-ducts, and that the obstruction is chiefly from inflammatory swelling of their mucous membrane.

It seems probable that acute atrophy of the liver is a *parenchymatous inflammation*, although we must admit that no precisely analogous disease can be found among those to which other organs are liable; and it leaves the striking atrophy unexplained. This was the view originally taken by Bright.

The cause of the inflammation may perhaps be soluble toxins absorbed from the intestine. Waldeyer and other pathologists have found microphytes in the liver, but they appear not to be constant. They were absent in Dr Cavafy's case ('Path. Trans.,' 1883), and in those examined by Klebs and by Senator.

Some uncertainty still prevails with regard to the origin of the leucine and tyrosine which, as we have seen, are excreted in the urine. It may be that in acute atrophy of the liver the metabolism of proteids in the portal capillaries is incomplete, so that, instead of urea and uric acid, the new products in question are formed; and this view accords with the fact that urea and uric acid are more or less wanting. But, since the normal liver during

decomposition contains leucine and tyrosine, it may be that these compounds are the direct products of the disintegration of the hepatic tissue.

Again, there is a doubt whether the granular and fatty changes in the epithelium of the renal tubules are the result of the disease of the liver, or whether both these conditions do not rather depend upon some common cause. The latter view appears to be the more probable.

What is the cause of the cerebral symptoms which form so striking a feature in acute atrophy of the liver? Frerichs thought that they depended on the presence of leucine and tyrosine in the blood, but experiments have failed to verify this supposition. Rokitansky supposed that the whole disease was of nervous origin, and the local changes only secondary. Virchow believed these symptoms to be uræmic, and dependent on the renal changes; but the character of the cerebral symptoms in acute atrophy of the liver is not the same as in uræmia, nor is nephritis constant.

Perhaps it is safest at present to regard this rare and puzzling disease as a peculiar form of hepatitis—as different from cirrhosis as from hepatic abscess, or as pneumonia from interstitial pneumonia, and due to some special form of toxæmia which affects the brain as well as the liver and kidneys. Its nearest alliance seems to be with cases of febrile or infective jaundice, including those of Weil (p. 511).

Diagnosis.—This is not difficult in most cases, if attention be paid to the characteristic symptoms, particularly the rapidly diminishing liver-dulness, the hæmorrhages, the delirium, the stupor, and the presence of albumen, leucine and tyrosine, in addition to bilirubin, in the urine.

In a patient whom the writer saw with the late Mr Toulmin in 1889, the early part of the case was like enteric fever, with diarrhoea and very slight jaundice, and it was only after more than three weeks that the appearance of leucine and tyrosine with decided icterus cleared up the diagnosis; the liver-dulness then rapidly diminished, cerebral symptoms supervened and ended in death by coma, after five weeks' illness and about ten days' characteristic symptoms. Unfortunately no autopsy could be obtained.

Poisoning with phosphorus is the condition which clinically resembles acute yellow atrophy most closely. The toxic effects of phosphorus are by no means limited to the vomiting and purging which immediately follow its ingestion. In a few hours these generally pass off, and the patient may appear to be perfectly well for three or four days; but at the end of this time jaundice often sets in, followed by delirium, coma, and death. The liver is then found to be in a state in some respects like acute yellow atrophy. Indeed, certain authors speak of phosphorus poisoning as one of the causes of the latter; but probably the two conditions are always distinguishable. In some cases of poisoning by phosphorus, at any rate, it is certain that the liver, far from being atrophied, is larger than natural. It is of a pale buff colour, very fatty, but mottled with numerous ecchymotic spots. Under the microscope the cells appear to have undergone destruction by fatty degeneration; but there is often a similar difficulty in detecting the hepatic cells in other cases of fatty liver—and even in the physiological steatosis of stall-fed cattle and of Strassburg geese—when there is every reason to believe that they are intact, although their outlines and nuclei are obscured by the oil-globules.

Clinically, poisoning by phosphorus differs from acute yellow atrophy in the absence of leucine and tyrosine from the urine; but liability to hæmorrhage forms a common and prominent feature of both diseases. After

poisoning by phosphorus the uriniferous tubules are loaded with highly refracting granules, like those in the hepatic cells, and the fibres, both of the voluntary muscles and of the heart, are found to have undergone a granular fatty degeneration. Thus the morbid state produced by phosphorus appears to be an acute steatosis of the liver, kidneys, and muscles. In England such cases are rarely seen, but in Germany they are far from uncommon, where, as a means of suicide, the heads of a bundle of lucifer matches seem to be frequently used.

In a case of phosphorus poisoning which occurred at Guy's Hospital the temperature of the body was very low, at least for some hours before death, the thermometer standing in the axilla at 96.8° , and afterwards at 91.5° .

In another case under the writer's care, a woman destroyed herself and her child, five years old, with phosphorus. The child perished quickly, as if by exhaustion, with little vomiting and no pain; but the mother lived for several days, and at first appeared to be recovering. There was no marked jaundice, no hæmorrhage, and no delirium, but she sank rapidly into coma. After death the liver was found large, in a state of fatty degeneration, and the same process had affected the kidneys and the heart, but there were no appearances like those of acute yellow atrophy. In the child the liver was very fatty, but the heart and kidneys were unaffected.

Statistics.—The results of Thierfelder's collection of 143 cases of acute yellow atrophy have been mentioned above (p. 547). The following is a summary of all the cases which have occurred at Guy's Hospital during twenty-five years from 1866 to 1890 inclusive. Some cases of hepatic atrophy were doubtful, *e. g.* a liver weighing 31 ounces but without icterus; and others were referable to "simple atrophy," diffuse forms of cirrhosis, or the atrophy secondary to the congestion of chronic cardiac disease.

Excluding these, there were during the twenty-five years eleven cases, five in men and six in women. The ages were as follows:—one boy only two and a half years old (Dr Goodhart's patient referred to above), a youth of eighteen, six patients between twenty-one and twenty-three, one of thirty-four, and one woman of forty-four, who appeared however to be older.

The total duration of the jaundice was from ten days to five weeks, but in three cases there was a period of several weeks in which jaundice was present without other symptoms. Beside jaundice, there were present in every case hæmorrhage (usually hæmatemesis) and delirium ending in coma. Albuminuria was seldom absent. The temperature was sometimes raised, sometimes subnormal, and in one case it rose to 104° F.

The weight of the liver after death varied in most cases (excluding the child's) between 26 and 33 ounces, but in three it was 40, 47, and 46 ounces, the last case being that of a woman. In these three cases the atrophy was partial but characteristic. As a rule the kidneys were in a state of tubular nephritis: in one only were they described as perfectly healthy.

Prognosis.—The event of acute yellow atrophy is not absolutely constant. Once Wilks had at Guy's Hospital a case in which there was a distinct history of a previous attack that had been recovered from. The patient had become delirious, and had such severe hæmatemesis that it was thought he would die in a few hours; but he rallied and lived two months longer, at the end of which he again became delirious. Leucine and tyrosine were then found in the urine, and he died a fortnight later. The liver was found to weigh forty-seven ounces. The left lobe and the adjacent

part of the right lobe were small and dark-looking; the lobules distinct, but scarcely any hepatic cells visible. The rest of the right lobe formed a soft, yellow, rounded, projecting mass. The marked contrast appeared to justify the supposition that the left lobe had become atrophied at the time when the cerebral symptoms first arose. Another case of recovery, followed by a second fatal attack, was recorded by the late Dr Frank Smith, of Sheffield ('Path. Trans.,' 1877, p. 236).

A list of no less than twenty-eight cases of supposed recovery from acute yellow atrophy will be found in Dr Wickham Legge's treatise 'On the Bile and Jaundice' (p. 676).

Treatment.—No remedies for icterus gravis are known. Our recent cases of this most singular disease have not been actively treated, for it was taken for granted that they must terminate fatally. However, there are, as we have seen, a few exceptions to this rule; and since the disease seems not to attack the liver as a whole, but often to spread through the organ from the left lobe, it is possible that its course may be influenced by treatment. Dr Budd recommended a drachm of the sulphate and fifteen grains of the carbonate of magnesia three times daily; advice which seems to have been founded upon the brilliant results which certain Irish physicians formerly obtained from purging in cases of icterus gravis. The most striking examples were the cases recorded in the year 1834 by Dr Griffin, of Limerick. Four children of the same parents were attacked within a few weeks by jaundice, with cerebral symptoms. Two of them died, but two recovered after having been in a state of almost complete coma. The treatment—bleeding, blistering, and active purging—was the same which failed in the hands of Dr Bright.

Large doses of quinine, sulphocarbolate of soda, and perchloride of mercury have proved as useless as the older treatment. In our present ignorance of the origin and nature of the disease we cannot expect to do good, and may easily do harm by interference.

If, however, symptoms indicative of the onset of this fatal malady should appear in a pregnant woman, it would be right to bring on premature delivery.

NEW GROWTHS AND DEGENERATIONS OF THE LIVER

Væ meum
Fervens difficili bile tumet jecur :
Tum nec mens mihi nec color
Certa sede manet, humor et in genas
Furtim labitur, arguens
Quam lentis penitus macerer ignibus.—HORACE.

*Carcinoma hepatis—its anatomy and histology—its rarity as a primary disease and its most frequent antecedents—its symptoms, diagnosis, and course—
Sarcoma—Cancer of the gall-bladder and bile-ducts—Angioma.*

Hypertrophy of the liver—diabetic, nutmeg, and obstructed liver.

Displacement of the liver—Enteroptosis.

Lymphatic overgrowth—Leuchæmia hepatica.

The fatty liver—General obesity ; its physiology and its treatment.

Malarial enlargement of the liver—Lardaceous disease—Hepatic tubercle.

Hydatids of the liver—Other cysts of the liver.

THE remaining diseases of the liver are somewhat heterogeneous, and most of them have greater pathological than therapeutical interest. They agree anatomically in all causing enlargement of the liver, and clinically may thus be separated from atrophic cirrhosis and acute yellow atrophy. With the exception of the first, malignant disease, they are mostly free from jaundice and from ascites. Indeed, it is surprising how little structural changes in this organ affect its functions, secretory or metabolic.

CARCINOMA.—Malignant disease of the liver has long been known to pathologists, and is of frequent occurrence. But it is rarely primary, and in most cases follows either cancer of the stomach or rectum, breast or uterus, or else cancer of the gall-passages or pancreas.

Anatomy.—The usual form of cancer of the liver is that of numerous separated nodules. They are scattered irregularly through its substance, but some are sure to reach the surface. They may now and then be seen as minute white or yellow points not bigger than hepatic tubercles, but most of them are much larger, and they may grow to be masses as large as a foetal head.

These tumours are usually soft (*encephaloid* or *medullary*), and yield an abundant white juice on scraping. They sometimes undergo caseous degeneration in the centre, like tubercle. They are often so vascular that hæmorrhage takes place into their substance, so as to justify the term *fungus hæmatodes* formerly applied to them, as to the vascular excrescences of

mammary cancer; and occasionally they become almost cavernous in structure. As the result of central softening, the great lumps which are seen on the surface of the liver are marked by a depression in the centre which gives them a characteristic "umbilical" aspect, like the leaves of navelwort or the seeds of *nux vomica*. They were described by Baillie, in 1795, as "large white tubercles, in greater number near the surface of the liver than near the middle. . . . They consist of a firm, opaque white substance, and are generally somewhat depressed or hollow upon their outer surface."

In exceptional cases the cancerous nodules are much slower in growth, harder in texture, and more uniform in size. This form of the disease has been called scirrhus (*carcinoma fibrosum*); and it sometimes distorts the liver so uniformly and renders it so tough, that to sight and touch it exactly resembles the cirrhotic livers with the larger and less regular kind of "hob-nails." So close was the resemblance in a case under the writer's observation (1876) that it was a surprise when the microscope showed the true nature of the transformation (Guy's Path. Museum, prep. 1922³⁰).

Whatever the form, the histological structure of the cancerous tumours of the liver is almost invariably the same, that of typical glandiform alveolar carcinoma. Columnar-celled cancer is occasionally seen; epithelial (corneous) cancer less frequently; and most rarely of all, colloid carcinoma. Melanotic cancer is also rare, and is always secondary (*ibid.*, prep. 1937, *et seq.*).

Cancer of the liver is, next to the lymph-glands, the most common seat of secondary cancer, as its size and vascularity and its relation to the portal circulation explain.

The most frequent seat of the primary growth is the stomach, rectum or colon, next perhaps in the pancreas, testes, and œsophagus: in women the uterus and mamma would probably come next after the stomach.

Cases of primary cancer of the liver are met with, but they are very rare, much more rare than in the pancreas among racemose or the testes among tubular glands. In most of the cases formerly described as primary cancer of the liver the growth probably began in the gall-bladder, gall-ducts, or in the pancreatic duct.*

When the disease is truly primary it sometimes assumes an unusual infiltrating character. In a case of this kind under the writer's care in 1878 and 1879, the liver was enlarged to the enormous size of 200 ounces. There was no other trace of cancer in the body; and other remarkable features of the case were its long duration, its painlessness, and the youth of the patient, who was a boy of only twelve ('Path. Trans.,' vol. xxxi, p. 125).

A liver affected with this diffused form of cancer has a peculiar appearance on section. The lobular markings are everywhere plainly visible, but they are coarser than natural. The substance of the liver is grey or white; all parts of the cut surface yield a milky juice, and the microscope shows that the cells in the lobules have the character of cancer-cells, although they are arranged in radiating columns occupying the meshes of the blood-vessels, like the secreting cells of the healthy organ. Probably these elements are directly derived from the pre-existing epithelial cells, and not from connective-tissue corpuscles.

* One source of fallacy may be mentioned, which is that cancer of the gall-bladder growing into the hepatic tissue has sometimes been mistaken for a primary cancer of the liver. The cavity of the gall-bladder may in such a case be so small that, lying in the centre of the tumour, it is easily overlooked.—C. H. F.

The largest livers observed are those affected with cancer. We have had two at Guy's Hospital which weighed each 18 lbs. In a case recorded in vol. xxiii of the 'Pathological Transactions,' a liver, which was full of cancerous tubera, weighed $19\frac{1}{2}$ lbs.; another case is there referred to in which the weight was 24 lbs., and the late Dr Arthur Jones, of Northampton, met with a case in which the weight of 28 lbs. was reached.

Signs and symptoms.—Many cancerous nodules may be scattered through the liver without enlarging it enough for its edge to be felt. But the tubera may reach such a size as to be felt through the abdominal walls, or may be seen to rise and fall with each breath. They are generally firm to the touch, sometimes of apparently stony hardness; but occasionally they feel very soft, so that one might imagine fluctuation in them. Indeed, their centres may really become hollowed into cavities containing fluid, as in an instance which occurred at Guy's Hospital: a cancerous tumour formed a cyst that would have held a cocoa-nut; it was so near the surface of the organ that it might have yielded fluctuation; and it was filled with a clear straw-coloured liquid. Sometimes in emaciated patients cancerous nodules can be felt to have a central umbilicus—a sign of importance, for it is not observed in any other affection.

Instead of several distinct nodules or tubera, cancer of the liver may be felt as a single large rounded mass, projecting from the right or left lobe downwards into the abdomen. Sometimes, again, a cancerous liver is enlarged without its shape being materially altered, even when it has reached an enormous size.

As above stated, cancerous tumours of the liver are often exceedingly vascular, and their vessels have very thin walls, so that hæmorrhage into the substance of the nodules is far from uncommon. According to Frerichs the extravasation of blood may be so copious as to give rise to a perceptible increase in the size of the tumour. Sometimes, when a vascular cancerous growth is situated just beneath the surface of the liver, the serous membrane covering it gives way, and blood escapes into the peritoneal cavity. A remarkable instance of this once occurred in our pathological theatre, when a large clot covered the surface of the organ. It would seem that the fatal issue is sometimes due to the rupture of the tumour, for patients in whom this has occurred have become collapsed some hours, or in one case three days, before death.

It was shown by Frerichs that cancerous growths in the liver derive their vascular supply mainly from the hepatic artery, and that they receive very little blood from the portal vein. As they increase in size, the trunk of the hepatic artery enlarges, while the area of distribution of the portal vein is diminished. Cancer not infrequently penetrates into the interior of one of the portal branches, and may then extend along its channel so as to obstruct the flow of blood, and cause ascites.

Ascites from cancer of the liver is, however, most often the result of chronic peritonitis which started from the serous covering of the organ, just as pleuritic effusion follows cancer of the mediastinum or lung; or it may be due to one of the secondary nodules pressing on the portal vein.

Enlargement of the spleen is occasionally seen during life or found after death, but it is quite the exception, and if present in a case of jaundice and ascites would be in favour of cirrhosis rather than cancer of the liver (Hawthorne: 'Edin. Med. Journ.,' June, 1901).

An almost constant symptom of cancer of the liver is *pain*, often

severe, and generally accompanied by tenderness on pressure in the right hypochondrium.

Jaundice is sometimes absent, or shows itself only when the case is about to terminate; but it is usually marked, and early in its appearance. In this respect there is a difference between cancer of the liver itself and cancer of the structures in the portal fissure, which is a still more frequent cause of jaundice.

There is sometimes slight *pyrexia*, for which no other cause can be found after death—100° or 101° Fahr., and in one case (that of the boy mentioned on p. 552) it reached 104°—106°. Pyrexia of this degree or pyrexia at all is certainly the exception.*

Diagnosis.—Cancer of the liver is often very easy to recognise. The liver is enlarged, painful, and irregular on its surface. There is sooner or later jaundice, and frequently ascites. The patient is at or beyond middle age, and usually shows signs of grave disease in loss of flesh, anæmia, and a sallow complexion. When such symptoms are present we should look for symptoms of gastric cancer, examine the breasts and uterus in a woman, and the rectum and the testes in a man, with a view to discover the primary seat of the disease.

Sometimes there is considerable difficulty in diagnosis, and this is almost always between cancer and cirrhosis. We have seen (*supra*, p. 535) that in cases of cirrhosis the liver is often enlarged, and can be felt below the ribs; its surface may be uneven, it is often tender to the touch; jaundice is frequently present (p. 511); and ascites may be moderate and late in making its appearance. Moreover intemperance in liquor does not preserve a man from cancer, and cirrhosis may develop itself in or even after middle age. The question, therefore, is sometimes extremely difficult, perhaps insoluble; and, as we have seen, even after death it can sometimes only be decided by the microscope (p. 552).

The *prognosis* is of course hopeless when the nature of the case is clear. The only chance for the patient is in the possibility of an error in diagnosis. Treatment can only be directed, and often with considerable success, to relief of distressing symptoms. It is remarkable how long such cases linger, and even revive for a time, when apparently at the point of death.

Sarcoma is excessively rare as a disease of the liver. When present it is probably always secondary, and sometimes melanotic.

Cavernous angioma has been observed, usually as new growths the size of a marble. Wilks and Moxon regard it as non-malignant.

Cancer of the biliary passages.—The gall-bladder and bile-ducts are frequently the seat of primary carcinoma. Dr Musser, of Philadelphia, published 100 cases collected from various sources in the 121st volume of the 'Boston Medical and Surgical Journal.' The disease is much more common in women than in men; 75—80 per cent. of the recorded cases occurred in women. The patient's age is usually between 55 and 65, and sometimes later still. The exact locality and extent of the growth vary widely. In many cases examined at Guy's Hospital its original seat was

* As Budd long ago remarked, "when tumours grow rapidly, some degree of fever is set up;" and Osler believes that "in all rapidly growing large neoplasms of the liver there is more or less fever, usually continuous and not reaching a high grade." Five cases published by Dr Hawthorne confirm the fact of occasional pyrexia, and—as an exception—of a temperature as high as 104° ('Brit. Med. Journ.,' March 16th, 1901).

the head of the pancreas; in others the pylorus, or the first part of the duodenum. In several it seemed to have begun in the walls of the gall-bladder, and passed downwards until it invaded the common bile-duct; while in others primary cancer of the rectum or colon has affected the glands about the portal fissure, and thus obstructed the flow of bile.

The extent of the cancerous disease, again, is very variable: there may be only a small nodule, no larger than a pea, involving the walls of the common duct; or a complete ring-stricture, like that of the œsophagus or rectum: in the latter case gall-stones are generally present. Occasionally all the parts in the portal fissure may be involved in a mass of cancer, which may spread to the peritoneum as scattered nodules, or lead to the formation of large and numerous secondary growths in the liver. In some of these cases also, gall-stones have been found. Their frequency in cases of cancer of the gall-bladder is stated by Musser at 69, by Courvoisier at 91, and by Siegert at 95 per cent.*

Malignant disease of the biliary passages is almost always true carcinoma, usually with columnar cells in the alveoli, less often with spheroidal, or with both. Sometimes the growth looks hard and dry, and yields little or no juice on scraping; but in one case, although the growth in the portal fissure looked fibrous rather than cancerous, the liver contained large secondary nodules the character of which was unmistakable. In Musser's list three cases of primary sarcoma occur; and Dr Rolleston has since recorded a fourth from St George's Hospital.

Sometimes the new growth forms a papilloma on the surface of the mucous membrane, and thus occludes the cystic duct; sometimes it infiltrates the body of the gall-bladder, and converts it into a tough, thick-walled, contracted cavity; sometimes it invades the opening of the common duct, and spreads to that of the pancreas. Colloid metamorphosis is only occasionally found, and still more rarely hæmorrhage. The cancer spreads most often to the liver, but sometimes to the colon, where it may form a fistulous opening.

Obstructive jaundice is a constant result of this form of malignant disease, and forms one of its most striking and early symptoms.

When there is a mass of cancer about the portal fissure, or in the lesser omentum, the portal vein is almost always pressed upon, and ascites follows. Thus the association of ascites with jaundice is strongly suggestive of malignant growth in the portal fissure of the liver; indeed, with the exceptions of secondary cancer in the liver and of cirrhosis, this is almost the only disease in which these two symptoms are found together.

Clinically we may recognise three forms of hepatic cancer: the numerous nodules secondary to some other growth, the rare primary infiltrated form, and that just described, which begins with jaundice, and is commonly, though not always, followed by ascites and enlargement of the liver.

The diagnosis is sometimes difficult between cancer of the gall-duct and enlargement of the liver from distension by an impacted calculus. Often, however, the difficulty is to decide between cholelithiasis with, and the same condition without cancer.

The only point ascertained in the ætiology of cancer of the gall-bladder is its very frequent association with gall-stones. The association seems too constant to be a mere coincidence of age and sex. It therefore seems

* The two latter figures are quoted from Dr Rolleston's paper ('Clin. Journ.,' April 7th, 1897). No doubt the lists overlap.

probable that the presence of calculi helps to excite cancerous growths as does irritation of the lip or tongue or mamma. But when the growth presses on the bile-duct, it may perhaps favour calculus by causing stagnation in the gall-bladder.

There is no treatment known for this local form of cancer except surgical interference, and this has occasionally proved successful ('Med.-Chir. Trans.,' 1896, vol. lxxix, p. 159).

The remaining structural diseases of the liver are degenerative or adventitious. Like cancer, they do not as a rule interfere with the physiological action of the liver, except by accidental mechanical pressure on its duct, and are for the most part of pathological rather than clinical interest; or if clinically important, it is because a recognition of their nature may lead to the diagnosis of a primary or concomitant lesion elsewhere. In all of them the liver becomes much larger than natural; and this increase in size is often the only indication of disease. They are all unattended with pain.

Simple Hypertrophy.—Dr Fagge once made a *post-mortem* examination of a case in which death occurred three weeks after an accident. The liver was found to project four inches below the ribs, and it weighed 130 ounces—fully double its normal weight. No morbid change could be discovered in the hepatic tissue, so that the case was set down as one of simple hypertrophy of the organ. This affection is recognised by other writers, but at present nothing definite is known of its origin.

In diabetes the liver is usually found larger than natural, sometimes considerably so, but it never approaches the bulk just described.

Enlargement of the liver without true hypertrophy occurs from chronic passive congestion in cardiac disease (*cf.* p. 240), and from distension of the bile-passages by long-continued obstruction of the common duct (p. 513).

Dislocation of the liver.—Mistakes are often made from assuming that if the liver is ascertained by palpation and percussion to reach below the ribs, we have conclusive evidence of its being larger than natural. It may, however, only be displaced downwards by large effusions into the right pleura or the pericardium, or by a subdiaphragmatic abscess (p. 483); or it may be pushed down by tight stays in women or a tight belt in men. Again, as it may tilt backwards and so seem smaller than it is, so it may fall forwards and downwards, so that most of the upper surface comes in contact with the abdominal walls, and thus seem much larger than it is.

Enteroptosis.—Not only the liver, but the spleen and other organs, particularly the right kidney and the stomach, are sometimes found displaced downwards, from their weight not being supported, as it normally is, by the muscular parietes of the abdomen. This condition, as a result of repeated pregnancy, has been long recognised, but of late several extreme cases have been recorded—not all due to pregnancy—by M. Glénard, of Lyons, and other observers, and it has received the name of Enteroptosis or "Glénard's disease."* Sometimes it begins by a loaded colon falling forwards, and dragging the stomach and liver with it; sometimes it starts with a dilated

* 'Société méd. des Hôpitaux,' May 16me, 1886. Among German writers may be cited Dr Haker, of Zürich ('Corresp.-Blatt f. Schweiz. Aerzte,' Juni, 1895); and, among English, Mr Treves, in 'Allbutt's System,' vol. iii, p. 587.

stomach; but probably the most frequent cause is weakening of the abdominal muscles by distension, or by injury, or from want of exercise and "neurasthenia." It is much commoner in women (apart from childbirth) than in men.

LEUCHÆMIC ENLARGEMENT.—In leuchæmia and splenic anæmia the liver often becomes considerably enlarged, owing to the overgrowth of the lymphatic tissue which forms the portal canals; and the spleen is enlarged at the same time. This affection will be noticed in a future chapter.

THE FATTY LIVER.—Another and more frequent cause of the liver becoming enlarged, without pain or other marked symptoms, is loading of its cells with fat. In one of our cases of this kind the organ weighed 7 lbs., and in another it weighed over 11 lbs., or about three times its normal weight. The 'Pathological Transactions' contain a case in which it weighed twelve pounds.

A cirrhotic liver often contains much fat, particularly when it is increased in size; but in primary fatty degeneration the liver remains perfectly smooth. Its edge is somewhat thick and rounded, and on section it is anæmic, and of a more or less yellow colour; but, as Rindfleisch remarked, one must not suppose that it has the same appearance during life, for it can be injected with ease. It is soft, and readily tears beneath the pressure of the finger. Its specific gravity is diminished, sometimes so much that it floats in water. When it is cut into, it greases the knife, and fragments held in the flame of a spirit lamp will sputter, and then burn brightly.

The microscope shows that the accumulation of fat takes place within the hepatic cells, especially in those which lie towards the periphery of the lobules. These often contain drops of oil so large as to obscure their walls, and an inexperienced observer might suppose that the cells had undergone destruction. This, however, is not the case. The oil can be extracted by ether, and the shrunken nucleated cells remain in their natural relation. In fact, the fatty liver of pathologists is only the fat-stored liver of the physiological absorption which follows every full meal; but what in health is intermittent becomes in disease constant and excessive. Possibly the Strassburg goose would lose her *foie gras* if allowed to go free.

As might be expected, a fatty liver can often be easily detected at the bedside. It may be found as low as the umbilicus, or even lower, and the smooth even surface and the soft doughy feel of the edge distinguish this from other enlargements of the organ. The deficiency of resistance may be so great that the liver slips away beneath the hand; one may then have great difficulty in feeling it, even though the parietes are perfectly soft and yielding, and percussion indicates that the organ is much increased in size. This very difficulty, however, points to fatty disease of the liver as the cause of the enlargement.

Ætiology.—The conditions under which the liver is apt to become loaded with fat are numerous, but they may be divided into two main classes, strikingly opposed to one another. In one of these an excess of fat is present in the body generally; in the other there is emaciation, often in an extreme degree.

The first kind of fatty liver occurs, along with general obesity, in persons who lead sedentary lives, who eat large quantities of farinaceous

and saccharine food, and who indulge freely in stimulants. This is evidently analogous to the affection that is artificially produced in geese by the purveyors of the *pâté de foie gras*. The birds are kept in a dark place, with but little space to move in, and are crammed with a farinaceous paste. The consumption of fat within the body is thus reduced to a minimum, while its formation is increased. It first accumulates in the blood, and is then deposited in the hepatic cells.

The other kind of fatty liver cannot be so easily explained. Cases of pulmonary phthisis are those in which it most frequently occurs. This was first noticed by Louis, who found it in one out of every three bodies of those who had died of consumption. At first sight one might be inclined to attribute this to deficient oxidation of fat from interference with the action of the lungs. But if this supposition were correct, the liver ought to become fatty in cases of asthma and of emphysema likewise; whereas, in fact, the other diseases in which it becomes so only resemble phthisis in being attended with wasting—as cancer, ulcer of the stomach, and chronic dysentery. In the course of progressive emaciation, the blood becomes in some way overloaded with fat, which is stored up in the liver; and we may connect the fatty infiltration of the liver with the facts that the patient has generally been bedridden for a long time before death, and has been well fed, often with cod-liver oil. According to Larrey, it is possible by keeping geese shut up in close, hot cages, without food, to induce a fatty enlargement of the liver while the rest of the birds' bodies become wasted.

It is remarkable that the fatty liver of phthisis is more frequent in women than in men.

Symptoms.—A fatty liver produces neither pain nor jaundice. Addison believed that a symptom, suggestive if not pathognomonic of the affection, was a peculiar state of the skin, which he described as looking semi-transparent and pale, somewhat like polished ivory, and as feeling smooth, so as to resemble the softest satin ('Addison's Works,' New Syd. Soc., p. 102). This is observed in the form of fatty liver which accompanies emaciation. On the other hand, when the patient is the subject of obesity, the skin acquires a shining, greasy appearance, apparently due to an excessive secretion of fat by the sebaceous glands.

Hebra noticed that habitual spirit-drinkers have usually a soft, smooth, and clear skin with free and active sebaceous secretion. It may be that this condition is only found when the liver also is fatty, or both may be the results of indulgence in alcohol.

Significance.—In a case of phthisis or other wasting disease, the detection of a fatty liver does not affect the treatment. Nor does it seriously influence the prognosis, since such cases are generally fatal. If the same affection should be detected as a part of general obesity, it is this, and not merely the state of the liver, which calls for interference.

Obesity in general.—This opportunity is the best that offers for the subject of obesity. It is notorious that very fat persons bear even slight accidents ill, and succumb to operations and diseases unattended with danger in others. After death their tissues are found to be soft and flaccid, and to break down under pressure much more readily than usual; and decomposition often advances with undue rapidity. The omentum, the mesentery, and the subperitoneal tissue generally, are loaded with fat. The large size of the abdomen in fact presses up the diaphragm during life, and hampers the

play of the lungs. The heart also is commonly covered with fat, and its substance is soft and lacerable, so that it readily tears, and has been compared to wet brown paper.

Some races tend to obesity, as the Jewish; others to the contrary, as the American. Yet the writer has seen more very fat boys and girls between eight and fourteen in the United States than elsewhere.

A certain amount of obesity is natural in the infant and child up to seven or eight years old, to the girl after the menstrual function is established, to the breeding woman, and to the later period of adult life. It belongs also to all castrated animals, including man. On the other hand the growing boy and youth up to twenty-five is normally and healthily thin, so is the girl between childhood and womanhood; and many of both sexes who become spare and lean will continue in perfect health after fifty-five or sixty. The two types of later life are shown with the exaggeration of rhetoric in the portly justice and the lean pantaloone described by Jacques.

The most important part of the treatment of obesity consists in the regulation of the diet. Thirty years ago popular attention was drawn to this subject by a pamphlet published by Mr Banting, the upholsterer, who, in less than a year, had reduced his weight from 202 to 156 pounds. He was at that time sixty-six years of age, and his height was five feet five inches. Before he began to diet himself he had great difficulty in stooping, was compelled to go downstairs slowly backwards, and used to puff and blow with every exertion, beside being liable to fainting. The articles which he avoided were bread and potatoes, butter, milk, sugar, and beer. He took a liberal supply of animal food, and when he had lost his excess of fat he felt better than he had done for twenty years, and the fainting fits altogether ceased. Dr Harvey, under whose advice the successful treatment was adopted, published the medical aspect of the case in 1872.

Such a change of diet should not be made without supervision by a physician, for it may doubtless be attended with risks of its own. But the dangers which obesity brings with it far outweigh them.

Habits of early rising and of active exercise are useful in preventing the deposition of fat, but active exercise is beyond the power of those who are already corpulent. Even hard riding does not always prevent increasing obesity. Liquor potassæ and other alkaline remedies have been recommended in the treatment of this condition, but they often prove altogether useless. The tendency is sometimes hereditary and insuperable, but walking, moderately restricted diet, and avoidance of beer, with occasional purgation and more frequent sweating, will in most cases succeed in diminishing the patient's bulk and relieving his discomfort. It appears to be more important to avoid carbo-hydrates and sugar than fatty food itself. In severe cases Dr Weir Mitchell's treatment by an exclusive diet of milk and eggs may be resorted to (see Sir D. Duckworth's article on "Obesity" in 'Allbutt's System,' vol. iv, p. 607).

THE MALARIAL LIVER.—Under the influence of repeated attacks of ague, the liver as well as the spleen may become enlarged so as to become palpable below the ribs. In such cases there is frequently some degree of jaundice present, and constantly the yellowish earthy pallor so characteristic of paludal cachexia (cf. vol. i, p. 392). The condition is apparently one of frequently recurring congestion leading to permanent enlargement. It may

result from remittent fevers or (it is said) from residence in a malarious district, even when no febrile symptoms have followed. There is increase of connective tissue, constituting, according to some authors, a special form of cirrhosis; and also, apart from jaundice, deep brown pigmentation of the lobules.

THE LARDACEOUS LIVER.—Prolonged suppuration, and occasionally syphilis without suppuration, leads to the conversion of the liver, spleen, kidneys, and many other viscera, into a peculiar translucent material, which is known by the epithets lardaceous, waxy, albuminous, or amyloid. Its chemistry and pathology will be described in the chapter on Bright's disease.

A lardaceous liver often reaches a great size. In one case at Guy's Hospital it weighed over eight pounds, and in another fourteen. Its density is much increased; Wilks mentions an instance in which the specific gravity was 1084. Its cut surface looks dry and bloodless, smooth, shining, and translucent; and it can be cut into thin sections without preliminary hardening. Iodine gives its characteristic reaction, as does also methyl violet.

The microscope shows that the capillaries are converted into lardaceous material. The earliest affected are those which lie in the zone of each lobule, intermediate between its centre and circumference. This position corresponds with the distribution of the hepatic artery; and here, in the muscular coat, the degeneration begins; but it gradually extends inwards to the centre of the lobule, and finally outwards to its periphery.

Duckworth has recorded cases in which the lardaceous liver became reduced in size. In one case, after reaching nearly to the pubes, it became in fifteen months about half the size it was, and at the autopsy weighed only 117 ounces ('St. Barth. Hosp. Rep.,' vol. x, p. 57, 1874).

As with fatty and leucæmic enlargement, there is neither pain nor ascites nor icterus. The disease is recognised by its physical signs alone.

The liver often reaches down to the level of the umbilicus. During life the edge can generally be felt very distinctly, more readily than that of a fatty liver, for it is firm and resisting. The surface of the organ is perfectly smooth, unless gummata be also present; but such a combination is sufficiently common to be borne in mind.

In practice, diagnosis of a lardaceous liver depends on our knowledge of its ætiology. We expect to find it in cases of disease of the bones or of the chest, accompanied with long-continued suppuration, and particularly in syphilitic cases; and we are confirmed in our conclusion if we find evidence of the same affection of the spleen, the kidneys, or the intestines.

TUBERCLE OF THE LIVER.—It is not uncommon in cases of adult tuberculosis, and very common in children, to find the liver full of miliary tubercles, usually yellow and more numerous on the surface than elsewhere; with similar tubercles of the spleen and kidneys. They probably appear not long before death, and have little or no clinical significance, although cases of jaundice from this cause have been recorded (p. 510).

Tuberculous cirrhosis has been described (p. 537), and some pathologists would perhaps have reckoned under this head a case recorded by the writer in a boy of thirteen ('Path. Trans.,' 1882). Miliary tubercles are not infrequently found in a cirrhotic liver, as well as elsewhere—but per-

haps not more frequently than to show that neither drink nor local cirrhosis protects from tuberculosis.

A large caseous tubercular mass softening into a pseudo-abscess or vomica, like the cavities of pulmonary phthisis or the tubercular growths of the brain or the kidney, is one of the rarest pathological curiosities. The hepatic vomicae referred to by Celsus (ii, 80) were no doubt abscesses; and the small number of such hepatic vomicae which have been recorded probably include cases of Actinomyces (cf. vol. i, p. 431). The writer, however, once met with a specimen of genuine caseous tuberculous vomica in the liver, as large as a marble, in a case of ordinary phthisis.

HYDATIDS OF THE LIVER.—In all the forms of painless enlargement of the liver hitherto mentioned, the organ is uniformly increased in size. In this respect they differ from a hydatid tumour, which is a rounded elastic swelling of only part of the liver. It may reach a considerable size before it is detected, and it often causes not the slightest discomfort to the patient.

The natural history and development of the echinococcus which produces hydatid tumours of the liver have been fully treated in a former chapter (p. 457 *seqq.*).

Anatomy.—A hydatid cyst of the liver forms a more or less globular mass, varying in size from that of a walnut to that of a cocoanut, or even larger; the largest on record is said to be one weighing thirty pounds, which was observed by Luschka. If it is subjected to no pressure in its growth, its form is spherical; but if it meets with more resistance on one side than on another, it may be flattened or egg-shaped.

In some cases it would seem that the six-hooked embryo originally lay just beneath the serous covering of the liver; and the hydatid may then form a globular mass depending from its surface. In other cases a great part of the sphere formed by the hydatid may lie within the hepatic substance; and its presence may be only indicated by a rounded projection. Dr Fagge recorded a case in which a hydatid reached both surfaces of the liver at once; and the original anterior edge of the liver was distinctly made out as a narrow ridge, passing obliquely downwards and to the right across the rounded tumour, which occupied the epigastric and hypochondriac regions. Lastly, a hydatid may be embedded entirely in the back part of the liver, or reach only that portion of its surface which is in contact with the diaphragm, and covered by the ribs.

Signs.—The tumour may be quite soft, and fluctuation may readily be detected, a wave being transmitted from one part of it to another; or it may be firm and tense, sometimes of stony hardness. In a certain proportion of cases a peculiar sensation may be elicited by percussion, which Briançon first described, and termed the *frémissement hydatique*.

The way to detect it is to place three fingers of the left hand upon the tumour, and then to tap the middle finger abruptly with the right forefinger. The other fingers of the left hand may then perceive a peculiar quivering sensation. This is not caused by the vibration of the daughter-cysts contained in the hydatid, for it may occur with cysts in which there is nothing but fluid; and it is far from decisive of the cyst being due to an echinococcus.

Diagnosis.—As a rule, the detection of a hydatid tumour, lying below the ribs, is not difficult. If the cyst should project far from the lower

edge of the liver it may be mistaken for a distended *gall-bladder*, or even for hydronephrosis. Distension of the gall-bladder, without jaundice—the common bile-duct being patent—is, however, exceedingly rare; and in *hydronephrosis* the tumour fills the lumbar region more than would a hydatid; moreover, the colon is generally to be detected in front of it by percussion, whereas the downward growth of an hepatic cyst would push the gut backwards.

When the cyst is deeply embedded in the substance of the liver, so that it is but little raised above the surface of the organ, and yet causes it to project a long way down into the abdomen, there is often great difficulty in determining the nature of the case. Dr Fagge met with several instances when what was supposed to be a hydatid tumour proved to be due to lardaceous disease of the liver, and the circumscribed tumour to its being intersected by fibrous bands, in connection with *syphilitic gummata*. One diagnostic character of syphiloma of the liver is immobility of the organ during inspiration, due to the adhesions of its surface, which are generally present. More or less pain and tenderness on pressure are also common symptoms in such cases.*

In other cases there may be a difficulty in determining whether a tumour of the liver is a hydatid or a *cancerous growth*. The distinction must be based partly on the physical character of the tumour, partly on the presence or absence of general symptoms, particularly pain. It must, however, be admitted that pain is not invariably absent in hydatid disease. Frerichs gives a case in which an hepatic cyst was the seat of violent pains after every manipulation, and they ceased almost immediately after tapping. Moreover, the capsule of a hydatid may inflame and suppurate, and thus cause severe pain. The health of patients harbouring this parasite often appears to be perfect; but they may suffer much inconvenience from its pressure on neighbouring organs.

Sometimes, as has already been stated, an echinococcus growing in the liver, instead of forming a tumour that can be felt in the abdomen, may project from the phrenic surface of the organ, under cover of the ribs. If it should attain a considerable size it may then cause the lower part of the chest to bulge, and the edges of the costal cartilages to form a much more open curve than on the opposite side of the body. The intercostal spaces over the swelling may feel more resistant than usual, and may even project beyond the level of the ribs. At the same time the lower part of the chest yields a dull note on percussion, so that the case is very likely to be mistaken for one of *chronic pleural effusion*. Such an error may, however, be always avoided by careful observation of the limits within which the dulness on percussion and the enlargement of the intercostal spaces can alone be detected. In cases of hydatid tumour below the diaphragm, the area of dulness is bounded above by a curved line, which descends as it approaches the spine posteriorly. In cases of pleuritic effusion the dulness reaches to quite as high a level in the dorsal region, close to the spine, as in the neighbourhood of the right nipple.

I once saw a case with Mr. Durham, which well illustrates this distinction. The patient, a young lady, had been sent to him by a physician, who considered her to be suffer-

* It once happened to me to direct the performance of exploratory operations in two cases on the same day, and in each of them the tumour proved to be solid; it was probably in both cases a syphilitic and lardaceous liver. These patients did well; but a third died from chloroform while undergoing an operation for a supposed hydatid of the liver, and in this case also the tumour was found to be of the same nature.—C. H. F.

ing from a chronic pleuritic effusion, the result of an attack of pleurisy two or three months before. The right lower ribs, in the lateral region of the chest, were bulging, the intercostal spaces were tense, and they seemed to yield a sensation of fluctuation. There was increased dulness on percussion over the same part; but in the back the physical signs were in all respects normal. In spite of the history we agreed that the case was one of echinococcus in the liver; and the aspirator proved it to be so.—C. H. F.

Whatever doubt there may be as to the nature of a cystic tumour of the liver, it is quickly set at rest by finding hooklets under the microscope, or by the chemical examination of the liquid removed by paracentesis (cf. *supra*, p. 459).

Suppurating hydatid.—Inflammation within an echinococcus cyst is not an uncommon occurrence, and one which is very important, since it modifies greatly both the physical signs of the affection and its symptoms. If the tumour can be seen or felt in the abdomen, it becomes painful and tender and hot, and there may after a while be redness of the skin, with attacks of shivering, and symptoms of hectic. In some cases, however, suppuration may apparently take place without marked symptoms.

When a dead hydatid is found in the liver at an autopsy, it generally contains a putty-like substance, made up in large part of calcareous salts, and mixed with the gelatinous relics of hydatid membranes, which often glisten with cholesterine crystals. The putty-like substance is very like that which occurs in a dried-up abscess, and in all probability is transformed pus within the capsule of the hydatid—the cause of the death of the parasite or its consequence. However changed the other parts of the parasitic growth may be, the microscope will always find the hooklets unaltered.

Rupture of the cyst.—A living echinococcus may burst its capsule and pour out its contents, or a suppurating hydatid may ulcerate and open in various directions. In the former case suppuration of the cavity follows, and thus, unless the death of the patient has immediately followed, one cannot determine whether the parasite was alive or not at the time when the cyst gave way.

A suppurating hydatid cyst sometimes, but very rarely, makes its way through the abdominal *parietes*. It is said that this may happen even when the parasite is still alive, and cause a discharge of clear water.

Another direction in which rupture may take place is into the *peritoneum*, and this is often the result of some injury to the abdomen, such as the patient's falling downstairs. Fatal peritonitis generally follows quickly upon an accident of this kind; but unless it is previously known that the patient had a hydatid tumour, it is of course impossible to say why the injury caused such severe symptoms. One or two cases, however, have been recorded in which rupture of a hydatid cyst into the peritoneal cavity seems to have taken place without the patient having been much the worse for it, although there was for some time afterwards fluctuation in the lower part of the abdomen, just as in ordinary ascites. In all probability the different results of rupture depend upon the circumstance that sometimes a large number of daughter-cysts and scolices are effused into the peritoneal cavity, but that in other instances only the hydatid fluid is extravasated, either from the hydatid cyst being sterile, or from the aperture being small and the escape of the contents gradual. It is in these cases that the curious symptom of urticaria has been observed to follow.

Much more commonly a hydatid cyst discharges its contents into either the *stomach* or *intestines*. The daughter-cysts are then vomited or dis-

charged *per anum*; and sometimes gas enters the tumour, which thus becomes tympanitic on percussion. The evacuation of membranous portions of hydatids in the fæces sometimes goes on for several weeks or longer; and in the majority of cases the patient ultimately recovers.

The rupture of a hydatid cyst into the *biliary passages* may be a cause of obstruction, and often of febrile jaundice (p. 510).

Occasionally the tumour bursts not downwards, but upwards, into the chest; in these cases its seat is almost always in the upper and posterior part of the liver, so that frequently no positive physical signs of its presence can be discovered either before or after its rupture.

Sometimes a hydatid cyst discharges its contents through the diaphragm into the *pericardium*; and sometimes into one of the hepatic veins within the liver, when the daughter-cysts pass straight into the right chambers of the *heart*, and plug the pulmonary artery. In both cases sudden death follows—or, at least, rapidly fatal syncope—in a person who perhaps has hitherto appeared to be in perfect health.

In rare instances, again, a hydatid tumour has been known to discharge its contents into one of the *pleural cavities* (generally the right), with the result of setting up a severe and rapidly fatal pleurisy. But far more frequently, when the diaphragm is perforated by a hydatid, the pleura has previously become adherent. The consequence is that the parasite makes its way into the substance of the *lung*, and sooner or later reaches a bronchial tube, into which it opens, so that its contents are expectorated.

Many years ago a boy aged six, a patient of the late Mr Fagge, of Hythe, had suffered for about eighteen months a pain just outside the right nipple, and a constant hacking cough, for which all treatment was useless. He became exceedingly wasted, and was supposed to be sinking. One day his cough left him, and he became exceedingly prostrate, but next morning the cough returned, and he spat up a hydatid cyst and a quantity of pus. From that time he began to recover, and his cough gradually disappeared. When he died of another disease, it was proved that the hydatid had begun in the liver.—C. H. F.

A young woman was once attending among the out-patients who had been expectorating hydatids for nearly a year when she first came to the hospital. Next week she had a most violent attack of coughing, which lasted three hours, and it seemed she would be choked. But at last she got rid of a large piece of hydatid membrane, which was apparently a part or the whole of the mother-cyst; for she coughed up no more hydatids, and to a great extent regained her health.—C. H. F.

In a case under the late Dr Barlow's care, when the writer was his clinical assistant, a young woman was admitted to Miriam Ward with jaundice, enlarged liver and pyrexia. After a few days she was seized with pleurisy on the right side, and then with expectoration of blood and pus, under which she rapidly sank. No traces of hydatids were found in the sputum; but Dr Barlow supposed that a hydatid cyst in the liver had suppurated and perforated the diaphragm, so as to set up first pleurisy and then suppuration of the lung. This diagnosis was confirmed after death, the original cavity being found in the liver and a large hydatid cyst unruptured in the lung.

When portions of hydatid membrane from the liver are expectorated they are generally colourless, but sometimes deeply stained with bile. In some instances the patient regains his health when all the hydatids have been voided, and the capsule has contracted so as to close the cavity; but this process is not free from risk.

In London and Dublin hydatids are less frequently found in the liver than formerly,* and in Edinburgh they are exceedingly rare. In the United States and also in the East Indies the disease is said to be very uncommon. On the other hand, in Australia it is far more frequent than in England, and among the inhabitants of Iceland it causes one seventh of the total mortality.

* For an account of hydatids in other organs, *v. supra*, p. 460.

Prophylaxis.—The easiest precautionary measure against infection with hydatids is taking care not to eat raw vegetables, unless they have been thoroughly cleansed before coming to the table.

According to our present knowledge, this parasite would soon become extinct if its cystic form infested no other animal than man. Dogs acquire the *Tænia echinococcus* only by eating the flesh of some creature in which the scolex is embedded, and in civilised countries they have no opportunity of deriving it from the dead human body. Sheep and pigs are believed to be the chief animals beside man which harbour hydatids. Consequently dogs should be prevented eating the offal of these animals, and should be excluded from all slaughter-houses. It has also been advised that the floor of every kennel should be frequently scalded with boiling water, so as to destroy any ova of the *Tænia echinococcus* that may have been voided with the dog's fæces.

Curative treatment of hydatid tumours belongs to surgery rather than to medicine. At one time it was thought that the internal administration of iodide of potassium would poison the echinococcus and lead to the disappearance of a hydatid cyst; but we have no drug that can exert this power, and at the present day paracentesis is attended with little risk to the patient. An operation should therefore at once be performed when a hydatid is detected, and all the more if it is increasing in size.

Part of the fluid may be withdrawn by a grooved needle, a subcutaneous syringe, or an aspirator; and, as a rule, this is followed by the death of the parasite and the shrinking of the cyst.* To do more is generally useless, and to exert forcible suction by an aspirator upon a cyst surrounded by solid tissue involves some risk.

Erythema or urticaria sometimes follows paracentesis, probably from escape of some of the fluid into the peritoneal cavity.

It often happens that, some weeks or even months after tapping, the tumour is found to have regained its former size, or even to exceed it. If paracentesis is repeated, the liquid obtained differs from hydatid fluid in containing more or less albumen; for the increase of the tumour is due not to the continued life of the parasite, but to the effusion of serum within its capsule. Indeed, after a second or third operation this serum generally contains leucocytes, and so gradually passes into pus. If, however, the real cause of the enlargement of a hydatid tumour after tapping be recognised, and if further interference be abstained from, it sooner or later decreases in size again, and finally disappears.

The retrograde changes which lead to the ultimate disappearance of such tumours are no doubt very slow in their progress.

A patient of Dr Moxon's once lay for several weeks in the clinical ward in bed on account of a pain in the hepatic region, which had come on some time after the performance of an operation for a hydatid tumour. The tumour itself could no longer be detected, but the pain was intense and very obstinate. Doubtless some of the sensitive structures of the abdomen were subjected to traction by the shrinking of the capsule of the cyst.—C. H. F.

When suppuration has occurred within a hydatid cyst, or perhaps in any case if the tumour is very large, it should at once be opened with due

* The operation of *electrolysis* was practised several times at Guy's Hospital by Fagge and Durham, who published in the 'Medico-Chirurgical Transactions' for 1871 the reports of eight cases, in seven of which (if not in all) this treatment was completely successful. At first this success was attributed to the decomposition of the saline liquid contained in the cyst; but it was no doubt due to the necessary withdrawal of some of the hydatid fluid.

antiseptic precautions, the cyst having been first fixed by sutures to the abdominal wall so as to obviate the risk of extravasation. A large trocar should be used, and if the tumour contains secondary cysts, as many of them as possible should be removed. A drainage-tube is then inserted, and the cavity is washed out every day. The obliteration of the cavity is always a very slow process, and not unattended with risk; but with proper management cases of this kind usually end favourably. An enormous multiple hydatid cyst, in a patient under the writer's care in February, 1886, was opened by Mr Jacobson with complete success, although innumerable echinococci continued to escape for many weeks.

Simple *retention-cysts* containing serum are occasionally met with in the liver; but they appear never to reach such a size as would enable them to be detected during life, and they are of merely pathological interest.

They sometimes occur in the broad ligament, as in a case noted in 1864 by the writer, when working in Virchow's pathological laboratory.

Microscopic cysts of the liver are sometimes met with in great numbers. They are probably not retention-cysts, but the result of vacuolation of the protoplasm of separate secreting cells, as was first stated by Dr Beale (*vide* 'Path. Trans.,' vol. vii, p. 234; see also vol. xxxii, pl. xvii, fig. 2). On this point, however, there is still difference of opinion.

DISEASES OF THE KIDNEYS

FUNCTIONAL DISORDERS

Falstaff. Sirrah, you giant, what says the doctor to my water?

Page. He said, sir, the water itself was a good healthy water; but for the party that owned it, he might have more diseases than he knew for.

1 *K. Henry IV.*

History—Arrangement adopted—Polyuria and oliguria; high and low specific gravity—Baruria and Hydruria—Diabetes insipidus—Renal inadequacy.

Reaction—overacid—alkaline—ammoniacal urine.

Urinary pigments: uro-chrome—uro-erythrin—haematoporphyrin—urobilin and indoxyl—chromogens—Alcaptonuria, etc.

Lithic (Uric) acid and lithates—Cystine—Oxalate of calcium—Phosphatic deposits—Calcic sulphate—Infected and putrid urine—Bacilluria.

Hæmoglobinuria—General characters—Toxic form—Infantile form—Paroxysmal form—its relation to malaria—Blackwater fever—Raynaud's disease.

IN the sixteenth and seventeenth centuries the inspection of the urine was a regular part of the physician's office, as is proved by many passages of the Elizabethan dramatists and many scenes of the great Dutch painters.* But the examination was as useless as the Chinese examination of the pulse, until modern chemistry made in some degree clear the causes of the colour, turbidity, or transparency of the secretion.

The first chemical step was to distinguish the deposits or precipitates which come down in turbid urine, after standing, into those consisting of blood, pus, and other organised matters, on the one hand, and those consisting of saline compounds on the other; and the latter, again, into red gravel, soluble in alkalis, and white gravel soluble in acids.

Next, the presence in some dropsical cases, of a substance which, like white of egg, coagulates on heating was established, though it was at first supposed to be due to an effort of nature to get rid of the dropsical serum through the kidneys.

The discovery of lithic (or uric) acid in calculi and in red gravel by Scheele, in 1776, was the next step. This was preceded by Rouelle's dis-

* For example, the beautiful "Visit of the physician," by Gerard Dow, in the *Salon carré* of the Louvre.

covery of urea in 1773; and by that of the earthy phosphates in white gravel. The discovery of blood-discs and pus-cells in morbid urine, and the description of the various crystalline and amorphous deposits and their chemical characters was due to the investigations of Prout, Bence Jones, and Golding-Bird in England, and of Rayer and many others in France and Germany.

The merit of Richard Bright was in connecting chemical examination of the urine with the morbid anatomy of the kidney. His discovery of the connection between dropsy, albuminuria, and nephritis, published in 1831, was followed up by his assistants Barlow and Rees, and by his successor Wilks, to whom was due the broad and lasting distinction between the large red kidney, the large white kidney, and the small red kidney. The late George Johnson mistook the second of these conditions (Bright's smooth mottled kidney) for fatty degeneration, but to him is owing the important recognition of the varied character, and significance of tube-casts in the urine as signs of the state of the kidneys.* To Virchow is due the discovery of the lardaceous form of Bright's disease and the capital distinction between parenchymatous or tubular and interstitial or cirrhotic nephritis; to Traube the exploration of the effect of the disease upon the circulation, which Bright had himself observed, and to von Gräfe the recognition of retinal hæmorrhage and albuminuric retinitis in morbus Brightii. Perhaps the most important new observations since Bright's time are those of Gull and Sutton on the wide-spread "arterio-capillary fibrosis" of chronic nephritis, and the remarkable results as to excretion of urea which Dr Bradford has obtained by the removal of renal substance in dogs. Christison's work on the granular kidney followed shortly after Bright's and Rayer's; and Bartels in Germany, Grainger Stewart in Edinburgh, and Dickinson in London, have done much to complete the pathology of the kidneys.

Though many important gaps in our knowledge remain, we recognise the principal normal and abnormal constituents of urine, and can determine their relative amount. In particular, we know what urinary conditions are due to physiological changes in the body, and what are purely chemical and take place after the urine is voided.

In dealing with the affections of the kidneys, we will begin with their functional disorders, and afterwards pass on to the forms of nephritis comprised under the title Morbus Brightii, of which the common symptom is albuminuria. Then will follow suppuration of the kidney and pyelitis, with the effects of obstruction by calculi or otherwise, affections characterised by the presence of blood or pus in the urine.

The concluding chapter will treat of tubercle and new growths of the kidney, and of renal parasites.

Diabetes, a disease in which the urine contains sugar, but which is not in any other sense a disease of the kidneys, has been discussed separately (vol. i, p. 435).

The present chapter deals with certain morbid conditions of the urine which are independent of any organic lesion of the kidneys, and are characterised by some alteration in its *quantity, density, colour, or reaction*, by the formation of *precipitates* or deposits, by the occurrence of sepsis and decomposition, or by the appearance of *hæmoglobin* in solution.

* The presence of these tube-casts or urinary cylinders in morbid urine was first noticed by the anatomist Henle.

There are many morbid states of the urine, such as the absence of chlorides in pneumonia, and the presence of bile-pigment in jaundice, or of leucin and tyrosin in acute yellow atrophy of the liver, which have already been described under the diseases of which they are symptoms, and require no further account in this place. The important condition of albuminuria will be found described under Bright's disease (*infra*, p. 606).

Our present task is to give an account of the changes in the urine which are primary pathological processes rather than symptoms of organic disease.

Changes in the quantity and specific gravity.—Between the amount and the density of the urine passed, there is an inverse proportion. In health, if the urine is unusually abundant, it is always pale and watery; if it is scanty, it is dark and of high specific gravity. Among diseases, diabetes is the only one in which pale and abundant urine is of great density; and a diminished flow of urine is not likely to be accompanied with a low specific gravity except in the last stages of Bright's disease.

In health, the daily amount of urine ranges in different persons and under different circumstances from forty to fifty ounces or more; it is liable to great variation from day to day; and some perfectly healthy people habitually void either larger or smaller quantities than others. The specific gravity of the collected twenty-four hours' urine is very near to 1020; but after drinking water freely (whether the pure element or in any form of liquor), single specimens will yield a much lower specific gravity, 1005 or 1001, and the effect of a meal is always to lower the specific gravity. Fasting, active exercise, warm weather, and abundant perspiration cause scanty and dark urine, as does an attack of diarrhoea. Food and drink, cold east winds, and diminished perspiration make the urine abundant, pale, and watery. The urine passed under nervous excitement (as often in the case of a candidate for insurance) is pale and of very low specific gravity, like that passed after a hysterical fit.

The quantity and density of the urine depend mainly upon the activity of the blood-current in the renal glomeruli. According to Ludwig the determining factor is the pressure of the blood within the vessels of the tufts; according to Heidenhain it is the rapidity of its passage through them.

The only local cause of obstruction of the renal veins is thrombosis of these veins, or of the inferior vena cava above their mouths. Thrombosis of the renal veins is not very infrequent as a complication of lardaceous and other forms of Bright's disease, and Moxon recorded in the 'Guy's Hospital Reports' for 1869 two cases in which it was associated with injuries to the lumbar spine. But in the former class of cases the existence of lesions in the renal cortex makes it impossible to determine the effect of the thrombosis upon the characters of the renal secretion; moreover, as the obstruction is probably developed very slowly and gradually, collateral channels have time to enlarge and can carry on the circulation. In both of Moxon's cases the arteries were plugged as well as the veins, so that the urinary secretion was entirely suppressed. Bartels related in 'Ziems-sen's Handbuch' a case in which the inferior vena cava was closed by a thrombus, from the point where it passes along the groove in the back of the liver, downwards; in that instance, however, the urine, which contained blood and albumen, was secreted in fair quantity, and was of sp. gr. 1011 to 1013.

When the systemic venous circulation generally is obstructed, as in cases of heart disease and of pulmonary emphysema, the urine is almost constantly found to be scanty and of high density. The pressure in the systemic arteries is, in these conditions, always lowered. When, under the influence of digitalis, the blood-pressure in the glomeruli can be brought up to a sufficient point, it is surprising how greatly the flow of urine increases.

One not infrequently sees glistening crystals of nitrate of urea form in urine to which nitric acid has been added; but this is due to defect of water, not to excess of urea.

Prout thought that he recognised a disease, which has since been termed *azoturia* or *baruria*, the principal symptom of which was an increase in the excretion of urea. In cases which have been placed under this category the flow of urine has been generally excessive; but they appear to have been wrongly interpreted. Urea is a powerful diuretic, and its quantity varies with the amount of nitrogenous food; moreover, the specific gravity of the urine depends on the salts as well as on the urea excreted, and also on the activity of the skin and lungs, and the amount of water taken in. Dr Ralfe relates a striking case of this condition, polyuria with excess of urea, which proved suddenly fatal, but no lesion was found at the autopsy ('Allbutt's System,' vol. iii, p. 251).

An opposite state of urine, in which, while the quantity is augmented, the density is reduced, is of frequent occurrence. We shall find it to be an important symptom of certain forms of Bright's disease, in which there is an abnormally high arterial tension. But it is also seen as an independent condition, which has been called polyuria, or hydruria, the term azoturia being applied to cases of polyuria with excess of urea, and phosphaturia to polyuria with excess of phosphates, a condition which, according to M. Tessin, of Lyons, is occasionally fatal.

DIABETES INSIPIDUS.*—In this disease, if disease it is to be called, the patient passes enormous quantities of urine, exceeding even those that are voided in saccharine or "true" diabetes itself. Apart from the absence of sugar, the specific gravity at once distinguishes between the two affections, for in diabetes insipidus it is often scarcely above that of water, and seldom reaches higher than from 1003 to 1007. The daily secretion of urine may range from fifteen to thirty and even forty pints. It is clear, and almost, if not quite, colourless; it has a faintly acid reaction, but early undergoes the ammoniacal fermentation. It of course contains a very small proportion of solid matters, but nevertheless the total daily amount of urea excreted appears to be rather excessive than diminished.

One abnormal constituent, *inosite*, or "muscle-sugar" $(\text{CH.OH})_6$ —so called from its sweetness, but chemically belonging to the aromatic, not the saccharine group—has been sometimes detected in the urine of diabetes insipidus;† but it is often absent, and it sometimes occurs in saccharine

* *Synonyms.*—Pseudo-diabetes — Polydipsia — Polyuria — Hydruria — Diuresis. The Greek word διαβήτης (*i. e.* a siphon, so called from its resemblance to a pair of compasses, from διαβαίνω, to straddle) was applied by Aretæus and Galen to the condition in which whatever a man drinks runs through him as through a siphon. Most of such cases were no doubt saccharine diabetes, others were chronic Bright's disease. The discovery that in many of them sugar is present was made by Thomas Willis in 1670, and from that time *diabetes insipidus* has been distinguished from *diabetes mellitus*.

† Inosite occurs in many unripe fruits (as French beans, whence it has been called

diabetes, in Bright's disease, and in other cases of polyuria. It forms crystals, is soluble, non-fermentable, and has no effect on the polarised ray. Since inosite is to be found in small quantity in the heart and other muscles (as also in the lungs, liver, spleen, and other organs), its excretion in the urine may be the result of the excessive transudation of water through the tissues; and Strauss is said to have discovered it in the urine of three healthy persons, who, for the purpose of experiment, had drunk a large quantity of water.*

Symptoms.—A tormenting thirst is a constant sign of the disorder. At one time it was imagined that this might be the essential feature of the disease, which should therefore be properly regarded as a "polydipsia" rather than a "polyuria." But experience has shown that the patients always pass more urine than healthy persons who drink the same quantities of fluid; moreover, when in a case of diabetes insipidus the amount of drink is restricted, the urine does not fall in the same proportion, and the tissues become dry.

In some instances the general health remains wonderfully good. Sir William Roberts cites the case of a farm labourer, aged fifty-one, who had been affected for twenty-four years, drinking from thirty-two to thirty-six pints of water daily, and voiding urine in proportion, and who yet remained able to do all kinds of hard work, such as threshing and mowing. And another case is recorded of a woman who bore eleven children while suffering from the disease. It is especially noted that the farm labourer's skin was moist, and that he perspired freely when at work. As a rule, the skin is obviously dry and harsh; and Roberts himself had under his care a boy, who, although rosy and plump, had a dry skin and tongue.

Most patients complain that their rest at night is disturbed by the frequent desire to micturate. Other symptoms are, according to Roberts, a painful dryness and heat of the mouth and fauces, pains in the loins and in the epigastrium, an indifferent or sometimes a voracious appetite, enfeeblement of bodily strength and of mental vigour, irritability of temper, and abolition of the sexual functions. Senator, in 'Ziemssen's Handbuch,' states that the temperature of the body is slightly lowered, possibly from the large quantity of water that is swallowed having a cooling effect. Enforced abstinence from drink aggravates most of the symptoms; the skin then becomes hot, there is an intolerable sense of sinking, or intense pain at the pit of the stomach, and at last the intellect becomes impaired. Sir Thomas Watson relates the case of a boy aged eleven, who was limited during twenty-four hours to drinking a pint and a half of fluid, and who nevertheless passed ten and a half pints of urine. That he absorbed water from the air seemed to be clear from the result of weighing him at short intervals. The polydipsia and diuresis lasted for three years, and the boy died of "scrofulous tubercles" in the brain and lungs.

phaseolo- or phaséo-mannite). It is crystalline, soluble in water, and sweet like glycose, but does not reduce copper or rotate the polarized ray. Scherer's test for inosite in solution consists in treating with nitric acid, evaporating cautiously to dryness, moistening the residue with ammonia and solution of chloride of calcium, and evaporating again, when a rose colour makes its appearance. This test only succeeds with nearly pure inosite; and therefore to detect it in urine Gamgee recommends Gallois's test of evaporating and treating with solution of mercuric nitrate and evaporating again, when a rose colour appears which fades on cooling and returns with heat ('Phys. Chem.,' vol. i, p. 338).

* See an interesting paper by Dr Edsall on the physiological effect of large draughts of water, in the reports of the William Pepper Laboratory in Philadelphia (1900, p. 368).

Diagnosis.—Diabetes insipidus is not a common disorder. In London hospital practice it is decidedly rare; and with regard to published statistics there is a doubt whether they are not more or less vitiated by the inclusion of cases in which Bright's disease would have been found present if an autopsy had been made. Sir William Roberts, indeed, avowedly places in his collection of seventy-seven cases three which ended fatally, although in each of them the kidneys were affected with a marked degree of atrophy in association with hydronephrosis, *i. e.* "consecutive Bright's disease." It is clearly important to distinguish from diabetes insipidus all cases in which the polyuria is a symptom of an organic lesion of the kidneys. But it is possible that in some of the cases in question (two of which were published by Sir Peter Eade, of Norwich) the hydronephrosis may have been a secondary result of the frequent micturition, just as dilatation of the bladder may be a consequence of saccharine diabetes.

The clinical diagnosis of diabetes insipidus must therefore always be somewhat uncertain in persons over fifty, for renal cirrhosis comes on insidiously and without much albuminuria. Probably, some mistakes might be prevented by careful observation of the state of the arterial tension, which in diabetes insipidus appears to be lowered rather than raised. Even in young subjects one must not overlook the possibility of the presence of hydronephrosis from calculous disease.

Ætiology.—Among Roberts's cases there were seven in which diabetes insipidus was said to have begun in infancy, or from the time of birth, fifteen in which it began between the ages of five and ten years, thirteen between ten and twenty years, sixteen between twenty and thirty years, fifteen between thirty and fifty, and four between fifty and seventy. Males bore to females the proportion of five to two.

Occasionally there is a well-marked history of the occurrence of the disease in several members of the same family; of this a striking example was recorded by Lacombe, in which a mother, her three sons, her daughter, her brother and his children were affected in turn. In some cases diabetes insipidus has been attributed to a blow or fall upon the head, or to a tumour of the brain.* Roberts remarks that in some of the traumatic cases polyuria set in with its maximum intensity on the day of the accident, but in others a few days later, and in one case not until six months had elapsed.

A condition like diabetes insipidus can be produced experimentally in animals. Bernard first showed that this effect followed puncture of the floor of the fourth ventricle at a point a little above the glycosuric centre. There is also experimental evidence of an excessive flow of urine being produced by irritation of the cervical sympathetic and by lesions of the spinal cord. The probability seems to be that the immediate cause of the affection is a dilatation of the renal arteries, from defect of the controlling action of their vaso-motor nerves. So far as is yet known, there are no renal nerve-fibres which are secretory—in Heidenhain's sense, *i. e.* trophic, affecting the secreting epithelium. It is worthy of mention that, in a case observed by Külz, diabetes insipidus was accompanied by spontaneous and persistent ptyalism (the patient spitting from twelve to eighteen ounces of saliva daily); for this is another symptom that has been produced in animals by puncture of the floor of the fourth ventricle.

Prognosis and treatment.—Diabetes insipidus varies widely in its onset

* See Dr Alexander Hughes Bennett's case, 'Brit. Med. Journ.,' Feb. 24th, 1883.

and course. Roberts cites one instance in which it set in with absolute suddenness: the patient, a woman aged thirty-four, went to her work one morning at six o'clock in her usual health; two hours later she was seized with intense thirst and diuresis, which continued from that time.*

In those cases which follow injuries to the head, the affection commonly subsides in the course of a few weeks or months, but cases are on record in which it had lasted six or seven years; and of the non-traumatic cases beginning from infancy, some are stated to have run on for fifty years or more. When there is a cerebral tumour, this is of course almost sure to prove fatal in a comparatively short time; but diabetes insipidus does not in itself appear to have much tendency to destroy life. In one of Roberts's cases the patient, a man aged sixty-two, is said to have suffered for twenty years from excessive thirst and diuresis. Fatal cases directly from diabetes insipidus appear to be very rare; the patient generally succumbs to an intercurrent malady, such as phthisis or pneumonia.

It is a remarkable fact that the occurrence of some inflammatory or febrile disease has in several cases led to the temporary, or even permanent, subsidence of diabetes insipidus. Thus one patient, who had suffered from it for eighteen years, recovered completely after acute rheumatism; and another after an attack of pleurisy.

In treatment, the most useful drug seems to be valerian, which was prescribed by Trousseau in enormous doses, two and a half drachms of the extract daily, or even more. To one patient he gave nearly an ounce, and in the course of four months recovery took place. Roberts relieved a boy under his care with the valerianate of zinc, increasing the dose until it reached twenty grains a day. Dr Dickinson has found benefit result from codeia; and ergot is another remedy that has been used with more or less success. The constant galvanic current has also been recommended. One pole may be applied to the loin on one side near the spine, and the other to the corresponding hypochondrium for five minutes; and then they may be transferred to the opposite side of the body in the same manner; or, as Külz advises, the positive pole may be placed upon the nape of the neck, and the negative pole, first to the loins for four minutes, and then to the epigastrium for the same period of time.

RENAL INADEQUACY.—Under this name Sir Andrew Clark described ('Brit. Med. Journ.,' i, 1883) a class of cases of which the main feature is that the kidneys appear unable to excrete more than the normal daily quantity of urine (from forty to fifty ounces); while even this is of low specific gravity (1002 or 1003 to 1008), and is deficient in urea (not containing more than 2 per cent.), though the amount of uric acid may be natural. Even if these patients drink freely of water, they do not pass a larger quantity of urine; and a liberal diet with a full allowance of wine aggravates the disorder. The urine in such cases is devoid of albumen and contains no casts. Although therefore it is possible that the kidneys are on the way towards chronic Bright's disease, yet when Clark had an opportunity of making an autopsy they appeared to be healthy.

The patients are generally ailing, without being definitely ill. They

* I once saw a patient at Dulwich, who had several distinct but very short attacks of what appeared to be diabetes insipidus. I unfortunately preserved no notes, but my impression is that each attack lasted a day or two, and I remember that he passed enormous quantities of urine, and became for the time exceedingly prostrate and exhausted. I think that this recurred at intervals of some weeks.—C. H. F.

are apt to take cold, and do not get rid of the cold easily; and are also liable to be attacked with pneumonia, pleurisy, or pericarditis, without apparent reason. They recover slowly from even slight injuries, and they do not bear surgical operations well—a fact noticed independently by Sir James Paget. They complain of weakness, and unfitness for work; they sleep badly, are subject to headache, and suffer from nervousness. Sir Andrew Clark did not refer to the arterial tension in these cases, but describes the patients as ultimately developing a condition like myxœdema, with pink and white faces, a dry, puffy skin, a slow articulation, and a somewhat staggering gait.

The main points in the treatment which he advised were a very sparing diet, and careful management of the skin.

REACTION OF THE URINE.—Urine has normally an acid reaction, which is probably due to the presence of the acid phosphates of soda and potass.* The greater acidity of the urine when a healthy man lives on a meat diet, and of carnivorous compared with herbivorous animals, is explained by the acid phosphates and sulphates derived from muscle. The same applies to the acidity of urine during pyrexia, when the muscles waste and the excretion of urine is increased.

The degree of acidity of the urine (that is of the whole collected through twenty-four hours) is commonly expressed in terms either of the dried carbonate of soda required to neutralise it, or of an equivalent weight of oxalic acid. Roberts found that in a healthy man it corresponded on an average to about fourteen grains of the carbonate; but there are wide variations, the range during a period of nineteen days being from six to more than twenty-three grains. Writers who give it in terms of oxalic acid find that it usually corresponds to about thirty grains.

After each of the principal meals the reaction of the urine becomes for a time decidedly less acid, and is often neutral. This now well-known fact was originally pointed out by the late Dr Bence Jones. One cause is the formation of gastric juice, which coincides with increase of alkalinity in the saliva, and perhaps in the pancreatic juice. Another cause of diminished acidity of the urine is the absorption into the blood of bicarbonates produced by the digestion of the citrates, tartrates, malates, &c., derived from the food. Consequently the urine is alkaline after meals consisting of fruits and vegetables in which salts of the vegetable acids are abundant. In medical practice the effect of food upon the reaction of the urine is less often noticed, because what is passed from the bladder is generally a mixture of the urine secreted by the kidneys for three or four hours. But one sometimes finds a specimen alkaline when it is passed in the consulting-room an hour or two after breakfast; and the urine passed by candidates for insurance about two o'clock, after lunch, is often neutral or alkaline.

Excessive acidity is met with in leuchæmia and grave anæmia, in scurvy, and in some cases of gout.

* It has been surmised that the organic acids of urine—especially hippuric and uric acids—may be wholly or in part in a free condition, and may take some share in causing the acid reaction. The behaviour of urine towards various organic colouring matters (especially towards congo-red, which is rendered of a deep blue or a violet colour by highly dilute solutions of free acids, including hippuric acid) is such, however, as to disprove this hypothesis. Other facts point in the same direction, as the non-precipitation by urine of a solution of sodium hyposulphite, and the impossibility of separating the hippuric acid of urine by simply agitating it with ether (*Gamgee*).

In some abnormal conditions the urine passed throughout the whole twenty-four hours is found, when collected, to have an alkaline reaction, not due to ammoniacal fermentation of the urine after it is secreted. Quinke has observed this in patients with chronic vomiting and dilatation of the stomach, particularly in those who have the stomach regularly washed out. This cannot, as Quinke supposed, be due to loss of acid, for the acid of the gastric secretion is neutralised by the bile and pancreatic secretion before it can be reabsorbed. Probably, as Dr Gamgee suggests, it is due to absence of absorption of the acid phosphates and sulphates of the food.

Under other circumstances a persistent alkalinity of the urine from fixed alkali is not common; but Bence Jones observed such cases, and so have Mr Hutchinson and Sir William Roberts. Some of these patients are anæmic, or chlorotic, dyspeptic, or phthisical; but others are apparently well. Often the urine is alkaline for two or three days together, and then acid for a time, becoming again alkaline later on. But sometimes it remains steadily alkaline for weeks.

With vegetarians and with Hindoos or Chinese, as with herbivorous animals, an alkaline state of the urine is natural; with those who eat little but animal food (as the Caffres and Esquimaux) we should expect it to be high-coloured and very acid, as with dogs.

Urine which is alkaline from fixed bases is turbid when passed, and on standing throws down a precipitate of the phosphates of the alkaline earths. This deposit will be further considered below with the other urinary sediments (p. 588).

Urine which is alkaline from volatile bases or "ammoniacal urine" is also turbid and on standing throws down earthy phosphates, but they are mixed with an ammonia salt of magnesium. Such ammoniacal urine is at once recognised by its odour and by the evanescence of its alkaline reaction. It is always the result of disease.

URINARY PIGMENTS.—The various shades of colour that the urine shows in health and in disease depend on pigments which have long been the subject of dispute. For, like most animal pigments, they are only present in minute quantities, they do not form crystals, they are of complex nitrogenous composition, they are readily decomposed, and they exist in closely allied forms.

In addition to the pigments which give the characteristic yellow colour to the urine, there are constituents of the secretion which are normally in a colourless condition, and only become pigments when oxidised or otherwise altered in chemical composition. To these the term *Chromogens* is applied.

Urobilin.—This substance, to which Jaffé first gave this name, is allied to, but probably not identical with the *hydrobilirubin* of Maly ($C_{32}H_{44}N_4O_7$). It is amorphous, soluble in water, more soluble in alcohol, and solutions give an absorption band at F in the spectrum; a green fluorescence is produced by its ammoniacal solution on the addition of chloride of zinc. Huppert, in his eighth edition of the well-known work of Neubauer and Vogel, first stated that urobilin is not present as a pigment (except occasionally, and in the smallest traces), but in the form of a chromogen, which becomes coloured on the addition of mineral acids or by other oxidising methods, and sometimes by exposure to air. In certain pathological conditions urobilin exists in the urine in a formed state; and the quantity that can then be extracted is far greater than natural, though it still amounts to only from $\frac{1}{32}$ to $\frac{1}{16}$ in a

thousand parts of the fluid. Urobilin can be derived from bile-pigment, or from hæmatin or hæmoglobin, according to Hoppe Seyler, by deoxidation, possibly combined with some process involving loss of nitrogen. Whether it can be oxidised to choletelin is very doubtful (Hopkins). Urobilin is allied to, but not identical with stercobilin, and the chief pigment of the fæces is closely related to reduced bilirubin.

There seems to be every reason to believe that the colouring matters of the urine (including urochrome and urobilin) are derived more or less directly, and probably by a process of reduction, from the hæmoglobin of the blood.

It is especially in febrile urine,* and in that passed when there is obstruction of the venous circulation, that urobilin is found in excess; but it must be remembered that such urine is generally scanty, so that the increase may not be so great as it appears. Salkowski found that constipation does not augment the urobilin in the urine. In jaundice there is often a great excess, but it can only be recognised after the bile-pigment has been precipitated and removed. The urine may likewise be found loaded with urobilin before an attack of jaundice, and after it has passed off.

Urochrome.—In addition to chemical characters, those afforded by the spectroscope have been called in to determine the nature of the colouring matter of healthy urine, but the difficulties are almost insuperable. At one time the statement of Salkowski that it is urobilin (above referred to as a chromogen) was generally accepted, but the spectrum of urine is not identical with that of solution of urobilin. Possibly the normal pigments of the urine are more than one. But Dr Archibald Garrod has established the constant existence of a yellow pigment ('Proc. Royal Soc.' 1894), to which he has given the name urochrome, applied in 1864 by Thudichum, and also by Schunk, to what was probably a mixture of urochrome with urobilin and other products. When purified, urochrome is a brown amorphous powder, readily soluble in water, less soluble in alcohol, and insoluble in ether and chloroform. Its solutions give no definite absorption-bands in the spectrum.

It is now ascertained that urochrome is closely related to urobilin, for it can be produced from the latter compound by oxidation with permanganate of potash (Riva, 1896), and converted into urobilin by reduction with aldehyde (A. E. Garrod, 1897).

Indican, &c.—The fact that *indigo-blue* is occasionally present in the urine was noticed many years ago by Prout, and afterwards by other observers, some of whom showed that this colouring matter in many cases made its appearance only when the urine had been exposed to the air. Schunck, of Manchester, first recognised in urine the constant presence of *indican*, a colourless material, which he had also discovered in plants, and which readily passes into indigo blue by oxidation. More recently, however, it has been found that the indican of urine is not identical with vegetable indican: according to Baumann, it is an indoxyl-sulphate of potass ($C_8H_6NKSO_4$). Jaffé in 1872, and Baumann and Brieger in 1879, observed that indican could be made to appear in the urine of animals in large quantity by feeding them with indol (H_7H_7N), or by injecting that substance under the skin. Now, indol is formed within the intestine in dogs, and to some extent in man, as the result of a change in albumen induced by

* MacMunn distinguishes between normal and febrile urobilin by the aid of the spectroscope; but Hopkins believes that its supposed special characters are due to admixture.

the pancreatic ferment. The absorption of indol from the intestine in all probability gives rise to the presence of indoxyl in the urine; and Jaffé detected the latter product in large quantity in cases of obstruction of the small intestine and of strangulated hernia, and also in dogs after ligation of a loop of small intestine. Senator investigated the conditions under which such an excess is met with, and found it in states of inanition and wasting, such as arise from cancer of the stomach, gastric ulcer, multiple lymphomata, phthisis with diarrhoea, or granular disease of the kidneys. Heninger found the excess most marked in cases in which wasting is dependent upon intestinal affections; not only when there was constipation, but also when diarrhoea was present. In cases of "catarrhal" (idiopathic or "simple") jaundice, and in cases of cirrhosis of the liver, the amount of indoxyl in the urine was always small. The recognition of indoxyl in the urine is from a practical point of view unimportant. The writer has found the indoxyl reaction most often present in cases of intestinal obstruction, carcinoma of the stomach or bowel, and ordinary obstinate constipation. It is often found in large quantity in urine which is pale and contains little formed pigment.

Jaffé's test for indoxyl is to add to the urine an equal volume of hydrochloric acid, and then to pour in drop by drop a solution of chloride of lime (calcium hypochlorite), shaking the fluid well, and adding no more of the chloride after a greenish colour begins to appear. If any considerable quantity of indican is present, a blue colour will soon show itself; and if the quantity is very large, indigo-blue will be deposited in flocculi. A dilute solution of bromine may be used instead of the chloride of lime. The blue pigment may be afterwards extracted by agitating with chloroform or ether; and in this way the amount of it may be roughly estimated. Another test, referred to by Dr Hopkins in his excellent article in the first volume of Schäfer's 'Text-book of Physiology' (p. 287), is to put a crystal of chlorate of potash in a test-tube and cover it with the urine to be examined; strong hydrochloric acid is then run carefully down upon the crystal, and when the urine floats upon the heavier acid a blue ring appears at their junction.

Indigo-red, instead of its isomer, indigo-blue, sometimes appears with Jaffé's test.

In cases of constipation, whether functional or from organic disease, when an equal bulk of hydrochloric acid and a few drops of nitric acid are added to the urine and the mixture boiled, a purple or claret colour is often produced, and when, after cooling, this is shaken up with ether the ether is reddish, while the urine, if indoxyl is present, is bluish.

This red pigment is produced, according to Brieger, by another constituent of human urine, an allied substance, skatoxyl-sulphate of potass, which is derived from *skatol* (C_9H_9N), this being, like indol, a product of the decomposition of proteids within the intestine. Both indol and skatol have a faecal odour.

Uro-erythrin.*—Among pathological urinary pigments, that which gives a pink or red colour to deposits of lithates was called *acide rosâique* in France, purpurate of ammonia by Prout, purpurin by Golding-Bird, and *uro-erythrin* by F. Simon. It has been lately investigated by MacMunn, Riva, and Archibald Garrod. It is amorphous, not very soluble in water,

* Uro-erythrin is sometimes spoken of as identical with what Dr Golding-Bird called "purpurin." Probably, however, he included under that term several substances which are now described as distinct, since his test was to add hydrochloric acid to the hot urine, when he obtained a colour "varying from a delicate lilac to the deepest crimson."—C. H. F.

but dissolved by hot alcohol, and when dry turned green by Liq. Potassæ. Its solutions are rapidly and completely blanched by light.

Hæmato-porphyrin.—This iron-free product of hæmoglobin is present in very small amount in normal urine, and also in the bile and fæces, as Dr A. E. Garrod has lately shown (Bradshaw Lecture, 'Lancet,' Nov. 10th, 1900); but it is comparatively abundant in the urine of various diseases (*e. g.* rheumatic fever, Addison's disease, and cirrhosis of the liver), and also in the urine of those who have lately taken sulphonal.

Alkaptonuria.—Dr A. E. Garrod (in the 'Med.-Chir. Trans.' for 1899) gave an interesting account, with a table of 31 cases, of a rare condition of urine known under this name since an observation of Bödeker in 1859. The urine is colourless when passed, but rapidly acquires a deep brown colour when exposed to the air, and this oxidation is hastened by the addition of alkalies. The chemical nature of Bödeker's "alkapton" is not yet certain, but is believed to be homo-gentisinic acid, probably derived from tyrosin.

The condition is not connected with any morbid symptoms, and its only clinical importance is that alkaptonuria causes reduction of copper, like glycosuria, though there is no other sign of the presence of grape-sugar. Most of the cases occurred in males; some are congenital with "family" distribution among brothers and sisters, as in five patients of Dr Pavy.

Medicinal pigments.—Other colours in the urine are due to the administration of drugs. Rhubarb colours the urine a deep gamboge-yellow, which is changed to red by the addition of ammonia. When operations were performed under the carbolic spray, patients used to pass urine of a dingy olive, sometimes almost black, colour; this is due to the excretion of pyrocatechin, $C_6H_4(OH)_2$, and its subsequent oxidation by exposure to the air. Senna imparts a brownish, and logwood a reddish tinge. Santonin gives a conspicuous orange-yellow colour to the urine if alkaline, a golden-yellow if acid.

A curious point observed by the late Dr Moxon is, that in persons who are taking iodide of potassium the addition of nitric acid to the urine produces an orange-coloured zone, the appearance of which is characteristic.

URINARY DEPOSITS.—We have already spoken of leucine and tyrosine as precipitated from the urine in cases of acute atrophy of the liver, and we shall in a future chapter discuss the subsidence of urinary casts, epithelium, pus, and blood.

The urinary sediments now to be described are saline or other inorganic compounds which separate from their solution in the urine. They are of clinical importance from two points of view: as indications of disturbance of the chemical processes in some other part of the body, or as involving the risk of the formation of gravel or calculus within the urinary organs. They are (1) lithic or uric acid and the lithates, (2) cystine, (3) oxalate of lime, (4) earthy phosphates, (5) sulphate of lime.

Lithic acid and lithates.^{*}—In urine having the normal acid reaction lithic acid exists in the form of acid salts (probably, as Bence Jones believed, quadrurates) of soda and other bases, which at the temperature of the body

^{*} Lithic acid was the name given by its discoverer, Scheele, in 1776, and was used by Prout and Bence Jones. Uric acid (Harnsäure) and urates are later synonyms and less distinctive, but they are as firmly fixed in chemical as lithic acid and lithates are in clinical literature.

are moderately soluble; but as the fluid cools, they are often precipitated. Very slight changes may disturb the balance between the lithates and other constituents, so as to throw down the lithic acid, which requires a very large quantity of water (14,000 parts in the cold) to hold it in solution. Sir William Roberts found the quadrurate to be decomposed by pure water into biurates (of soda, potash, or ammonia) and uric acid. The biurate in the presence of acid phosphates (of soda and potash) is reconverted into quadrurate, which is thrown down in crystals.*

Lithic or uric acid ($C_5H_4N_4O_3$), *red gravel*.—As a deposit from urine, this substance appears in the form of crystalline grains which have a reddish colour, so that they often look almost exactly like cayenne pepper. They usually lie at the bottom of the fluid, but sometimes adhere to the sides of a glass vessel. Their colour is not proper to the acid itself, which is colourless; it really belongs to urinary pigment, for which uric acid seems to have a strong attraction. Dr Lionel Beale has three or four times seen colourless crystals of uric acid deposited from urine which happened to contain hardly a trace of colouring matter.

The form of uric acid crystals is primarily that of a rhombic prism or lozenge, but they present a great many varieties of shape. Sometimes they are very short and thick cylinders; sometimes they form rods which seem to have rectangular extremities; sometimes they appear as flat plates, pointed, oval, or of a halberd shape. Very often they form large stellate aggregations, and sometimes fan-shaped masses. Dr Ord has stated ('Med.-Chir. Trans.,' lviii) that the deviations in form which those crystals present from the regular four-sided or six-sided plates that are seen when the pure acid is crystallised from water, depend upon the presence of mucus and of colouring matter, which substances favour Rainey's "molecular coalescence" rather than crystallisation, so that by a kind of compromise the resulting crystals, instead of having sharp angles and straight sides, are more or less rounded off, and also tend to cohere together in masses having a common centre. He has found that the presence of albumen in the urine still further modifies the form of the crystals, rendering them small and thick, with their angles more or less nearly equal, so as to give them the barrel or cask shape. The association with sugar, on the other hand, tends to produce flat and elongated crystals, which may have the typical hexagonal shape that is otherwise rarely seen.

A microscopical deposit of lithic acid is sufficiently distinguished from any other that occurs in the urine, by being crystalline and of yellow colour. Its weight, its red colour, its solubility in alkalies, and insolubility in dilute acids are equally characteristic to the naked eye. Moreover, unless the quantity be very small, the well-known murexide test can be applied.

Mixed amorphous lithates or urates.—The commonest of all urinary sediments consists of a loose pulverulent substance, which varies in tint, but is always deeper coloured than the urine from which it is deposited. It is often spoken of as brick-coloured, or "lateritious" (*later*, a brick). It generally settles quickly, leaving the urine above almost clear, but some-

* Scherer many years ago asserted that during the first few hours after being voided, urine, as a rule, undergoes what he terms an "acid fermentation." This, however, is not now believed to be the case. By Voit and Hoffman ('Bayerische Acad. Sitzungsbericht,' ii, p. 79) it is maintained that the acid phosphate of soda (the cause of the acid reaction of urine) gradually takes away from the uric acid more and more of the bases with which it is combined, so that an appreciable diminution in the acidity of the urine may be produced in this way, since the uric acid, deposited in a solid form, can no longer affect the reaction.

times it remains a long while diffused. Not infrequently, if the urine has been put aside while still warm, different strata of the deposit in the same glass have different colours—fawn-coloured, orange, brick-red, or purplish. A part adheres to the side of the vessel as a sort of film or bloom, which is easily cleaned off by a little liquor potassæ or hydrochloric acid. With the microscope this precipitate is seen to consist of minute amorphous granules, which are coarser or finer, according to the closeness of its aggregation.

As soon as the turbid urine is warmed, it becomes bright and clear: and even when albumen is present, there is little difficulty, for the lithates dissolve at a far lower temperature than that at which albumen coagulates.

The lateritious deposit was formerly spoken of in this country as urate of ammonia, while German writers described it as urate of soda. It always consists of urate of soda in the largest amount, mixed in different proportions with those of potass and lime, to which that of ammonia is only exceptionally added. The quantity of uric acid in it is much in excess of what would correspond even with the ordinary acid salts of these bases (for uric acid is bibasic), being in fact about twice as much (quadrurates), and making up 80 or 90 per cent. of the whole precipitate. Roberts says that this loosely combined acid can be separated from the acid lithates by warm water (which must of course be used sparingly), or even by cold water, with which the deposit is to be repeatedly washed upon a filter.

Crystalline lithates or urates.—It is a curious fact that urate of soda is never deposited from urine in those needle-shaped crystals with which we are familiar in gouty concretions (*cf.* vol. i, p. 468), and which may be readily obtained artificially from solutions of the salt. In some cases, however, it forms opaque globular masses from which project spiny crystals, straight or curved. These “hedgehog” or “thorn-apple” (*Stechapfel*) bodies occur in putrid ammoniacal urine; but they are also sometimes seen in children who are feverish, when the urine is scanty and concentrated. Roberts relates the case of a child three years old who was suffering from fever, and had passed no water for three days: while he was examining the abdomen, the child cried, and urine began to flow; the first that came was turbid and of a gamboge-yellow colour, and was found to be full of the spiny globules of sodium urate.

Clinical significance.—In medical practice, deposits of lithic acid and of lithates have to be looked at from two points of view.

First, there is the question whether they are likely to be the cause of calculi, with lumbar pain, hæmaturia, and other symptoms of pyelitis or of renal colic.

As regards free uric acid, if it is present in the urine as it is passed, it is always morbid and frequently gives pain while it traverses the urethra. If it is precipitated soon after the urine is passed from the bladder—if, as Roberts puts it, the crystals are seen before the urine cools—there is always a risk that they may also separate in the urinary passages. If the deposit takes place within three or four hours it is certainly not natural, but if not until after twelve hours, it has no pathological significance.

Amorphous urates cannot in themselves produce irritation of the kidneys or bladder, since they are never precipitated so long as the urine is of the temperature of the body. But the hedgehog crystals of sodium lithate are perhaps less innocent.

Secondly, there is the question how far deposits of lithic acid or of

lithates indicate a disturbance of the chemical processes by which nitrogenous compounds are prepared for excretion by the kidneys.

We cannot too strongly insist that the formation of such precipitates is not in itself a proof that the quantity of uric acid is excessive. In the case of the amorphous urates a great deal depends upon the *temperature* to which the urine falls when it cools. There does not appear to be any other cause than the lower temperature for the fact that red deposits are so much more often observed in winter than in summer. Again, whatever diminishes the *amount of water* excreted by the kidneys increases the likelihood that urates will be precipitated. This seems to be the reason why amorphous urates are so often seen in healthy persons after violent exercise and after profuse sweating, and also in patients who are suffering from any disease, such as rheumatic fever, attended with perspiration in its whole course, or who are passing through the crisis of an attack of pneumonia. Again, the degree of *acidity* of the urine is also of importance. Most observers say that urates are never deposited except from urine which is acid, but according to Salkowski the reaction is occasionally found to be neutral. In the case of free uric acid, the degree of acidity of the urine is the most essential cause of its precipitation. In addition to a high degree of acidity, Roberts mentions poverty in chloride of sodium and other mineral salts and low amount of pigment, as conditions which favour deposit of uric acid, independent of its abundance in the urine.

On the whole, therefore, we conclude that cold after the urine has been passed, and acidity with concentration before its being passed, are the most common causes of deposit of the mixed lithates; and that a high degree of acidity is the most common cause of the deposit of crystals of lithic acid.

Quantitative analysis of lithic or uric acid.—An obvious method is to precipitate with hydrochloric acid, to collect the uric acid upon a filter, and to weigh it; a variable amount of the uric acid remains in solution and must be allowed for.* Ludwig and Salkowski introduced a method which depends upon the formation of urate of silver; this is very accurate but somewhat troublesome. Dr Pavy has advocated ('Med.-Chir. Trans.,' lxii) the use of the ammoniated cupric solution, which he employs for the estimation of sugar. Dr Haycraft has since devised a modification of the silver method, which was described in the 'Journal of Anatomy and Physiology,' vol. xx, p. 695. It is more rapid, but less accurate.

Dr F. G. Hopkins has introduced a process of estimating uric acid as

* The details of this process and of the silver process next mentioned in the text are given as follows by Dr Gamgee:—"In order to determine the amount of uric acid, 200 cubic centimetres of the filtered urine (from which any albumin which may be present has been separated by boiling) is treated with 10 or 15 c.c. of hydrochloric acid of specific gravity 1.12, and set aside for forty-eight hours.

"At the end of that time the uric acid is collected on a small weighed filter; the filter and uric acid are carefully washed, and the washings added to the filtrate. The filter and uric acid are then weighed, and by subtracting the weight of the former that of the greater part of the latter is found.

"The combined volume of the urine and the washings of the filter is now carefully ascertained, and for each 10 c.c. 0.00048 grm. (nearly half a milligramme) of uric acid is added to the amount already directly obtained by weighing.

"Salkowski and Ludwig's method consists in precipitating a known quantity of urine with a specially prepared ammoniacal solution of silver. The precipitated urate of silver is collected on a filter, washed with ammoniacal water, and then decomposed by boiling it with a special solution of sodium sulphide, silver sulphide being precipitated whilst all the uric acid is obtained as urate of soda in solution. From the latter, after concentration, the uric acid is precipitated by means of hydrochloric acid."

ammonium urate, which is at once accurate and neither tedious nor difficult. It will be found in the 'Proc. Royal Soc.' for 1892, and more fully described in the thirteenth volume of the 'Journal of Pathology,' for 1893 (p. 451). A hundred c.c. of urine are saturated with NH_4Cl , and, after two hours' standing, the insoluble ammonium urate is filtered off, washed with solution of the same ammonium chloride in hot water, and, after treatment with HCl , the resultant uric acid is in its turn filtered off, washed, dried, and weighed.

Pathology.—The total quantity of uric acid excreted on mixed diet by healthy persons in the twenty-four hours is not large; as a rule, it is from five to eight grains. The proportion between the uric acid and the urea is usually as one to fifty or as one to sixty.

It is remarkable that uric acid crystals are very common in the urine of children, even infants at the breast; and calculi of uric acid are frequently found in the bladder of boys. The explanation of this fact is not obvious, nor the equally curious one that these calculi, though common in hospital patients, are rare in private practice.

Liebig supposed that lithic acid is formed by oxidation out of the same materials as urea, and represents a stage in the formation of the latter substance in which the oxidising process is as yet incomplete. This hypothesis, however, has never been proved. The most obvious way of testing it was that of adding uric acid to the food, and determining whether or not the amount of urea excreted afterwards undergoes an increase. Wöhler and Frerichs tried this experiment, and came to the conclusion that the uric acid was mainly converted into urea. Salkowski found that in dogs allantoin appeared to be formed out of the lithic acid; and oxaluric acid and oxalic acid are also products of lithic acid.

In birds lithic acid is derived from the same aspartic acid, glycin, leucin, and asparagin, which in mammalia pass into urea; and urea itself, when given to birds, is said to undergo conversion into lithic acid. But according to the latest evidence the formation of lithic acid in mammals is separate from that of urea throughout, and is not a stage in the process. Indeed, it seems probable that the output of uric acid is nearly constant so far as food is concerned, and that its ratio to urea varies with the output of urea.

Clinical facts which seem to support the view that lithic acid may be excreted in excess as the result of a deficient supply of oxygen, and take the place of urea, are open to question in every case. Thus there is, no doubt, in leuchæmia a marked increase of uric acid in the urine, both absolutely and relatively to the urea; and this has been attributed to the deficiency of red discs as oxygen carriers; but Pettenkofer and Voit found that in a case in which the excess of lithic acid amounted to 64 per cent., the absorption of oxygen and the evolution of carbonic acid from the lungs were normal. Cohnheim stated that the "uric-acid infarcts" in the kidneys of newly born infants, are common in healthy children who had breathed well, but are often absent when there had been pulmonary atelectasis, bronchitis, or broncho-pneumonia; and Bartels many years ago ('Deutsches Arch.' 1865) endeavoured to show that in various conditions of insufficient aëration of the blood the amount of lithic acid in the urine is increased in proportion to that of the urea, from the normal rate of one to fifty or sixty, up to that of one to thirty-five.* But experiments made on animals by Senator and

* If Bartels' figures were based on the results given by Heintz's method of estimating uric acid, they were probably low rather than exaggerated.

others contradicted these observations; and it has since been shown by Fränkel, and also by Fleischer ('Virch. Arch.,' 1882), that the effect of dyspnœa is actually to augment the secretion of urea. Possibly the over-activity of the respiratory muscles plays a part in bringing about this result; but, as Salkowski observes, the conditions in dyspnœa are far too complicated to allow of our attributing changes in the urine solely to a deficient supply of oxygen.*

The meaning of deposits of uric acid or of urates in gastric and hepatic disorders is not always clear. Roberts made during seven days a series of observations on a person who dined at 2 p.m. and afterwards took no solid food till the next morning. He found that during the period when the urine was alkaline after the meal—which was from 4 p.m. till 7 p.m.—the quantity of lithic acid excreted in each hour was three times greater than it was from 9 p.m. till 11 p.m., or later on during sleep. The proportion which the acid bore to the rest of the urinary solids, and even to the water, was likewise greater, though, being alkaline, the urine of course threw down no deposit of lithates. The food taken while these observations were being made was very simple.

As a matter of fact, many persons who habitually live plainly, and pass clear urine, find that the occurrence of a brickdust deposit is sure to follow indiscretion in diet; while others, who habitually indulge in the pleasures of the table, no less habitually pass urine that becomes turbid with urates as it cools. No doubt this does not prove an excessive excretion of lithic acid; it may depend on deficient water or excessive acidity.

That deposit of lithates is not an indication of excessive nitrogenous elimination from eating too much nitrogenous food seems clear from the fact that it is most apt to follow "rich" (*i. e.* fatty) and sweet dishes, port wine, and champagne, which contain little or no nitrogen.

The presence of excess of *colouring matter*, and particularly of uroerythrin, in the urine is generally held to afford evidence of lithiasis, apart from the mere presence of the uric acid or of the urates. The deeper the tint of a lateritious deposit occurring in a non-febrile patient, the more surely is it often regarded as a proof of "lithæmia" or a gouty diathesis; and the same conclusion is drawn from the formation of pigment in large quantity on the addition of mineral acids to urine.

The late Dr Golding-Bird was convinced that the presence of a violet colour on the addition of hydrochloric acid to urine and heating was a proof of "derangement of the hepatic function." He thought it was sometimes a valuable clinical aid to diagnosis in cases in which there was an abdominal tumour of doubtful nature, or in which it was uncertain whether ascites was due to hepatic disease or to chronic peritonitis. This indoxyl-sulphate reaction has, however, no connection with the pink pigment just mentioned. Nor can we attach much significance to the coloured zone that is often observed when urine is poured upon nitric acid. This seems to depend upon the presence, in different proportions, of the chromogen of urobilin, indican (or rather indoxyl-sulphate), and skatoxyl-sulphate of

* Dr Haig believes that the excretion of lithic acid and lithates is increased by taking alkalis and diminished by acids; the explanation apparently being that in the former case the lithic acid or acid lithates in the liver and spleen are rendered more soluble, and thus carried off in larger quantity to the kidneys ('Journ. of Physiology,' vol. viii, p. 211); but Dr Hopkins states that there is no foundation for the conclusion that the excretion of uric acid varies inversely as the acidity of the urine ('Schäfer's Text-book,' vol. i, p. 595).

potash (p. 577).* The tint is sometimes crimson, sometimes purple, sometimes bluish black. The darker purple is usually seen in cases of marked constipation.

To conclude, the effect of food on the formation and excretion of uric acid is still undetermined, the amount excreted varies with individuals, and the mere appearance of urates or crystalline uric acid in the urine is no evidence of excessive formation or excessive excretion of these products.

The following facts are well ascertained, though at present they have neither physiological explanation nor clinical importance. Uric acid often forms as crystals in the kidneys and urine of new-born infants. The output of urates is in most cases increased by excessive exercise. Their excretion does not appear to be increased by the use of alkalies, although it is probable that the uric acid and the less soluble urates may thus be excreted in a more soluble form. Salicylates increase the amount passed in the twenty-four hours.† In gout, according to Sir Alfred Garrod's original observations, the uric acid excreted is diminished, except during an acute attack, when it is decidedly increased. In leuchæmia the output of lithic acid (usually as lithates) is constantly increased.

Cystine ($C_3H_5NSO_2$).‡—In 1810 Wollaston discovered, in analysing an urinary calculus, that it was composed of a peculiar substance, to which he gave the name of "cystic oxide." Subsequently the same substance, which is now known as cystine, was found to occur as a light flocculent deposit from the urine. It looks like a fawn-coloured sediment of amorphous urates, but with the microscope it is seen to be crystalline, consisting of hexagonal tablets, which Roberts describes as having an iridescent, mother-of-pearl lustre, and as being often chased on the surface by lines of secondary crystallisation; there may also be thick rosettes of great brilliancy.

Acetic acid throws down from urine yielding this deposit an additional quantity of cystine, which had remained dissolved; and sometimes, perhaps, might reveal its presence in urine which contained it in too small an amount to yield a spontaneous sediment. But the occurrence of cystine in any form is very rare.

Urine which deposits cystine is usually faintly acid, and is described as having "a honey-yellow colour, an oily appearance, and a peculiar sweet-briar odour." The sediment is instantly dissolved by caustic ammonia; when this evaporates the hexagonal crystals reappear, and also highly refracting prisms, lying singly or forming stars, which are not otherwise seen.

Cystine is a derivative of amido-lactic acid; its formula corresponds with sulph-amido-pyruvic acid, pyro-racemic or pyruvic acid being derived from lactic acid. A remarkable fact is the large amount of sulphur it contains, nearly 26 per cent. When urine holding cystine in solution decomposes, it evolves sulphuretted hydrogen, so that it blackens a beaker in which it stands by its action on the lead in the glass. It is said that the

* Dr Hopkins informs the writer that one of the pigments thus produced from its chromogen is uro-rosein, a very definite substance.

† See paper by Dr Fawcett in the 'Guy's Hosp. Reports' for 1895.

‡ Seven atoms of hydrogen or six are sometimes assigned to cystine (Dewar and Gamgee, 'Journ. Anat. and Phys.,' vol. v). See A. S. Lea's foot-note to the Appendix to Foster's 'Physiology,' 5th ed., p. 150.

silver in the pockets of persons passing cystine in the urine may be discoloured.*

Cystine has several times been observed in the urine of brothers or sisters. It is more often seen in males than females, and in children or young adults than in those who are older, but Roberts met with it in a patient aged fifty-seven. No ill-health is associated with its presence, but it may lead to the formation of calculi. Some patients go on voiding it for many years; or it disappears from the urine for a time, and then returns, or may never be seen again. It is said that some patients have passed as much as fifteen grains of cystine in the twenty-four hours.

The treatment that has been advised is the administration of nitrohydrochloric acid, or tincture of perchloride of iron, but it is doubtful whether either is of use.

Xanthin (CHNO), which appears as a very rare calculus, was first detected in the urine, by Strecker, in 1857. It is now often found to be present, and can be exactly estimated. It and its allies hypoxanthin and guanin (the alloxuric bodies) are closely related chemically and physiologically to uric acid, and all these have been observed as urinary deposits.

Leucin and tyrosin have been already described as present in the urine of acute atrophy of the liver (*supra*, p. 546).

Oxalate of lime ($\text{CaC}_2\text{O}_4 + 4\text{Aq.}$).—This substance produces in the urine a slight precipitation-cloud, so little noticeable that a patient's attention is scarcely attracted by it. In a glass vessel it often appears to the naked eye as bright points floating on a thin film of mucus; or it clings to the sides of a glass, and marks it with minute lines, running transversely or obliquely, as if it were scratched; these are due to the crystallisation of the salt along minute irregularities left on the surface of the glass when wiped. Or, near the bottom of the vessel, a soft, grey, mucus-looking sediment supports a white denser layer with an undulating but sharply limited surface.

If a drop of the deposit be taken up with a pipette, and placed under the microscope, the oxalate is generally seen to form small, colourless, octahedral crystals, beautifully transparent and lustrous. So transparent are these octahedra that all their facets and angles are visible at the same time. They have a flattened shape, the principal axis being much shorter than the other two. In size they vary considerably; the late Dr Golding-Bird found that the length of the sides of different specimens ranged from $\frac{1}{5600}$ to $\frac{1}{750}$ of an inch. They usually present one apex, and then have the appearance which is aptly compared to that of a square envelope. But in other positions they may seem to have a rhombic, or occasionally rectangular or hexagonal, outline. They are really octahedra, two four-sided pyramids set base to base. Other modifications in their shape are due to flattening of their lateral edges, which gives them a dodecahedral form, so that they consist of a regular prism with a pyramid on each summit. It is also said that half-crystals are sometimes seen—pyramids on a square base.

Occasionally oxalate of lime appears in the form of "dumb-bells." The

* The composition of cystine forms an obvious point of resemblance between it and taurine, a constituent of bile. Cystine has recently been artificially obtained by Mörner from horn.

real shape is that of a flattened, rounded disc, with a central depression in each surface; and when these discs lie edgewise, they look like dumb-bells. According to Golding-Bird the long diameter ranges from $\frac{1}{1120}$ to $\frac{1}{500}$ of an inch; their short diameter from $\frac{1}{2500}$ to $\frac{1}{750}$ of an inch. There was at one time doubt as to the chemical nature of the dumb-bells. Dr Bird was disposed to think that they differed in composition from the octahedra, and might possibly consist of the oxalurate of lime. But this question has been finally set at rest by the observations of Dr Ord, who has shown that when oxalic acid and lime come into contact in gelatine, the dumb-bell form may be observed. The assumption of a discoid, rather than of a crystalline character, is doubtless due to the presence of colloid substances, such as gelatine, gum, and mucin.

Dr Lionel Beale has recorded the presence of dumb-bell crystals in the interior of renal tube-casts.

Chemically, calcium oxalate may be distinguished by its being insoluble in potash and in acetic acid. The shape of the octahedra, however, is in itself a sufficient proof of their nature; it is only the dumb-bells which might be mistaken for like bodies of a different composition.

Oxalate of lime is no doubt in part derived directly from the food. Many vegetables and fruits contain this salt; not only rhubarb (which is so largely eaten in the spring in England) and sorrel (which on the Continent forms a common article of diet), but also, to a less extent, spinach, cauliflower, asparagus, tomatoes, apples, and grapes. Buchheim and Piotrowski found that from 10 to 14 per cent. of the quantity of oxalate ingested could be recovered in the urine.

Auerbach detected oxalate of lime in the urine of dogs when fed entirely on animal food; so that oxalic acid may be formed within the body as the result of chemical changes, possibly, from uric acid.* Schunck's discovery, that oxaluric acid in minute quantity is a normal urinary constituent, may perhaps point to a step in the process in question. English physicians of a former generation supposed that oxalic acid arose out of sugar; its abundant presence in the urine has often been observed in diabetes.

Clinical significance.—Prout and Golding-Bird regarded the recognition of the oxalate of lime in the urine as affording a key to the successful treatment of a group of symptoms which included pain or sense of weight across the loins, irritability of the bladder, incapacity for exertion, impairment of sexual power, dryness of the palms of the hands, nervousness, hypochondriacal depression, and loss of flesh. But in the urine of many such "neurasthenic" patients no oxalate can be found, while in that of many who are not so it is present. Again, Fürbringer has shown that no conclusion as to the quantity of the salt contained in the urine can be drawn from the fact that crystals are discovered in the urine. Healthy persons, according to Schultzen ('Reichert's Archiv,' 1868), pass about a grain and a half in the whole of the twenty-four hours; occasionally, in cases of jaundice, as much as seven and a half grains.

The writer at one time used to look for oxalates in the urine of nervous and dyspeptic patients, passing a large amount of pale colour and low

* Dr Rees maintained in his 'Croonian Lectures' for 1856 that all the oxalic acid arose out of uric acid in the urine itself, especially when heat was applied to it for testing purposes. This is very doubtful; and Neubauer ('Ztschrift. f. anal. Chem.,' 1868) found that even on adding oxalurate of ammonia and chloride of calcium to urine no oxalate of lime was formed, the only change being the conversion of the oxalurate into carbonate of ammonia when the fluid putrefied and became alkaline.

specific gravity, and no doubt the characteristic crystals were often to be found, independently of eating rhubarb tart. But they were perhaps as often absent, and they certainly sometimes form in the course of twenty-four hours after the urine has been passed.

It has long been known that the octahedral crystals are more numerous and larger in urine that has stood for a time than they were when it was first passed; and Voit and Hoffman have satisfactorily accounted for the gradual separation of the oxalate of lime from urine which held it in solution. As far back as 1856, Neubauer showed that this salt can be kept dissolved by a solution of the acid phosphate of soda. But when urine is allowed to stand, the urate of soda in it is acted upon by the acid phosphate of soda, so that an acid urate of soda, and ultimately free uric acid, are formed, while the phosphatic salt loses its acidity. Consequently the condition of the oxalate of lime being held in solution is no longer present, and it crystallises out.

The fact, therefore, that octahedra are discovered in a patient's urine is in itself no reason for supposing that there is danger of the deposition of oxalate of lime in the renal pelvis or in the bladder. It would also seem to follow that the danger must be less in proportion as the urine is more highly acid, but oxalate crystals may often be found in very acid urine; Salkowski alone speaks of them as being present chiefly in urine that is only just acid, neutral, or faintly alkaline. They are sometimes seen in association with deposits of urates or of uric acid; and radiating crystals of phosphate of lime are not uncommon in urine containing oxalates.

Oxalate of lime is insoluble in hot and cold water, in potash, and in acetic and dilute hydrochloric acids. Only strong hydrochloric acid, with the aid of heat, decomposes this very stable salt.

From what has been already stated, it is evident that *treatment* is by no means necessary for all cases in which oxalate of lime is discovered in the urine. Dyspepsia and the effects of dyspepsia must be dealt with in the same way as if no such deposit were present; and as a rule the nitrohydrochloric acid does more good than alkalies. Patients whose urine contains oxalates and who have had lumbar pain or hæmaturia should be cautioned against eating rhubarb. To prevent the deposition of the salt within the urinary passages the best plan would seem to be that of maintaining the naturally acid state of the urine; but Roberts says that in some cases the urine has temporarily ceased to contain the crystals when it has been rendered freely alkaline.

Earthy phosphates.—The amorphous phosphate of lime ($\text{Ca}_3\text{P}_2\text{O}_8$) is by far the most abundant constituent of the "white gravel" which is thrown down in alkaline urine. It collects as a flocculent deposit, always much paler than the supernatant liquid, and thus distinguishable from the fawn-coloured or brick-red amorphous lithates. It also forms an iridescent film upon the surface, which has a greasy look but does not contain oil drops, and is said to be due to the escape of carbonic acid gas from the urine. Mixed with this is occasionally found another phosphate of lime ($\text{CaHPO}_4 + 2\text{Aq.}$), which is crystalline. It forms rods or needles, club- or bottled-shaped, and generally grouped together into stars, rosettes, fans, or sheaf-like bundles. Still more rarely present is the phosphate of magnesia ($\text{Mg}_3\text{P}_2\text{O}_8$), which forms elongated plates with oblique ends,

and was first recognised as a precipitate by Tollens and Stein. It is a soluble salt, so that it is not likely to be precipitated unless present in very large quantity; and if ammonia is present it unites with the phosphate of magnesia to form the well-known "triple-phosphate."

All phosphatic deposits are dissolved by acetic acid without effervescence, a test which distinguishes them from all other urinary precipitates.

Carbonate of lime seems to be frequently mixed in small quantity with the amorphous phosphate of lime, and sometimes it appears in small spheroids. It is soluble in acids with effervescence.

Not infrequently urine when warmed in a test-tube becomes cloudy and opaque, as if it contained albumen; but this is owing to a separation of phosphate of lime that had hitherto been retained in solution. A drop of acid (nitric, hydrochloric, or acetic) decides the point by dissolving the cloud.* This precipitate has been attributed to the heat driving off carbonic acid from the urine. But this can scarcely be the case, since, when the turbidity is but slight, the urine becomes clear again on cooling.

The writer has found that if the test-tube is heated until all the precipitate has fallen, and this is allowed to subside, the clear supernatant urine will, after being decanted off, again deposit a cloud of earthy phosphates on being heated, and this may be repeated several times—another proof that they are not held in solution by carbonic acid.

Urine which thus deposits its phosphates on being heated does not always do so on neutralisation by alkalies; indeed, it is sometimes alkaline or neutral to begin with.

Clinical significance.—The presence of a phosphatic deposit, in urine alkaline from fixed bases, is of no consequence, so far as concerns the formation of concretions within the urinary passages, since the amorphous phosphate of lime which forms the bulk of the precipitate does not easily cohere into solid masses.

Nor is there any ground for supposing that visible precipitation of phosphates, whether spontaneous or as the result of heat, is an indication that these salts are being excreted in excess. To determine that, it is necessary to make an exact quantitative analysis of the twenty-four hours' urine. Such analyses have been often made, but with the most meagre results, so far as their clinical value is concerned. There is therefore no ground for the opinion that a "phosphatic" state of urine points to excessive disintegration of the nervous tissues. Equally baseless is the treatment of "neurasthenia" by phosphates, hypophosphites, phosphoric acid, or phosphorus itself, with the object of increasing the formation of lecithin; a more harmless if equally absurd deduction from the theory is recommendation of fish as appropriate food for "those who work with their brains."

Triple or ammonio-magnesian phosphate.—In ammoniacal urine, the earthy phosphates are as insoluble as in that which is alkaline from carbonates of soda and potash, and white gravel is therefore precipitated.

The phosphate of lime ($\text{Ca}_3\text{P}_2\text{O}_8$), with a little of the corresponding magnesian salt, comes down in the form of amorphous granules, not as dumb-bells or crystals. But along with these salts are also thrown down

* This fallacy was unknown to Prout and to Bright, and nearly led the latter to abandon his hypothesis of the connection of albuminuria with dropsy and disease of the kidneys. It was detected by Dr Owen Rees ('Guy's Hosp. Reports,' 1836, p. 401).

brilliant crystals of phosphate of magnesia and ammonia ($\text{MgNH}_4\text{PO}_4\text{Aq.}$), formerly spoken of as "triple phosphate." The form of these crystals is that of a triangular prism with bevelled ends ("tombstone" form); but they are liable to many modifications by their edges and angles planing off, or by their sides being hollowed out. Some of the prisms may be so short that their bevelled ends meet one another on one edge; they then look not unlike the octahedra of oxalate of lime. The addition of acetic acid at once distinguishes them; it dissolves all phosphatic deposits.

The precipitate of mixed phosphates does not carry down urinary pigment with it, like the amorphous lithates which occur in acid urine; and it therefore preserves its white colour.

An important character of the mixed phosphates thrown down as the result of the ammoniacal fermentation is their liability to undergo concretion into a mortar-like mass; this forms a large part of many calculi, and it may also collect on the end of a catheter left in the bladder, and on the surface of the mucous membrane itself.

Sulphate of lime ($\text{CaSO}_4 + 2\text{Aq.}$).—Considering that both sulphuric acid and calcium are normal constituents of the urine, it is remarkable that the not very soluble compound which they form is so rarely deposited. It was recognised first by Valentiner in 1863, and afterwards by Fürbringer ('Deutsch. Arch.,' xx). It formed a bulky white sediment consisting of long needles and prisms with oblique ends, arranged in sheaves and rosettes. Valentiner's patient was an anæmic boy, Fürbringer's a wasted man affected with paraplegia.

AMMONIACAL URINE.—If exposed to the air, urine soon becomes turbid and offensive, with a pungent ammoniacal smell. This change is accelerated by the presence of pus or blood or mucus, and by admixture with even the smallest quantity of urine already putrid. It occurs less rapidly in urine when concentrated and highly acid than when it is pale and of low specific gravity, with a faintly acid, or neutral, or alkaline reaction.

The same ammoniacal decomposition may take place within the urinary passages while the urine is still unpassed, and may become a source of grave mischief.

Decomposed urine may be recognised by its penetrating foetid odour, quite unlike that of recent and healthy urine. It gives off carbonate of ammonia, which may irritate not only the nose but the conjunctivæ; a product of the decomposition of the urea, thus: $\text{CH}_4\text{N}_2\text{O} + 2\text{H}_2\text{O} = (\text{NH}_4)_2\text{CO}_3$. It gives to the urine an alkaline reaction, and the change which produces it is called the alkaline or ammoniacal fermentation. That the reaction is due to this cause, and not to the presence of fixed alkali, can be easily determined. A piece of reddened litmus or turmeric paper, suspended in the mouth of a vessel over the urine but without touching it, will be found slowly to change its colour. Or the paper, after being dipped in the urine, may be left exposed to the air to dry, when its original colour will presently reappear. It must be remembered, however, that urine which was originally alkaline from fixed alkali may afterwards become putrid: in that case it will change the colour of paper suspended over it, but nevertheless paper dipped in it will continue to show an alkaline reaction after being dried.

Another character of urine that has undergone this change is its

turbidity, a condition which filtering will not remove. The microscope shows that its cause is the presence of innumerable minute plants, occurring singly, or two or more in chains, or in a group with mucus as zooglœa. It is by these microphytes—*Micrococcus ureæ*—described by Pasteur and by Kohn, that the ammoniacal fermentation is brought about, probably immediately by a ferment which they secrete, just as alcoholic fermentation is brought about by the yeast plant.*

Urine placed in perfectly clean vessels, and guarded from the approach of microbes from without, can be kept free from this change for an indefinite length of time; and the same is the case in the body. It was formerly supposed that mere stagnation of urine in the bladder, or in the pelvis of a kidney, suffices to render it ammoniacal, especially if it contains mucus or cast-off epithelium; but that this is not the case has been proved by experiments on animals, for when the neck of the bladder or the ureter has been ligatured the urine has remained acid.

In too many cases the surgeon has introduced the microphytes into the bladder by using catheters which had not been cleaned with the scrupulous care necessary to render them aseptic.†

It is perfectly true that stagnation of urine in the bladder favours its decomposition. Cohnheim found that fluid containing bacteria may actually be injected into the bladder of a healthy dog without ill effects, because they are all expelled the next time that the animal micturates. And in men, when this change has once occurred in the urine, nothing tends so much to keep it up as an inability on the part of the bladder to empty itself completely; so that some of the putrid fluid is always left behind to induce the same process in that which is afterwards secreted. Moreover, formerly, when catheterism was constantly practised without any antiseptic precaution, the urine seldom became ammoniacal unless either the bladder was paralysed or the urethra in some way obstructed.

The fact no doubt is that septic as well as specific microbes differ in their effects according to quantity as well as quality. One bacterium or micrococcus can scarcely cause serious effects, just as one spermatozoon is unlikely to impregnate an ovum. Unless there are numerous infective microphytes, they cannot excrete large doses of toxin, and a small number would probably be effectually destroyed by healthy phagocytes. But repeated and numerous invasions will end by overpowering resistance and establishing a permanently diseased condition.

In some cases the putrefactive change takes place in the urine when no instrument has been passed. An abscess may have opened into some part of the urinary passages, and brought streptococci with the pus. Or bacteria may have found their way along the urethra from outside, possibly through a layer of mucus which may have been left on the surface of the meatus in the act of micturition. Or conceivably they may have come from the blood through the renal glomeruli.

One effect of the occurrence of ammoniacal fermentation, whether in the body or out of it, is at once to precipitate the earthy salts, chiefly tribasic calcic, and ammonio-magnesian phosphates, together with some magnesian phosphate, and also occasionally lithates of soda and of ammonia.

* See a note by Dr A. S. Lea in the 'Physiological Journal' in 1885, vol. xi, p. 10.

† This danger was pointed out by Traube in 1864. The case which drew his attention to it was that of a man whose bladder had apparently been distended for two years, but whose urine was clear and acid when it was first drawn off by a catheter, whereas the next day it was turbid, and within six days became ammoniacal and slightly fœtid.

The lithate of soda appears as round or irregular masses, from which project spiny crystals, straight or curved, like horse-chestnuts or the "morning star" of mediæval warfare; the lithate of ammonia as opaque, globular bodies, or very small, slender dumb-bells, which may lie across one another, or be united into a rosette.

The precipitate consisting of these various salts is usually white, but Roberts records urate of ammonia in putrid urine of a beautiful violet hue.

It is often important to determine whether the alkaline fermentation is limited to the urine in the bladder, or whether the bacteria which produce it have made their way up into the ureters and into the renal pelves. Sometimes this may be made out by carefully washing out the vesical cavity, and drawing off a few minutes later the first few drops of urine that collect. If this is found to be acid the change takes place only in the bladder. Or one of the ureters may be catheterised, and thus the renal pelvis in fault determined.

How quickly the reaction may be reversed is well shown when there is extroversion of the bladder, so that the orifices are exposed to view. In a case of this kind, Dr Owen Rees, many years ago, found ('Croonian Lectures on Calculous Disease,' 1856) that the urine as it flowed out from the orifices possessed its natural amount of acidity; but when reddened litmus paper was applied a quarter of an inch lower down, so as to test the urine after it had passed over that short distance of mucous membrane, its blue colour was restored. This result, however, he attributed, not to ammoniacal fermentation (for which there was not sufficient time), but to the admixture of alkaline exudation from the reddened and inflamed surface.

If cystitis is not already present before putrefaction of the urine in the bladder begins, its occurrence is not long delayed; and the exudation of pus quickly follows. This, however, does not retain its usual characters, but is converted by the ammonia into a viscid, semi-transparent mass, which often blocks the urethra, and causes the patient severe pain. In a vessel it collects at the bottom, and coheres so firmly that when the urine is poured out it does not separate, but glides away as a gelatinous mass, which hangs from the vessel like a liquid rope.* The cystitis is often intense, and may cause sloughing of large tracts of mucous membrane. Moreover, the inflammatory process often extends along the ureters, and reaches the kidneys, as will be described further on.

In this way ammoniacal fermentation of the urine becomes indirectly the cause of death. It is therefore a matter of the highest importance to prevent its occurrence, and to check it whenever it shows itself. The strictest antiseptic precautions should be observed whenever a catheter is introduced, and this should never be done without necessity, particularly when there is any difficulty in emptying the bladder.

If the change has already taken place before the patient is seen, the bladder should be washed out regularly with warm water containing borax and boracic acid. Roberts found that the urine may sometimes be restored to its natural condition by making the patient drink large quantities of fluid at regulated intervals, so as to keep the bladder washed out by the renal secretion. We have often kept the urine sweet and acid, or restored it after it had become ammoniacal, by giving salicylate of soda or benzoate of soda in ten- or fifteen-grain doses three or four times a day.

* The same ropy condition is seen in urine containing pus, or in pus itself, when liquor potassæ is added to it in a test-tube; it is the effect of the alkali, not of ammonia in particular.

Ammoniacal urine without sepsis.—It would seem that an ammoniacal state of the urine, at the time when it is voided from the bladder, is not in itself a proof of the occurrence of putrefaction. At least Roberts observed this in two cases of advanced Bright's disease without there being any evidence of delay in evacuation, and without any part of the urinary passages being afterwards found inflamed at the autopsy. Moreover, long ago Graves recorded two cases, in each of which the urine, although free from any smell of putrescence, contained a large quantity of carbonate of ammonia.

Non-septic bacilluria.—Even the presence of bacteria in the urine is not in itself proof that the ammoniacal fermentation has taken place, or is about to take place in it. For Sir William Roberts, in the 'Transactions of the International Medical Congress,' held in London in 1881 (p. 157), related several cases in which the urine when voided was opalescent, and full of micrococci and moving rods. In two of the cases it had a disagreeable odour; but it was acid in reaction, and showed no greater (or perhaps less) tendency to decomposition than healthy urine. Moreover, in the course of about twenty-four hours the bacteria sank slowly to the bottom, leaving the urine itself clear, and it retained its acidity for several days afterwards. The microbes seemed to be incapable of multiplying in the urine, even when it was kept at blood-heat. Two of the patients were women, and one of them had no symptoms beyond a slightly increased frequency of micturition, especially at night. The others were men, and they had long suffered from scalding pain in passing water, with frequent desire to micturate. The administration of salicylate of soda, in doses of twenty to thirty grains twice or thrice daily, usually proved effectual; the urine became free from bacteria within a few days, and all the symptoms disappeared.

A similar case occurred in the clinical ward of Guy's Hospital several years ago. A boy who was under the writer's care for another disease, without any symptoms of renal or vesical disorder, passed urine which contained rod-shaped bacteria, when examined immediately after it was voided. The urine was not ammoniacal, and in all other respects was normal.

Sarcinæ.—Several observers have recognised the presence of sarcinæ in the urine, sometimes in sufficient numbers to form a greyish-white amorphous deposit (p. 264). They resemble those derived from the stomach, and may occur in either acid or alkaline urine. The patient is generally troubled with vesical or renal symptoms, but these are perhaps attributable to some concurrent disease, such as stricture or enlargement of the prostate. In a case recorded by Munk the sarcinæ were abundant in the summer, but almost wholly disappeared in the winter, notwithstanding that the patient, being paraplegic, was bedridden. Dr Bateman, of Norwich (cited by Dr Beale), met with a case in which the urine on four separate occasions contained sarcinæ for a few days at a time.

Kyestine (from *κῡσις*, gestation) is a name given to a product of decomposition which shows itself occasionally in alkaline urine on the second or third day. It was first observed in cases of pregnancy, and was supposed to be peculiar to that condition; but this is now known not to be the case (Braxton Hicks, 'Guy's Hosp. Reports,' 1861). It forms a thick scum upon the surface, and consists of crystals of ammonio-magnesian phosphate and bacteria—the ordinary products of ammoniacal decomposition.

In some cases urine, with kystine in large quantity, is said to resemble chyluria, for the urine is then "milky" throughout; but no albumen or fat is present.

HÆMOGLOBINURIA.—The urine may be reddened by the colouring matter of the blood, and yet no red discs be found in it. This condition must be carefully distinguished from hæmaturia, where blood-corpuscles are present; which will be considered in the chapter on calculi, tubercle, and cancer of the kidneys, for these diseases are its most frequent cause.

Characters.—Urine containing hæmoglobin is sometimes of a pinkish hue, usually dark red or chocolate-brown, and occasionally almost black. The patient often compares it to strong tea or to porter. It is clear and transparent when passed, but as it cools it throws down a thick sediment of lithates having a chocolate colour. Or the hæmoglobin itself may form a more or less abundant precipitate. With the microscope there are often only amorphous granules to be seen, but in some cases a few shrivelled or altered blood-discs are visible. It must be remembered that the hæmoglobin may appear in the form of rounded reddish-yellow drops, looking not unlike red discs, but variable in size and arranged in rows like the beads of a necklace. It may also be moulded into casts of the renal tubules, which have an opaque granular appearance and a reddish-brown colour; but with few, if any, exceptions, there is no true hyaline cast of fibrin enclosing the pigment. Sometimes, but very rarely, it assumes the form of crystals. Oxalate of lime crystals are not infrequently met with, and their presence is in certain cases so constant that it seems likely to be more than accidental.

When urine containing hæmoglobin is heated, it yields a coagulum of a brownish-red colour, which floats on the surface of the urine. This coagulum is said to be formed solely by the globulin of hæmoglobin itself, no serum-albumen being present; for Roux, quoted by Cohnheim, found that there was enough iron in the urine to correspond with all the proteids.

If there is any doubt as to the nature of the colouring matter, the chemical tests of hæmoglobin are applied; or with the spectroscope a characteristic appearance is obtained. This consists in the presence, not only of the well-known absorption bands of oxyhæmoglobin in the yellow and in the green between the solar lines D and E, but also of a third broad band in the red between the lines C and D, but nearer to C. This third band belongs to a modification of hæmoglobin—Methæmoglobin. Hoppe-Seyler believed it to be intermediate in oxidation between oxyhæmoglobin and reduced hæmoglobin, while Sorby concluded that it contained more oxygen than oxyhæmoglobin. But Gamgee, confirmed by Hufner and Külz, has satisfactorily proved that the oxidation of methæmoglobin and oxyhæmoglobin is the same, but that in the former the excess of oxygen is much less loosely combined than in the latter. In most cases of hæmoglobinuria both forms of hæmoglobin are present, and, in many, hæmatin or urobilin as well.

There is no doubt that hæmoglobin is set free in the blood before its excretion by the kidneys, inasmuch as Marchand has shown that in poisoning by chlorate of potass (which we shall find to be one of the causes of hæmoglobinuria) the blood itself yields a spectrum showing the band between C and D. Moreover, blood has been taken by cupping from patients

with hæmoglobinuria, and after clotting, the serum has been seen to be red instead of yellow.

Morbid anatomy.—In fatal cases of hæmoglobinuria the kidneys are found of a deep chocolate colour, with radiating striæ of a still darker tint. With the microscope the renal tubes are seen to be completely plugged with masses of hæmoglobin. These concretions have also been observed by Dr Bridges Adams in the Malpighian capsules—a point of some importance as showing that the hæmoglobin is excreted through the glomeruli rather than the epithelium of the tubes. In one instance Hofmeier noticed that the medulla of the femur was brown. Ecchymoses have also been observed in the spleen, the stomach and intestines.

Pathology.—Hæmoglobinuria is probably always the result of a disintegration of some of the red discs within the circulating fluid, or at least of the escape of their hæmoglobin from their “stroma.” It is not due to any primary alteration in the structure, nor even to perversion of the functions of the kidneys.

The causes of the change in the blood are various, so far as we know.

Transfusion of blood from one kind of animal to another—as when, for example, lamb’s blood is thrown into the veins of a dog—will produce hæmoglobinuria in the dog; apparently, the foreign red discs are broken up by the blood-serum of the animal into which they are introduced.

Cohnheim found that hæmoglobinuria is a common symptom after extensive burns, if they are not too rapidly fatal.

Hæmoglobinuria has been observed in cases of heat-stroke. It has been recorded by Immermann as a complication of enteric fever during a relapse (‘*Deutsches Arch.*,’ xii), and by Heubner in a case of scarlet fever on the twentieth day (*ibid.*, xxiii). In its paroxysmal form it is frequently the result of exposure to cold; it is probably due to malaria in many cases, and to other forms of vaso-motor disturbance in many more.

Toxic hæmoglobinuria.—Among the various poisons which have been known to cause it, either in animals or in man, are arseniuretted hydrogen, hydrochloric and sulphuric acids, chlorate of potass, nitro-benzol, naphthol, and carbolic acid (phenol). Eitner recorded a fatal case (‘*Berl. klin. Woch.*,’ 1880) in which it was set up by the inunction of pyrogallie acid for severe psoriasis.

Eitner also recorded four cases due to the entrance of *arseniuretted hydrogen* gas into the air-passages; the sufferers were a professor of physics and three of his students. The professor had two attacks of hæmoglobinuria, separated by an interval of some days, and resembling the paroxysmal form of the affection in all respects except perhaps in having a rather longer duration. He and his pupils had been repeating Tyndall’s experiment of inhaling hydrogen gas, for the purpose of showing that it alters the pitch of the voice; and the zinc used in generating the hydrogen was impregnated with arsenic.

From a clinical point of view, *chlorate of potass* surpasses in importance all the other toxic causes of the affection. Hofmeier collected (‘*Deutsche med. Woch.*,’ 1880) no fewer than twenty-seven cases, all but four of which proved fatal. Some of the patients took the salt by mistake for sulphate of soda or for some other saline aperient. But for most of them the chlorate of potash was prescribed, and the mischief arose either from the dose being too large or too frequently repeated, or else from their swallowing large quantities of a solution intended only as a gargle. In

children it would appear that from one to two drachms in the course of twenty-four hours is a dangerous quantity; in adults perhaps from two or three drachms upwards.*

The symptoms of toxic hæmoglobinuria differ in intensity, but seem always to belong to a common type. In the most marked and severe cases the patient is seized with a rigor; vomiting and diarrhœa then set in; he becomes collapsed and cyanosed, falls into a state of stupor, and so dies.

Infantile hæmoglobinuria.—Spontaneous hæmoglobinuria occasionally occurs as an epidemic among infants. Winkel related ('Deutsche med. Woch.,' 1879) a remarkable outbreak, which occurred in that year, in the lying-in hospital at Dresden, where, between March 20th and April 29th, twenty-four new-born infants were attacked by it, of whom twenty-three died. The symptoms were in every instance similar; the child, generally about the fourth day after its birth, became suddenly cyanosed and collapsed, with cold extremities; and it usually succumbed within thirty-six hours. There was seldom any diarrhœa or vomiting, but the skin had in many cases an icteric tinge. The respiration and the pulse were very rapid; but the temperature in the rectum was scarcely raised. The superficial veins became visible as dark lines. The urine was brown and showed the other characters of hæmoglobinuria. Death was preceded by convulsions. On *post-mortem* examination the kidneys were dark brown, and their papillæ contained plugs of hæmoglobin. The chief other morbid appearance was an enlargement of the mesenteric lymph-glands and of the spleen, the latter being tough and of a brownish-red colour. Every effort was made to discover the cause of this infection, but without success. Winkel described it as *Cyanosis infantilis icterica perniciosa cum hæmoglobinuriâ*. No doubt the symptoms were due to an intense and rapid disintegration of red corpuscles within the blood-vessels; but why this should have occurred in numerous infants in succession—themselves apparently healthy at birth, the offspring of healthy mothers, and in a hospital where no like disease had been observed before—remains a mystery.

PAROXYSMAL HÆMOGLOBINURIA.†—In marked contrast with this fatal infantile form of hæmoglobinuria is one to which adults as well as children are liable, and which recurs again and again without danger to life or severe disturbance of health.

The first account of this remarkable disorder as distinct from hæmaturia was (as Dr Wickham Legg has shown in a valuable paper in the tenth volume of the 'St Barth. Hosp. Rep.') an article by Dressler, published in 'Virchow's Archiv' as long ago as 1854, under the title "Intermittent Albuminuria and Chromaturia." In this country Dr Dickinson, Dr George Harley, and Dr Hassall each published typical cases in the same year (1865). Dr Harley's single and Dr Dickinson's four cases appeared in the forty-second volume of the 'Med.-Chir. Trans.;' Dr Hassall's in the 'Lancet' (ii, 368). A case was described by Sir William Gull in the 'Guy's Hospital Reports' for 1886 as "intermittent hæmatinuria;" but the best name

* In many instances the disease for which the chlorate of potass was ordered has been diphtheria, and one cannot help fearing that in these cases the salt may have produced ill-effects without their real cause having been suspected, the state of the urine having been attributed to diphtheritic nephritis, and the severe constitutional symptoms regarded as indicative of "collapse."—C. H. F.

† *Syn.*—Intermittent chromaturia (Dressler)—Winter hæmaturia—Intermittent hæmaturia—Paroxysmal or periodical hæmaturia—Hæmatinuria—Methæmoglobinuria.

appears to be "paroxysmal hæmoglobinuria," used by Dr Pavy in 1866 and 1868. Greenhow, Druitt, and many others at home and abroad afterwards wrote on the subject. Lichtheim published three cases in 1878 (No. 134 in 'Volkmann's Sammlung') under the title "periodical hæmoglobinuria."

Etiology.—In some few instances no cause can be discovered. Two such cases occurred in Guy's Hospital during the height of summer, in June, 1876, and in August, 1880. In neither case were there any subjective sensations except pain in the loins, associated with the discharge of urine, which was almost black with hæmoglobin.

In the great majority of cases, however, the affection is due to one and the same exciting cause, namely, *cold*. Sometimes the degree of cold that causes an attack (especially the first attack) is excessive, or, at any rate, far beyond that to which the individual is accustomed.

The late Dr Fagge observed hæmoglobinuria for the first time in his own person one day after fishing in Scotland in a biting wind, and his next seizure occurred some months later, after skating without a great-coat. But he subsequently became liable to the affection when the provocation appeared inadequate to produce such a result, as, for example, after standing four or five minutes at a railway station on a foggy morning, or riding two or three miles in a hansom cab. In some instances it is stated that all but the early attacks have been independent of cold. Thus a patient of Sir William Roberts said that he was just as bad in the summer as in the winter. As a rule, however, the affection ceases entirely during the warm part of the year, returning in the cold season, perhaps for many years in succession. That this is entirely a question of temperature was shown by Rosenbach, who, by means of a cold foot-bath, brought on an attack during the summer in a person liable to the disease. Roberts relates two cases, in each of which there were as many as three daily attacks. But commonly they recur at much longer intervals and quite irregularly.

They are most apt to take place in the *morning*, when the temperature of the body is lower than in the afternoon and the evening. It is especially when the patient is exposed to cold directly after breakfast, before the meal has been digested, that hæmoglobinuria is apt to show itself; and a cup of hot beef-tea on first waking may prove an effectual preventive. An outbreak never comes on during the night, when the patient is warm in bed. Bodily or mental exhaustion, as from sexual intercourse, or from study late at night, favours the occurrence of an attack. It also may follow the free use of wine at dinner, probably from its relaxing the cutaneous vessels. Hæmoglobinuria is often seen in persons whose fingers are apt to "go dead"—*digiti mortui*,—cases closely allied to Raynaud's disease (p. 600).

The previous occurrence of *ague* has been noted in some instances, and it seems probable that this is a more or less direct cause of hæmoglobinuria, as it is of splenic leuchæmia. The writer had an instance of an elderly man with ague and enlarged spleen, who passed hæmoglobin in his urine during the cold stage of each paroxysm.

Syphilis was present in a case observed by Ehrlich; and a patient who was under the author's care in the clinical ward of Guy's Hospital in 1882, and who did not improve under the usual remedies, was subsequently cured by Dr Moxon with mercurial treatment; in that instance the spleen was much enlarged. Dr Wickham Legg met with a similar case, and of late years several cases have been reported in which hæmoglobinuria followed syphilis.

Fleischer recorded ('Berlin. klin. Woch.,' 1881) an instance in which a soldier was attacked every time he marched for an hour or two.

The disease is much more common in men than in women. It is most apt to occur in young adults, and may continue up to the age of forty or fifty; but Dressler's patient was a boy between ten and eleven, and Dr Greenhow's a child only two years old, while Dr Druitt was above fifty when he was first attacked.

Seven cases of this remarkable disorder have been under the present writer's care. They all occurred in men—the youngest fourteen years old, the eldest above sixty, and the remainder between nineteen and thirty-three. Two were pretty clearly of malarial origin, associated in one patient with dysentery contracted in the Zulu campaign, and they were both benefited or cured with quinine; two were complicated by gangrene of the ears or fingers and toes. One case occurred in a schoolboy three times at intervals, the first after bathing; and the third attack was marked by high fever.

Symptoms.—One of the most constant is a feeling of languor and weariness, with a disposition to yawn. With this there may be a feeling of chilliness, so that the patient is inclined to huddle up by the fire. The fingers and the toes—some or all of them—may turn white and dead. A graphic account of the disease was given by the late Dr Druitt in the 'Medical Times and Gazette' for 1873; it is now no secret that he was himself the patient. He describes his palms and soles as becoming "cold, wet, blue, and cramped, like those of a cholera patient." At other times he felt numbness of the right foot and the left hand, without coldness; or his nose, or some part of his cheek, would become first pale, then red, then purplish, and at last almost black. On several occasions the ears not only turned livid during the seizures, but failed to regain their natural appearance afterwards, so that a reddish-brown eschar formed along the edge of the helix, leading to a slight but permanent loss of its substance.

Until these symptoms have passed off and been succeeded by a feeling of warmth and comfort over the whole body, there may be nothing to show that the urine is other than normal. There is often no desire to micturate. In Dr Fagge's own case it was sometimes only after the lapse of several hours that any water was passed, when he had almost forgotten that he had been chilly; so that he was quite surprised to find it was like chocolate or porter. In other cases the bladder is irritable, and on some occasions Dr Druitt suffered great pain in the bladder, and was obliged every half-hour to void scarlet urine. Sometimes, but not always, there is pain in the back, sometimes radiating across the abdomen or down the thighs. Retraction of the testes has also been recorded.

The urine, beside methæmoglobin, often contains a few red discs, more abundant leucocytes, blood-casts, and sometimes crystals of calcic oxalate. The last constituent is certainly not the cause of the attack, for the crystals are frequently present without an attack, and they are only occasionally present during a paroxysm.

That hæmaturia with passage of abundant red blood-discs is often associated with hæmoglobinuria is certain. Most often the hæmaturia precedes the latter, sometimes they alternate in successive attacks. Albumen is occasionally present, but more frequently, as first stated by Gull, globulin.

The patient's temperature during a seizure of hæmoglobinuria is, as a rule, normal. Dr Druitt says that during the course of his illness (which had then lasted six years) he had severe attacks of remittent pyrexia, in which

the thermometer would rise to about 103° in the evening, and fall to 100° in the morning; during these periods jaundice was always present, but the urine never contained any blood-colouring matter. His pulse generally fell to 55 or 50 when the hæmoglobinuria was about to occur. In one of the writer's cases the patient, a robust youth of nineteen, had pyrexia reaching 103° on one, and exceeding 104° on another occasion, with a pulse of 90 to 100 during the attacks.*

One of the most remarkable features of the disease is the rapidity with which, after a seizure, the urine regains its normal characters. The patient may once or twice, or oftener, have voided a fluid like porter; and what he passes an hour or two later is perfectly clear and pale. Even in Roberts's two cases, in which there were two or three attacks in the course of a single day, colourless urine was secreted in the intervals. It is also curious how completely the appetite is unaffected; a patient who had a well-marked attack in the morning may at one o'clock eat a hearty dinner as usual, sometimes even before he has discharged from his bladder the dark urine formed during the paroxysm.

Pathology.—It has now been ascertained that the starting-point of paroxysmal hæmoglobinuria (as we found in its other forms) is disintegration of a certain—probably a small—proportion of red discs in the circulating blood. Microscopical examination of the blood does not always show any marked change in it, as the writer and several other observers have found. Küssner, however, took blood with a cupping glass ('*Deutsche med. Woch.*,' 1879) from a patient on six occasions during a seizure, and each time found that the serum was of a ruby-red colour, whereas at other periods it had the normal yellowish appearance. A still more striking experiment has since been performed by Ehrlich (*ibid.*, 1881). Having under observation a woman who was liable to the disease, he bound an elastic ligature round one of her fingers, which he placed for a quarter of an hour in ice-cold water, and afterwards for the same length of time in tepid water. Such an experiment on a healthy person produces no change in the blood, but in the patient in question it caused the red discs to break down in considerable numbers. When a drop of blood from the finger was placed in a capillary tube and allowed to coagulate there, the serum was distinctly seen to be reddened; and under the microscope the blood was found to contain "phantom discs—stromata or *æcoids* of discs that had more or less completely lost their hæmoglobin—though there were also many normal corpuscles, as well as pœcilocytes and microcytes. The disease therefore seems to originate in an undue sensitiveness to cold on the part of certain of the red blood-discs.

In some patients, however, there may be observed a symptom which at first sight seems inconsistent with this view of the disease. It is that attacks of chilliness, which fall short of the degree of severity necessary to bring about an excretion of hæmoglobin, render the urine albuminous. This fact has been verified on many occasions, the patients being perfectly free from any indication of Bright's disease. It was markedly the case in the youth of nineteen whose case was mentioned above; and indeed was observed in the earliest case published, that by Dressler in 1854.

Possibly when the disintegration of red discs occurs only to a moderate extent, all the hæmoglobin which is set free splits up into globulin and

* The second attack had been recorded by Dr Penny, Medical Officer to Marlborough College, and was also accompanied by pyrexia.

hæmatin; and the albumen (or globulin) may be excreted as such by the kidneys. Indeed, it is probable that even in ordinary cases of hæmoglobinuria a part of the hæmoglobin is decomposed, so as to form a pigment resembling bilirubin, if not identical with it. The evidence of this is the sallow bilious appearance presented by patients who frequently suffer from the disease, and sometimes apparent during or after a single severe attack.

In not a few cases hæmoglobinuria follows or is replaced by hæmaturia, and occasionally both conditions are accompanied by the symptoms of acute nephritis, as in a remarkable case recorded by Dr Donald Hood in the 'Lancet' (October 4th, 1890).

According to Dr Noel Paton hæmoglobinuria is in some cases accompanied by large increase in the excretion of urea, and in the secretion of bile.

Prognosis.—Paroxysmal hæmoglobinuria has never been known to destroy life, nor to entail very serious consequences, though one can hardly doubt that if the attacks were allowed to recur frequently, the patient's health would at last break down.

The attacks may continue for many years in succession, or they may cease after a few weeks or months. Dr Druitt had suffered for six years when he published the account of his case; in another recorded instance the disease ran on for eleven years. In twenty cases collected by Roberts there was no fatal issue, and twelve were well when their cases were published.

Treatment.—During an attack the essential requisite is warmth. The patient should be put to bed, and given a basin of hot soup. When there is severe pain in the back, it may be relieved by the application of mustard, or by dry cupping, or (as Dr Druitt found) by full doses of extract of hyoscyamus.

But the important object is to prevent the recurrence of the disease. The utmost care should be taken to avoid exposure to cold in the early part of the day. The meals should be so arranged that there may be food in the stomach at a time when a cold journey is to be made, or when any unaccustomed or anxious task is to be gone through. The clothing must be warm; flannel underclothing, fur-lined boots and gloves, wash-leather waistcoats, and cork soles are all useful.

The question of removal to a warm climate during the winter should in severe cases be seriously considered; Dr Druitt himself went to Madras, where he escaped the disease almost entirely. During the summer a more bracing air is advisable.

The patient should not spend his evenings in heated rooms, and should go to bed early. He should have nutritious food in the latter part of the day, but little or no alcohol; for its effect is an immediate stimulation that rapidly passes off.

The one medicine that seems to have a marked effect in warding off the attacks is *quinine*; but it must be given in full doses. It sometimes proves perfectly successful, so that the patient becomes again able to live his usual life without fear of his complaint returning. But in severe cases, like that of Dr Druitt, no permanent benefit can be obtained by it, even when it is used in such quantities as to cause ringing in the ears and other disagreeable effects. Salicine, the tincture of iron, and arsenic, may each be of service in their turn. Chloride of ammonium is said to have done good in one instance. If syphilis is present, iodide of potassium or the bichloride of mercury will sometimes work wonders.

Blackwater fever.—Hæmoglobinuria is a constant symptom of the remarkable and apparently only recent endemic disease known as West African or blackwater fever. Though most common in Western Equatorial Africa, it is present elsewhere in that continent and in Madagascar, and also in the West Indies and Spanish Main. It is distinct from yellow fever and from malarial fever, although Crosse in West Africa, and others in Texas, where “redwater fever” is supposed to be identical with the African disease, have found a plasmodium in the blood-discs. After a rigor, fever follows, with icterus and hæmoglobinuria. (See vol. i, p. 391; also Dr Crosse’s Notes on the malarial fever on the River Niger, 1892; and Dr S. M. Copeman’s article in ‘Allbutt’s System,’ 1897, vol. ii, p. 742.)

Raynaud’s disease.—Another remarkable condition in which hæmoglobinuria is a frequent, though not a constant, symptom, is that of local “asphyxia” (*i. e.* cyanosis) with symmetrical gangrene of the extremities (*acro-sphacelus*), which was described by the late Dr Maurice Raynaud in 1862, and which, in its most severe form, is known by his name.*

The age of the patients, the coldness of the extremities, and the mortification of the ears, or fingers, or toes, suggest this relation; and in typical cases of Raynaud’s disease hæmaturia or hæmatinuria has been frequently observed. Curiously enough, in the twenty-five cases on which his monograph is based (five of his own and twenty collected from previous writers), no mention is made of the state of the urine, and since then some apparently typical cases have been quite free from any abnormality of the urine.

There are other points of difference: of Raynaud’s twenty-five cases, twenty occurred in women and only five in men. Some of the most severe cases of hæmoglobinuria show no tendency to gangrene, and the patient’s circulation is perfect in the intervals between the attacks. On the other hand, some of Raynaud’s cases are not accompanied by any change in the urine, or the change is to true hæmaturia. At present it is perhaps best to keep together the cases of hæmoglobinuria (and even of hæmaturia) which are decidedly paroxysmal and unaccompanied by gangrene, and to reserve the title of Raynaud’s disease for the continuous and severe cases described by him. Of late years the relation of Raynaud’s disease to syphilis has been generally admitted; the relation is probably more than a coincidence, as it may be also in the case of hæmoglobinuria.

Apart from hæmoglobinuria, the natural allies of Raynaud’s disease are cold and pale extremities (*digiti mortui*), blue ears and noses, chilblains and mottled limbs.

* “De l’asphyxie locale et de la gangrène symétrique des extrémités” (‘Thèse de Paris,’ 1862). See a case of Prof. Billroth’s in the ‘Wiener med. Wochenschrift,’ 1878, reported in the ‘Lond. Med. Record’ of that year, p. 343; also Dr Southey’s papers in ‘St. Bartholomew’s Hospital Reports,’ vol. xvi, and in the Clinical Society’s ‘Transactions’ for 1883; Dr Thos. Barlow’s communication in the same volume, with the sequel to his cases in 1885 (vol. xviii, p. 307); Dr T. C. Fox’s two cases (*ibid.*, p. 300), Weiss, ‘Ueber symmetrische Gangrän,’ and Hutchinson, ‘Med. Press and Circular,’ Oct. 16th, 1891.

BRIGHT'S DISEASE

"Crescit indulgens sibi dirus hydrops,
Nec sitim pellit, nisi causa morbi
Fugerit venis et aquosus albo
Corpore languor."
HOR., *Carm.*, II, ii.

History—The two chief forms—Additional varieties—Common characters of all.

Albuminuria: its detection and estimation, pathology, and significance.

Casts: their structure, varieties, and diagnostic meaning.

Renal dropsy: its pathology, its primary and secondary form—*Albuminuria without Bright's disease*—*Dropsy without albuminuria*.

Serous inflammations—*Colitis*—*Dermatitis*.

Retinitis and retinal hæmorrhage—*Optic neuritis and atrophy*.

Changes in the heart and arteries: the renal pulse, hæmorrhage.

Uremia: *eclampsia*, *coma*, *amaurosis*, *vomiting*, *dyspnœa*; theory of *uræmia*.

Anæmia.

Parenchymatous or tubular Nephritis, acute and chronic—causes—anatomy—three stages—symptoms—event and prognosis.

Lardaceous disease generally—its chemistry, causes and distribution—anatomy of the lardaceous kidney—symptoms—diagnosis—treatment.

Cirrhosis of the Kidney—pathology—causes—anatomy—symptoms and clinical aspects—*Consecutive renal cirrhosis*—its relation to local disease of the bladder, urethra, and uterus—*Hypertrophic cystic degeneration*.

Mutual relation of the several forms described—*Geographical and comparative distribution*—*General treatment and prognosis of Bright's disease, and of its complications*.

DROPSY was well known to the ancients both as *anasarca* and *ascites*;* it was associated with the effects of drink and afterwards with disease of the heart; but renal, as distinct from hepatic and cardiac, dropsy was only recognised about ten years after Laennec's great discovery, although its characters must often have been noted, as in the quotation at the head of this chapter.

In 1827 Dr Richard Bright published, in his 'Reports of Medical Cases,' the fact that in many cases of dropsy there are well-marked lesions of the kidneys, and that the urine is albuminous. His discovery was of the connection between the clinical symptoms of dropsy and albuminuria and the anatomical fact of renal disease. That dropsy is often attended with the

* Aretæus, who flourished in the first century A.D., distinguishes *tympanias*, or "windy dropsy," from *ascites*; and a general swelling of the body with white, thick, cold phlegm (*phlegmatias*), from *anasarca* (ὕδρωψ ἀνὰ σάρκα), which is defined as a liquefaction of the flesh into a thin watery humour ('Morb. Chron.,' lib. II, cap. i).

Celsus (probably a contemporary) writes much to the same effect, that there are three kinds of dropsy: "primum *tympaniten*, secundum *leucophlegmasiam* vel *ὕδωσάρκα* (not *anasarca*), tertium *ἀσκίτην* Græci nominaverunt" (lib. III, cap. xxi).

presence of albumen in the urine had been observed a short time before by Dr Wells of St Thomas's Hospital (the author of the 'Essay on Dew'), by Dr Blackall of Exeter, and Dr Osborne of Dublin; indeed, it had been noticed in the previous century by Cotugno and by Cruikshank. But notwithstanding that Wells and Blackall each made autopsies in which they found the kidneys "remarkably hard," they both regarded the change in these organs as accidental. Blackall's hypothesis was that serum was excreted by the kidneys, because it was vitiated from having already formed part of a dropsical exudation, and having been reabsorbed into the blood. To Bright belongs the full credit of first showing the real and constant relation of disease of the kidneys both to dropsy and to albuminuria, and throughout the world his name is justly associated with renal dropsy.

After the publication of his first cases and drawings, two wards at Guy's Hospital were devoted to further investigations into the new disease, and Dr Bright, with the help of Dr G. H. Barlow and Dr Owen Rees, made the further observations which were printed and illustrated in the first volume of the 'Guy's Hospital Reports' (1836), and in the fifth of the second series.

Varieties of the disease.—The lesions of the kidneys described by Bright are far from being uniform. He himself and his fellow workers admitted three forms of the affection, leaving it an open question whether or not they should be regarded as separate diseases.

The three anatomical types of morbus Brightii recognised by Barlow, Rees, and Wilks, were the *large red kidney*, the *large white kidney*, and the *small red kidney*.

Rayer, who first followed Bright's footsteps in France, divided what he called *néphrite albumineuse* into a much greater number of varieties (1839-41). In 1851, Professor Frerichs, then of Breslau, expounded the doctrine that the diverse appearances presented by the kidneys belong to successive stages of a single morbid process. This view, however, was refuted two years later by Wilks, who showed in a paper in the 'Guy's Hospital Reports' (second series, vol. viii) that, under the name of Bright's disease, there are included at least two independent affections differing in their causes, in their mode of onset, their symptoms, and their course—"the large white kidney with considerable dropsy," and "the hard contracted kidney, often destitute of symptoms,"—and added a summary of twenty-three cases of the former, and thirty-three of the latter affection.

Wilks reasoned that since anasarca is particularly associated with the "large, smooth, white kidney," one ought, if that were an early stage of the "small, rough, red, mottled kidney," to obtain a definite history of precedent dropsy in each case in which the small red kidney was found after death. Frerichs, however, had not brought forward a single instance in which his sequence of events had occurred, nor were any furnished by Wilks's own experience. The statement that the large white kidney does not pass on into the small red kidney was perhaps put too absolutely; but in the main it is undoubtedly correct.

Among the distinctions pointed out by Wilks are the following:—That, whereas the large white kidney often results from scarlet fever, or from exposure to cold, the small red kidney is not traceable to either of these causes, but is in many cases associated with gout or plumbism; that whereas the former occurs in children and young adults, the latter is rare under five-and-thirty, and is not frequent before the age of fifty; that the clinical features found along with the former are an abrupt onset and

acute course, while the latter begins insidiously, and is slow in development; and lastly, that although either form may be attended with changes in the heart and arteries, such changes are far less marked in the former than in the latter kind of Bright's disease, which often appears clinically under the mask of cardiac symptoms, or of apoplexy due to rupture of an artery in the brain.

These arguments from anatomy, origin, and symptoms were greatly strengthened by Virchow, who showed, in 1858, that two distinct pathological processes are concerned in the two forms of Bright's disease, and that they attack two distinct anatomical elements of the kidneys. He demonstrated that in the large smooth mottled white kidney the secreting tubules, in the small red kidney the interstitial tissue, is mainly affected. Later observations no doubt have shown that this distinction is far from absolute; and therefore the two names proposed by Virchow, "parenchymatous nephritis" and "interstitial nephritis," can no longer be regarded as mutually exclusive. In both forms the tubes are sooner or later affected; in both there is more or less intertubular exudation; in both the cortex is the seat of the lesion; but the distinction in the origin, course, and result of the two processes is in most cases as marked as in their original seat. The histological origin in both forms is obscure, and mixed cases occur; but it remains true that the tubular parenchyma of the cortex is swollen in the large smooth kidney, whereas in the small rough kidney it is atrophied, as in other chronic interstitial inflammations.

Pathologically, therefore, as well as clinically, we must admit the broad and important differences above stated between the two principal forms of Bright's disease. As physicians, we are quite justified in regarding them as distinct affections when we find that they differ in their causes, in their symptoms, and in their clinical course.

Some writers have described separately "acute Bright's disease," "chronic Bright's disease," and "contracted or granular atrophy of the kidneys." Such a division, however, keeps apart cases that should be brought together, and it brings together cases that should be kept apart. An acute nephritis may be the result of scarlet fever, or of cold, or of pregnancy; but frequently it does not soon end either in recovery or in death, and becomes chronic. All such cases should be held to belong to a single form of Bright's disease; and since an affection of the tubal epithelium is a more or less constant feature, we may call them *tubular*, or *parenchymatous nephritis*. On the other hand, Ernst Wagner's "contracted or granular atrophy" includes two sets of cases that are essentially distinct. One set belongs to the originally chronic affection which produces the "small red kidney" of Bright and Wilks, the "raspberry" or "granular" kidney of later writers, *interstitial nephritis*, or *cirrhosis of the kidney*. The other far less numerous cases, only recognised of late years, are those in which a small contracted "granular" white kidney is proved by history and anatomy to be only the final stage of the large smooth kidney of tubal nephritis.

The large red, smooth kidney of Bright and his immediate followers is now regarded as only the acute or early stage of the large white or "mottled," smooth kidney which they described and figured; if the patient survives, but is not cured, the process goes through a final stage, that of the small contracted white kidney. The small red, rough, or cirrhotic kidney, on the other hand, is the result of a process chronic from the first, and distinct in its symptoms as well as its anatomy.

These two chief forms of Bright's disease, however, are not the only ones. In some cases of chronic dropsy and albuminuria the primary lesion is a *lardaceous degeneration* in the arterioles and in the glomeruli of the kidney. It is true that the lardaceous affection always becomes complicated sooner or later with parenchymatous or with interstitial lesions, or with both. But inasmuch as its causes are peculiar, and as there are also differences in its symptoms and course, it is desirable to keep it apart.

There are two other subordinate forms: one is the affection commonly known as *cystic disease of the kidneys*; the other is in its origin secondary to lesions of the renal pelvis or of the lower urinary passages, so that it may be termed "*consecutive Bright's disease*." Both are varieties of chronic interstitial degeneration, *i. e.* of renal cirrhosis.

It might be thought that the recognition of two independent forms of Bright's disease would render it advisable to drop the common designation. But, first, the more important characters belong to both. Secondly, in clinical practice one is not infrequently in doubt as to which form is present. Thirdly, we often meet with transitional forms:—the acute tubal nephritis is passing on to, but has not reached, the chronic stage of the large white kidney, as red hepatisation passes into grey; the granular atrophic organ is overtaken by an acute tubal inflammation, as an acute bronchial catarrh may supervene on chronic cirrhosis of the lung; lardaceous disease usually complicates tubal nephritis, or is complicated by it, and sometimes it is found associated with a granular contracted kidney.

A satisfactory definition of Bright's disease is therefore difficult. It certainly must not include temporary symptomatic albuminuria, such as accompanies the venous congestion of heart disease, or the secondary albuminuria of cholera, or the febrile albuminuria of erysipelas, diphtheria, or typhus. It must not include these or any other cases in which transitory albuminuria occurs without there being reason to suppose any organic change in the kidneys; nor those in which the presence of albumen is only due to that of blood, of pus, or of hæmoglobin. We may perhaps say that Bright's disease is primary diffused nephritis, attended with persistent albuminuria, with raised blood-pressure and changes in the arteries and the heart, and with more or less dropsy and anæmia. Its divisions we will treat as follows:

I. *Acute and chronic tubal or parenchymatous nephritis*.—This is attended with marked anasarca, and often by ascites or hydrothorax. The onset is sudden, when scarlet fever or cold is the cause, but in other cases it may be more gradual. In the acute stage the kidneys are swollen and red; when the disease has become chronic they are whitish yellow, and sometimes very large. Finally, they may shrink and become granular (small white kidney). The urine is at first dense, high-coloured, scanty, containing much albumin, tube-casts, and often blood. Later on, it may be pale, with a variable amount of albumin. Retinitis and uræmia are of frequent occurrence. In advanced cases the heart becomes hypertrophied, and the systemic arteries are thickened. This division includes the large smooth red and the large smooth white kidney, which are two stages of one pathological process, and also, as a third stage, a few cases of the small contracted kidney.

Glomerular nephritis is the name given by Klebs to the form of acute diffuse inflammation which follows scarlatina. The glomeruli are often most affected, but not always; the tubules also share in the change, and

also the intertubular connective tissue; so that it is best regarded as only an anatomical variety of acute diffuse nephritis.

The following varieties of nephritis with albuminuria are not accompanied by dropsy, and are best not included under Bright's disease.

Febrile diffuse nephritis is a secondary condition common in all cases of prolonged pyrexia. The kidneys are swollen and full of blood from active congestion, and on microscopic examination the epithelium of the tubes is in a condition of "cloudy swelling." This state is common to other secreting glands, and, though it produces albuminuria, subsides as the primary febrile state passes away. It appears never* to lead to chronic disease of the kidney. It is present occasionally during scarlatina (independently of post-scarlatinal nephritis), and frequently in typhus, enterica, erysipelas, diphtheria, pneumonia, and other fevers.

Congestive nephritis is the result of long-continued passive or venous hyperæmia of the kidneys, usually the result of chronic disease of the heart. The organ, at first only "hard" or tough "like india-rubber," becomes gradually "coarse," the capsule somewhat adherent, and the section of the cortex blurred. Hyaline casts as well as albumen appear in the urine, and finally the kidney would probably atrophy.

II. *Cirrhosis of the kidneys, or chronic interstitial nephritis*.—This is a slow and insidious affection, of which the chief known causes are gout, lead-poisoning, and probably drink. Occasionally it can be traced to scarlatina, even when there is no clear evidence of an acute nephritis having intervened or preceded it. It is very rare in early life, but begins to occur towards forty years of age, and beyond this it is frequent up to seventy. It gradually destroys the renal cortex until this may not be more than a line in thickness; the surface of the organ remains of a red colour, but it becomes very uneven and "granular." The urine is abundant, clear, pale, of low specific gravity; it contains only a small quantity of albumen, or there may be none at all for days together. Marked general dropsy occurs only when the affection becomes complicated with parenchymatous nephritis. Cardio-vascular changes are constantly developed, and reach an extreme degree. In many cases the patient dies with the symptoms of heart disease, including obstructive dropsy, which affects the dependent parts of the body. Uræmia is common towards the end. Cerebral hæmorrhage is another frequent cause of death. This division includes the great majority of cases of the small contracted red kidney. The two following sub-forms belong to the same category:

a. Consecutive Bright's disease.—This is seen as the result of such affections as stricture of the urethra, stone in the bladder, prolapsus uteri, compression of the ureters by an abdominal tumour, calculous pyelitis, and tuberculous disease of the kidneys. The kidneys become tough, hard, and whitish; they may be either of normal size or shrunken, either smooth on the surface or puckered by cicatrices, or granular. The general symptoms and the characters of the urine are those of renal cirrhosis.

β. Cystic disease of the kidneys.—The peculiarity of this affection is the presence of innumerable cysts of various sizes. Commonly, the kidneys are small and contracted, but sometimes they are sufficiently large to be felt as abdominal tumours during life. In its clinical features it resembles renal cirrhosis; but it is sometimes congenital, and due to malformation.

* Since writing the above, the writer has had one case of diphtherial albuminuria, which continued after recovery and developed into chronic Bright's disease.

III. *Lardaceous disease of the kidneys*.—This is caused by protracted suppuration or by syphilis. The kidneys give a characteristic reaction with iodine. Gradually they become very large, pale yellow, and waxy-looking. Finally, if the patient lives they may shrink and become granular. The urine is excessive in quantity, pale, and contains much albumen; but when there is also acute nephritis, it may be scanty and high-coloured. General dropsy is frequent. Cardio-vascular changes, retinitis, and uræmia are seldom observed. This is the form of Bright's disease discovered by Virchow after the two main types had been made out.

Before entering upon a separate and detailed description of these different forms of Bright's disease, we will consider certain symptoms and effects which belong, in a greater or less degree, to all of them in common:—Albumen and tube-casts in the urine, dropsy, and serous inflammations, colitis, retinitis, anæmia, cardio-vascular changes, hæmorrhages, and uræmia.

1. *Albuminuria*.—The coagulable constituent of the urine in Bright's disease is a mixture of two proteids, *serum-albumin* and *serum-globulin* or "paraglobulin," both of which are naturally present in the liquor sanguinis. They can best be separated by Hammarsten's process of saturating with crystallised sulphate of magnesia; this precipitates the paraglobulin, but leaves the serum-albumin in solution. Estelle stated ('Revue Mensuelle,' 1880) that in some cases of albuminuria in which he investigated the point, the whole of the so-called albumen, and often two thirds, was really paraglobulin. Subsequent investigations by Dr Halliburton of King's College, and by Dr Noel Paton of Edinburgh, show that the proportion of globulin to serum-albumin differs greatly, not only in different cases but in the same cases at different times. Globulin may exceed the albumen, and again may be only present in traces.

Globulin is somewhat more diffusible than serum- or egg-albumin, and is precipitated by carbonic acid gas, as well as by all the reagents which coagulate albumen. Very dilute acetic acid will precipitate globulin, but redissolves it in excess by changing it to syntonin.

The proportion of serum-albumin to globulin in the urine is not of great pathological or clinical importance, but in the slighter and transient forms of albuminuria globulin is usually absent.*

A third proteid, the more diffusible *peptone* (or rather, according to Dr Martin, *deutero-albumose*), is occasionally present in the urine, uncoagulable by heat, but recognised by the biuret test—a rose (instead of the albuminous purple) tint with cupric sulphate and soda. Its presence does not imply disease of the kidneys. It occurs in cases of pneumonia, in many fevers and suppurations, and sometimes in healthy persons during digestion.

Another allied proteid, *albumose* (hemi-albuminose or pro-peptone), has been found in the urine in certain cases, but its presence has but limited diagnostic significance. Albumosuria was recorded originally by Bence Jones (1848), and lately in several cases of osseous sarcoma, myeloma, and osteomalacia (Bradshaw and Warrington, 'Med.-Chir. Trans.,' 1899, vol. lxxxii, p. 251).

Acid albumen appears to be never present in urine, and alkali albumen only after ammoniacal decomposition has taken place.

Tests.—The oldest method of detecting albuminuria is by *heat*. A con-

* See Dr Halliburton's paper ('Path. Trans.,' 1900, vol. li, p. 128) and the discussion which followed, particularly the remarks of Dr Brodie and the table of cases by Dr Bradshaw.

venient way of applying it is to fill two thirds of a test-tube with urine and then to hold it obliquely near the bottom, while the upper part of the liquid is gently warmed over a spirit lamp until it boils. When the urine is acid any albumen that may be present is precipitated at once or, if scanty, forms a cloud which very slowly subsides. The temperature at which this occurs depends, first, upon the amount of albumen (if it is very small, no change is perceived until the boiling-point is reached; if it is large, an opaque coagulum forms at about 75°C . or 150°F .); secondly, on the reaction of the urine (when alkaline, a higher temperature is required, or the albumen may remain dissolved at boiling-point); and thirdly, on the amount of neutral salts, which, if very small, raises the temperature requisite for coagulation.

When urine is turbid with lithates, the first effect of heat is to redissolve them, and to make the fluid transparent; presently the albumen begins to appear and renders it again cloudy. In a long test-tube all three conditions may be seen at the same time: at the bottom, a part which is cold and turbid; above it, one which is warm and clear; still higher, one which is hot and opaque.

In alkaline urine heat may produce no change, although a trace of albumen is present; but adding a drop of acetic acid before beginning to warm it obviates this fallacy.*

The application of heat frequently produces an opacity which looks like that due to albumen, but really consists of a precipitate of phosphate of lime. By adding a drop or two of acetic acid, we can redissolve the phosphates, and thus distinguish this from precipitate albumen. If urine also contains albumen, the opacity will not be removed by acetic acid.

A second valuable means of detecting albumen in urine is Heller's *nitric acid* test. The best method is to pour a little strong acid into the tube, to hold it in a slanting position, and then to let the urine slide gently down until it floats on its surface. If no albumen is present, the contact of the two fluids is undistinguished, or marked only by a reddish layer, from oxidation of a chromogen. If albumen is present it forms a milky zone or ring, at the line where the acid and the urine meet. When the amount is small, the zone may appear only after the interval of two or three minutes. It is made more conspicuous by holding up the test-tube against a dark background, in good reflected light. Employed in this way, nitric acid is a very certain test for albumen; as certain as heat, but not so delicate.†

In urine lithates are sometimes precipitated by nitric acid; but they first appear, not at the line of junction of the two fluids, but a little above it. If there is any doubt, it may be removed by adding a drop of liquor potassæ or by warming the test-tube, when the urates will at once disappear.

* If the amount of acetic acid be at all excessive, it may itself prevent the heat from throwing down any small quantity of albumen that the urine may contain, unless the urine is also rich in salts. The safest way, therefore, for scientific purposes, is, besides adding the acetic acid, to mix with the urine about one sixth of its bulk of a concentrated solution of common salt, or of magnesian or sodic sulphate. On heating the liquid after treating it in this manner, the slightest trace of albumen comes down.

† Sir Wm. Roberts showed that the readiness with which albumen is precipitated by nitric acid is to some extent affected by the presence of other dissolved matters. The proof is that if two samples of the same albuminous urine be diluted, the one with successive quantities of pure water, the other with the same quantities of healthy urine, the former continues to yield an opaque zone with nitric acid, after the latter has ceased to do so. The same observation was made independently by Dr Grainger Stewart. It depends on the solubility of all proteids being related, not only to the temperature and reaction of the solvent water, but also to the presence of neutral salts.

In concentrated urine also, nitrate of urea may crystallise out, but this takes a longer time, and the glittering crystals which form are quite unlike the white cloud of albumen.

In the urine of patients who are taking copaiba or cubebs, a resinous excretion is made opaque by nitric acid, but not in a well-defined zone. On heating, the opacity diminishes.*

Many other tests for albumen are known besides the two already given. Acetic or citric acid with *potassium-ferrocyanide* is convenient and free from fallacies, but is less delicate than either heat or nitric acid. Dr Pavy has invented pellets of citric acid and the ferrocyanide, which form a convenient and portable test. In the absence of the former the ferrocyanide would act perfectly if a little vinegar were added.

A favourite reagent is *picric acid* in saturated solution. One advantage which it possesses over nitric acid is that it can be carried about without the risk of damage if spilt. On the other hand, picric acid, like heat, fails to precipitate albumen in alkaline urine, and sometimes, as the writer has found, in neutral urine. Moreover, like nitric acid, it may precipitate lithates or lithic acid. It may precipitate peptones (which are redissolved by heat), and it causes a cloud in the urine of persons taking quinine in full doses, which is also cleared by heat. Lastly, it precipitates mucus or nucleo-proteid when neither serum-albumin nor globulin is present.

If acetic or citric acid be added to the solution of picric acid, and the mixture be carefully floated on the surface of the urine, the absence of a cloud may be taken as conclusive of the absence of albumen. If a cloud appears, recourse should be had to heat or nitric acid.

Tincture of galls was used by Owen Rees as a sensitive precipitant of proteids in urine; so is metaphosphoric acid, salicyl-sulphonic acid, and trichloroacetic acid, and also a solution of mercuric iodide in iodide of potassium, but this, like picric acid, is too sensitive, for it precipitates not only peptones and other proteids, but also mucus.†

Dr Oliver, of Harrogate, has brought out a series of paper slips, saturated with picric acid, potassio-mercuric iodide, tungstate of soda, and other solutions which precipitate albumen, and these tests are certainly as portable as could be wished.

On the whole, nitric acid is the most trustworthy of all tests, and if used by the contact method, and with a few minutes' grace, as delicate as is desirable. Heat with acetic acid is still more delicate and, if carefully used, no less certain and more convenient. Acidulated picric acid is quickest and most handy, but it is not more delicate than heat; it is less easy to get a good contact action than with nitric acid, and, while it never fails to detect albumen, it may show a cloud when it is not present. Its chief value is as a rapid and convenient routine test when seeing out-patients or candidates for insurance, or patients with no sign of Bright's disease. A negative result saves further trouble; a positive result needs confirmation.

The other proposed tests are either inconvenient, untrustworthy, or *too* delicate, *i. e.* they show such mere traces of proteid as may be the result of a little globulin or nuclein from epithelial cells, pus, or mucus-corpuseles.

* I remember a man under treatment for gonorrhœa, who came out with the copaiba rash, and was supposed to have scarlet fever with secondary nephritis.—C. H. F.

† With respect to the relative merits of tests for albumen in the urine, including his own, of acidulated brine, see Sir William Roberts's valuable criticism in the 'Discussion on Albuminuria,' at Glasgow, in 1884, p. 16. Also the report of a committee published in the 19th volume of the Clinical Society's 'Transactions' (p. 339).

Traces of albumen may be present which are not of renal origin, and therefore not symptoms of Bright's disease. Such are derived from leucorrhœa in women, from gonorrhœa or seminal emissions in men, and from cystitis in both sexes. The presence of blood or pus always involves that of albumen and globulin, but not of more than is contained in liquor sanguinis or liquor puris.

In women the presence of albumen is so often due to a uterine or vaginal origin, that one must make careful and repeated examinations of specimens free from traces of blood, of pus, or of mucus, before one can be sure of the presence of renal albuminuria. When much mucus is present, the only plan is to filter the urine before applying the tests for albumen.

We must remember that we have a vast accumulation of facts as to albumen which refer to "urine coagulable with heat and nitric acid," and that albumen means a different thing if it is defined by reaction to newer and less certain tests.

Quantitative estimate.—To determine with accuracy the amount of albumen in a given quantity of urine is a tedious process; it must be precipitated, washed, dried, and weighed. In clinical practice, however, there is no sufficient object to be gained by this trouble.

Sir William Roberts in 1876 proposed a method which is far easier, and which appears to yield sufficiently satisfactory results. It consists in diluting the urine with water until it almost ceases to give a reaction with nitric acid, the point fixed being that at which the opalescent zone at the junction of the two liquids begins to be visible between thirty and forty-five seconds after the addition of the acid to the urine. To calculate the number of grains of albumen per fluid ounce of urine, all that is necessary is to multiply the figure 0.0148 by the number of dilutions with an equal bulk of water that the urine has undergone.

A still easier plan, but one that yields only comparative results, is to take the albumen in it with heat or with nitric acid, to let it stand until the coagulum has sunk to the bottom, forming a layer the depth of which can be expressed as a fraction of that of the urine, a half, or a quarter, or one sixth, as the case may be. Vogel, however, found that the space occupied by the same quantity of albumen might vary widely according as it happened to be thrown down in larger or smaller masses; and it is also influenced by the specific gravity of the urine, the range of error from these causes being, according to Esbach, as much as from 30 to 50 per cent.

A mixture of 10 grammes of picric and 20 of citric acid to the litre was devised by Esbach, which, when added to urine, precipitates any albumen present quickly and completely, and the precipitate falls more perfectly than that of picric acid or heat alone. After twenty-four hours it may be read off in a graduated tube, and the percentage of albumen may thus be calculated. For comparative estimates of the albumen passed in a given case this method is quite sufficient; but it fails when the amount is very small: when it is very large, the urine must be diluted before using the test, and the result corrected accordingly.

The late Sir Geo. Johnson and Sir Grainger Stewart both found that Esbach's method gives results nearly the same as those obtained by precipitating and weighing the dried albumen.

In fact, however, the precise amount of albumen is not very important, and may almost be left to rough estimation.

The actual weight of albumen contained in the most bulky coagulum is

but small. Accurate analysis seldom gives more than 5 per cent., even when the urine becomes solid when boiled.

Theory of albuminuria.—The first question in the inquiry why albuminuria occurs in Bright's disease is why normal urine contains no albumen. What prevents its escaping from the blood with the water, urea, and salts which transude in the glomeruli? Only one answer to this question seems possible, namely, that it is kept back by the epithelial layer which covers the capillary tufts; and, as Cohnheim remarks, it is interesting to notice that the vessels of the choroid plexuses, which also yield a non-albuminous fluid, are the only ones that have a similar investment.*

That when albumen appears in the urine it escapes through the glomeruli is rendered probable by Nussbaum's experiments on frogs ('Arch. f. Phys.,' 1878), in which animals these structures have an arterial supply distinct from that of the renal tubes; he ligatured the glomerular arteries, and found that after this operation egg-albumen, injected into the stomach or into the blood, no longer passed into the urine, as it does when the circulation of the kidneys is undisturbed. Again, Ribbert ('Centralblatt,' 1879), having set up an artificial albuminuria in rabbits with egg-albumen, excised the kidneys, and placed them directly in alcohol, so as to coagulate *in situ* the albumen in their interior; he then found that the spaces within the Malpighian capsules always contained coagulum as well as the tubes.

It is no doubt possible that albumen may transude through the tubal capillaries also; but in that case it would naturally find its way, not into the urine, but into the lymph-spaces between the tubes.

The earliest theory of albuminuria was that of Owen Rees (revived by the late Prof. Semmola, of Naples), according to which it depends upon a chemical change in the albumen of the liquor sanguinis, enabling it to pass through the walls of the glomeruli. Stockvis ingeniously disproved this notion by the direct experiment of injecting albuminous urine from patients with Bright's disease into the veins of animals, when he found that the albumen did not, as a matter of fact, escape with the urine that they passed. He also obtained no experimental corroboration of the idea, formerly common, that hydræmia may be a direct cause of albuminuria. A low specific gravity of the blood probably obtains in most cases of anæmia, but albumen is always absent from the urine.

Nor does there seem more reason to attribute albuminuria to the increased presence of neutral salts in the blood; this would increase osmosis through a membrane, but not transudation through living endothelium. The same criticism applies to diminished alkalinity of the liquor sanguinis, of which, moreover, there is little evidence.

Experiments on animals do not support any of these chemical or physical theories of albuminuria, nor that which ascribes it to increased blood-pressure; whereas they do support its being a result of diminished arterial pressure, or of cutting off the flow of blood from the kidneys for a time, or of increased pressure in the renal veins.

The most probable immediate cause of albuminuria is neither chemical nor hydraulic, but anatomical, the occurrence, namely, of some nutritive

* A theory was once current, and received the high authority of Ludwig, according to which albumen was supposed to be present in the transudation from the glomeruli, but to be taken up again and restored to the blood by the epithelial cells of the convoluted tubes; but this was refuted by the observations of Posner ('Virch. Arch.,' 1880). See Dr Starling's critical account of this theory in the first volume of Schäfer's 'Text-book of Physiology' (p. 658).

change in the epithelium covering the glomeruli, which renders it no longer capable of resisting the passage of albumen.

Whenever a full stream of arterial blood is not kept up through the capillary tufts, their epithelium is liable to be damaged, so that it can no longer fulfil its normal function. The instance which Cohnheim adduces as most obviously supporting such an opinion is that of the albuminuria which follows the suppression of urine during an attack of cholera (vol. i, p. 275). This, he maintains, is precisely analogous to the albuminuria which can be experimentally produced by temporary obstruction of the circulation through the renal artery, and which lasts for hours or even days after the obstruction is removed. Another cause of deficiency of blood-supply to the glomeruli may be an impeded outflow through the veins of the kidneys; for Ribbert found that after arrest of the circulation through the renal artery the cells of the glomerular epithelium become obviously swollen and altered.

According to this view albuminuria has no essential relation to the state of the blood-pressure in the arteries of the kidneys. The doctrine that the escape of albumen is favoured by an increase of (systemic) blood-pressure was the conclusion at which Stockvis arrived, and it was adopted by Bartels. It appears, however, to rest on scanty evidence, experimental or pathological. The occurrence of albuminuria as the result of venous obstruction certainly lends it no support; because the pressure in the glomeruli is probably thus diminished rather than excessive; and Runeberg goes so far as to maintain that albuminuria is always dependent upon *deficiency* of arterial blood-pressure. The quantity of urine passed is increased by a high blood-pressure in disease just as in health, but no fresh constituents are added.

A conclusive pathological argument against albuminuria depending on increased blood-pressure is that it is least in constancy and degree in those cases of advanced Bright's disease in which the blood-pressure is the highest.

*"Physiological" albuminuria.**—If now we pass on to discuss the conditions under which albuminuria occurs in practice, we find, in the first place, that it may appear in persons who are apparently in good health. Leube tested ('Virch. Arch.,' 1878) the urine of 119 soldiers, and found albumen in the urine passed in the morning by five of them, and in that passed at midday after a march by no fewer than nineteen; the urine passed in the evening was never albuminous. Capitan found albumen in the urine of nearly 45 per cent. of French soldiers. For observations on the presence of albumen in English soldiers, see Dr Forest's paper in the 'Brit. Med. Journ.' for March 20th, 1897, and Sir T. G. Stewart's figures given below.

Fürbringer tested the urine of sixty-one children, and detected albumen in seven cases, always in the latter part of the forenoon.

In some instances the presence of albumen in the urine of otherwise healthy persons has been traced to a definite cause. Thus Sir George Johnson recorded ('Clin. Soc. Trans.,' vol. vii) several cases in which it was temporarily produced by cold bathing, and ('Brit. Med. Journ.,' 1879, ii) others in which it followed walking exercise.

In another case which came under the writer's notice, temporary albuminuria followed a day's hard riding; and any unusual and prolonged exertion is liable in some persons to be productive of temporary albuminuria.

The late Dr Moxon described in the 'Guy's Hospital Reports' for 1878, several cases in which albumen was from time to time discoverable in the urine of boys and young men who were pallid, listless, and languid; with

* *Syn.*—Functional, intermittent, or cyclical albuminuria.

all in whom he was able to trace the further progress of the affection, it sooner or later passed off, usually in the course of a few months. Sir William Gull had previously observed the same condition, also in youths, and associated with similar symptoms; and Ernst Wagner noticed similar cases in anæmic and weakly girls. In some of Moxon's cases, "albuminuria of adolescents" (as he terms it) was associated with oxaluria (cf. *infra*, p. 673). Dr Dukes, of Rugby, shortly afterwards stated that he had seen ten cases in schoolboys of thirteen to seventeen, in whom albuminuria had occurred as the result of cold, over-exertion, or fatigue, but subsided when they were kept in bed and on a milk diet ('Brit. Med. Journ.,' November 30th, 1878). Dr Pavy has described the same clinical condition as "cyclic albuminuria," by which is denoted the recurrence of traces of albumen in the urine at more or less regular intervals, which correspond chiefly to the periods of taking food. As the same author long ago proved (1863), the amount of albumen passed in cases of Bright's disease varies according to the amount and nature of the diet. Mr Lucas met with albuminuria among surgical out-patients, associated with flat-foot, in youths whom he believed to be addicted to masturbation ('Brit. Med. Journ.,' May 3rd, 1884).

Dr A. W. Stirling examined the urine of 369 boys between twelve and seventeen years of age, on a training-ship at Grays, on the Thames ('Lancet,' 1887, vol. ii, p. 106). He found more or less albumen in 77, without any other sign of renal disease. It was far from constant, and most often present soon after rising. Beside the exposure to cold on leaving bed, two other causes were probable—one, that assigned by Mr Lucas; and the other, exertion in blowing wind instruments.

Sir Grainger Stewart published a series of careful observations which bear out to a certain extent the assertions of Capitan on French, and Leube on German soldiers, and of Dr Stirling on the boys at Grays. Among 205 soldiers in Edinburgh he found albumen in no less than 47;* among 100 presumably healthy civilians in only 7; among 150 workhouse children he found it in only 5; among 100 old people in 17. He found considerable increase in the frequency of albumen after severe muscular work, and less marked but decided increase after food. He found albuminuria in the case of boys after playing wind instruments, as Dr Stirling did, but not nearly so often. More recently a series of careful observations has been published by Dr Samuel West ('Brit. Med. Journ.,' Feb. 25th, 1899, p. 462) on albuminuria in apparently healthy persons at various ages. The observations were made by Dr Levison.

Although, as Parkes and Pavy long ago showed, albuminuria if present is increased after food, there is no good evidence that eating eggs or anything else not poisonous will cause albuminuria in a previously healthy person.

The view which is taken by some writers of what they call "physiological albuminuria" is that it depends upon a congenital deficiency in the power of the glomerular epithelium to resist the passage of albumen through it. In support of such a theory there were two pairs of brothers among the seven cases of which Moxon gives details; and Leube also mentions having met with the affection in two brothers. But such cases are quite exceptional.

The condition is not a constant but an intermittent or occasional one,

* The numbers were obtained by nitric or by picric acid: the figures quoted in the text are those obtained by the former method, for the reasons given above (p. 608).

and its causes are often not far to seek. Cold to the surface, causing congestion of the kidneys, is the most important. Thus, staying long in a cold bath is a cause of this occasional albuminuria, as it also is of intermittent hæmoglobinuria; and in the case of the schoolboy cited above (p. 597), as in others, the same cause produced on some occasions hæmoglobinuria, on others albuminuria. The same explanation applies to the not infrequent presence of traces of albumen in the urine passed on first getting out of bed. There is no absolute line between physiology and pathology; and if we believe (as there is good reason to believe) that exposure to cold over a large surface of the skin leads to acute congestion of the kidneys with passage of albumen, and even to acute nephritis, it is surely reasonable to suppose a less degree of the same effect in the cases under consideration.

The effect of long-continued and severe muscular exertion, and particularly of prolonged efforts of expiration, as in blowing trumpets, would produce passive renal congestion like that which leads to albuminuria in cardiac disease.

As to albuminuria associated with oxaluria and "nervous debility," we may ask whether it is quite certain that in reported cases of "physiological albuminuria" the albumen was always renal in origin. Notwithstanding care to exclude such cases (which was certainly not taken by all observers), were not many of the cases in French and other armies due to *la goutte militaire*? Have not many old men in workhouses granular kidneys or enlarged prostates? Do not young men often pass urine on rising which contains traces of spermatic secretion? And has not a precipitate of mucin or nucleo-proteid by picric acid sometimes been taken for albumen?

But the most important question is whether in such cases the occurrence of albuminuria indicates any tendency to the development of organic renal disease. Ought a young man in whom this affection is discovered to be regarded as eligible for life insurance at the ordinary rate? Leube and Fürbringer would doubtless have answered this question in the affirmative, and so would Moxon, if it were clearly ascertained that the urine contained albumen only occasionally, and that in the forenoon. Both Fürbringer and Moxon detected a few hyaline casts in more than one instance, but this is certainly an exception; as a rule, the amount of albumen is small, it is intermittent, not constant, and there are no other signs of disease.

It seems clear that the cases above recorded by so many independent observers are far too numerous to be set down as examples of latent Bright's disease. Had they all been of that nature, we may be sure that some of them would have shown their real character while they were still under observation; moreover, latent Bright's disease is of the cirrhotic kind, which is almost unknown in early life. When, however, we have made allowance for errors of observation, and thus reduced the number, are we justified in regarding such albumen as physiological? May not some at least have been examples of incipient Bright's disease, or at least of the want of power to resist external influences which is the preceding condition of all diseased action? Johnson expressed ('Brit. Med. Journ.,' 1879) a decided opinion that temporary albuminuria, even when traceable to over-exertion, or exposure to cold, will, if neglected, sooner or later lead to persistent albuminuria and to fatal disease of the kidneys. Even when albuminuria is the only indication of ill-health, we know that the kidneys may be seriously diseased. Clearly, therefore, an insurance office which ignored the presence of albuminuria in applicants who appeared otherwise well would often lose by accepting them at ordinary rates.

The United States Company in New York inquired into the subsequent state of health of persons whose lives had been declined on account of albuminuria. Among those who made applications in the three years, 1878-80, there were sixty-nine (or from 10 to 12 per cent. in each year) whose urine was found to be albuminous. Before the end of 1880 four of these persons died, and it is stated by Dr Munn that the "general appearance of the majority of the others who had been under observation for more than a year was gradually deteriorating." It is to be noted, however, that few of them were under the age of thirty, and that the albumen was often present in considerable quantity. Consequently, although the results of this investigation amply justify the requirement to have the urine of applicants for insurance tested, they do not throw much light upon the question of occasional albuminuria in young subjects.

It is a significant fact that Mr Eales, of Birmingham, found retinal changes in five out of fourteen cases of supposed temporary albuminuria in persons between the ages of eleven and twenty-eight ('Birmingham Medical Review,' 1880).

On the whole it seems to the writer to be a mistake to recognise physiological albuminuria. Accidental traces of albumen, not of renal origin, undoubtedly occur without one being able in every case to explain them. But if albuminuria is recurrent it is probably not accidental, and is most likely due to some defect in the kidneys which may only indicate proclivity to organic disease, or may be the first step in that course.

In many cases, what seems at first accidental and occasional becomes constant and serious. Even when other symptoms of Bright's disease do not supervene, the writer has noticed that youths postponed for insurance on account of slight albuminuria sometimes develop symptoms of anæmia or of tuberculosis—renal or pulmonary. He has also been struck with the frequency of a history of long past scarlatinal dropsy in cases of Bright's disease in adult life; * probably in some cases of albuminuria without other symptoms, there may be some unrepaired injury to the renal epithelium from previous scarlatina, which, under unfavourable circumstances, may develop into nephritis.

Albuminuria without Bright's disease.—Albumen occurs in the urine without there being any marked or permanent lesions of the kidneys, under various morbid conditions, which may be briefly enumerated under the following heads:

i. *Mechanical causes.*—(1) Venous congestion; usually from obstruction of the general venous circulation as the result of valvular disease of the heart, or occasionally from dilatation of the right ventricle following emphysema or like affections of the lungs.

Albuminuria from pressure on the inferior cava and renal veins, by ascites or an abdominal tumour or a pregnant uterus, is not uncommon.

(2) Obstruction in the renal arteries, usually by an embolus detached from the heart, and causing escape of albumen or of blood into the urine, by the same mechanism as explains cerebral hæmorrhage from a similar cause.

(3) Arterial anæmia, leading to deficient blood-supply to the kidneys

* Johnson mentions the case of a medical man, actively engaged in a large practice until shortly before his death from uræmia at forty-five years of age, whose urine had been albuminous from the time when he was a student, and probably earlier still, for he had scarlatinal dropsy when fifteen years old.

through the renal arteries, as in cholera. A like explanation probably applies to albuminuria occurring with abdominal pain and collapse. (Fischl, 'Deutsches Arch.,' 1881.)

(4) Albuminuria appearing after obstruction of the ureters has been removed. This cause has been established by experiments on animals, and a case in point is given by Bartels in which the obstruction was produced by a calculus. If, as seems likely, this is caused by the distended renal tubes pressing upon the veins, this cause of albumen in the urine may be included under the head of venous obstruction.

ii. *Fevers*.—Pyrexial albuminuria occurs most frequently in acute pneumonia, erysipelas, and diphtheria, but also in typhus, enterica, cerebro-spinal meningitis, and pyæmia. In scarlet fever, also, albuminuria may occur during the pyrexial stage, and ought probably to be distinguished from that which appears later and is dependent upon nephritis. Cloudy swelling of the renal epithelium is constantly found in the bodies of those who have died of febrile maladies, but it is doubtful whether this causes the albuminuria, which is far less frequent. If we ask the exact pathogenesis of febrile albuminuria, we are unable to say whether the supposed physical change in the epithelium of the glomeruli is due directly to the heat of the blood, or to the action of the heat on the renal nerves, or to the chemical changes in the blood, or to the disturbance of the circulation through the kidneys.

iii. *Poisoning* by substances which act as direct renal irritants, *e. g.* cantharides and turpentine. Phosphorus probably acts in a different way.

iv. In certain affections of the nervous centres, as cerebral hæmorrhages, epilepsy, tetanus, delirium tremens, albuminuria may occur; but it is doubtful whether it is caused by the nervous disorder. Cerebral hæmorrhage is notoriously frequent in cases of cirrhosis of the kidney, and drunkards are liable to Bright's disease. The albuminuria which follows a fit should always awake suspicion of uræmic eclampsia, and in true epilepsy it is probably the result of renal cyanosis. The latter explanation may also apply to the occasional albuminuria of tetanus. But the relation of albuminuria to venous obstruction is capable of various interpretations; it may depend on a deficient supply of arterial blood to the glomeruli, interfering with the nutrition of their epithelium; or distension of the veins at the junction of the renal cortex with the medulla may press upon the glomeruli in the Malpighian capsules.

It may be stated generally of so-called "functional" and of "symptomatic" albuminuria, that in none of its varieties does it approach in amount, either absolutely or in proportion, to the urine which is usual in Bright's disease.*

2. *Tube-casts or "cylinders."*—These microscopic objects were discovered in the urine by Henle, in 1844; but the merit of distinguishing and figuring their varieties, and of applying their discovery by the microscope to the diagnosis of renal disease, is undoubtedly due to the late Sir George Johnson, of King's College, London (1852).

* We must remember that the urine as we obtain it is, after all, a mixture of the fluids poured out by an almost infinite number of glomeruli and renal tubes, which may yield secretions of very different quality. So that when renal emboli, or localised new growths, are surrounded by zones of hyperæmic and inflamed kidney tissue, any albumen that may be contained in the secretion from these parts is necessarily distributed over the very much larger quantity of normal urine poured out from the rest of the cortex.

There are several different kinds of casts, which may be enumerated as follows:

(1) *Hyaline or fibrinous casts*.—These are delicate, transparent, and colourless, with defined outlines, but so little refractile that they are not always recognised under the microscope in the fluid in which they float, unless stained by carmine or iodine or aniline dyes. They vary greatly in breadth, from 0.01 to 0.05 mm.; their length may be only a few times greater than their breadth, or may reach 1 mm.; they are the longest of any kind. They may be either straight or curved.

Occasionally minute crystals of calcic oxalate or uric acid may be seen in a tube-cast.

(2) *Blood-casts, i. e. fibrinous cylinders* filled with red blood-discs. Their presence shows that the exudation is hæmorrhagic and probably acute.

(3) *Corpuscular casts*, containing small round nucleated cells, which may be pretty certainly identified as leucocytes or “exudation corpuscles,” though often confounded with epithelial cells. Like the hyaline casts they are signs of nephritis.

(4) *Epithelial casts*, containing glandular cells from the convoluted tubules, more or less altered and granular, but by their larger size and more angular shape distinct from white blood-corpuscles. They point to desquamation as the result of nephritis.

(5) *Oil-casts*, or fatty casts, containing highly refracting oil-drops. These prove that the nephritic process has become chronic.

(6) *Granular casts*.—These are the most common and the least distinctive, for the granules may result from the disintegration of blood-discs (when they have a yellow tint), or of epithelium or leucocytes, and, in fact, are often mingled with these elements in the same cylinders. Or they may be fat-granules, shown as bright dark points. Or they may be only lithates accidentally deposited after the urine is passed.

In acute Bright's disease the casts may, for weeks together, contain red blood-discs, to the exclusion of all other elements. In other instances tube-casts look as if they consisted almost entirely of epithelial cells, packed so closely together that little or none of the hyaline sheath can be seen. The fat-granules or fat-drops are probably always derived from disintegrating epithelium; casts in which they are abundant are opaque and conspicuous, almost black by transmitted light. Wagner described granular casts as sometimes “opaque like ground glass,” “appearing as if eroded or breaking down at their edges,” and “sometimes presenting numerous indentations, or looking as though they were made up of a number of square pieces fused together.” These, no doubt, are the remains of cubical or columnar epithelium.

(7) Lastly, *lardaceous or waxy casts*, which show the characteristic reaction with iodine, occur, though very rarely; they are highly refractile, and show more resistance to reagents than the common hyaline casts.

After death casts may be seen in every part of the kidneys, from the convoluted tubes near the glomeruli down to the wide collecting tubes in the pyramids; but they are most abundant in the looped tubes. It has been doubted whether it is possible for casts from the convoluted tubes to traverse the looped tubes so as to be discharged with the urine; but they are so elastic and flexible that this does not seem impossible, for at least the smaller casts, and probably some of the larger casts, are formed in the convoluted segment just before the collecting tube. Possibly, exudation

that originally solidified in the highest tubes close to the glomeruli may afterwards be, so to speak, recast, so as to take the form of tubes lower down.

The chemical nature of the hyaline material which appears to be the basis of all recently formed tube-casts was studied by Rovida, who concluded that it is not identical with either fibrin or albumen, and can only be described as a proteid.

There have been various opinions as to the mode of origin of casts. Bartels supposed them to be produced by a process of secretion from the epithelial cells of the tubes, on the ground that plasma may often be seen protruding from the cells into the lumen of a tube; but Wagner observed the same appearance in healthy kidneys. According to Weigert they arise by the fusion together of altered epithelial cells ('Volkmann's Sammlung,' 162-3) in animals when nephritis is set up by injection of chromate of potass under the skin; and in Bright's disease such an origin seems probable in the case of certain casts which have indented margins, or look as if they were made up of angular pieces. It looks, too, as if casts which turn reddish brown with iodine had been formed out of lardaceous epithelial cells. But for the ordinary hyaline casts the most probable view is that they result from the coagulation of fibrinogen exuded from the glomeruli, just as in any other case of plastic inflammation. The shortness of the interval between the onset of acute nephritis and the appearance of casts in the urine is in favour of the view that they arise by coagulation of exuded plasma.*

Casts sometimes appear in the urine several hours before albumen is discoverable. In the urine of jaundiced patients casts of a greenish-yellow colour are often found, and Dr Finlayson says that in such cases, as a rule, no albumen is present. Sir William Roberts cites cases of venous obstruction from heart disease or emphysema in which tube-casts were found in the urine without albuminuria. In acute Bright's disease casts sometimes continue to be passed after albuminuria has ceased, but they may have been retained in the renal cortex after their formation. As a rule, the abundance of casts in a case of Bright's disease is proportionate to the amount of albumen in the urine; but to this there are exceptions, and in the same patient the number of casts may vary from day to day.

The presence of tube-casts in the urine is of great clinical importance as evidence that the kidneys are diseased. In cases in which the urine contains pus or blood, which may have been derived from the renal pelvis or the lower urinary passages, the discovery of casts is good evidence that the renal cortex is affected; although, if none can be found, it proves little. If albumen only is present, the presence of a few hyaline casts is not absolute proof of the existence of Bright's disease, rather than of those slight or temporary changes in the glomeruli which occur in pyrexia, or from venous congestion, for the writer has found them during life, when after death the kidneys were perfectly healthy to the naked eye and to the microscope. But when their number is considerable and their contents varied, the diagnosis of renal disease is practically certain.

3. *Dropsy*.—In Bright's disease we meet with two kinds of dropsy. The one has been always justly associated with Bright's disease as its

* Bartels states that in a patient who underwent the operation of transfusion with lamb's blood, and whose urine up to that time was normal, urine passed two hours afterwards contained not only albumen, but also hyaline casts. In another case, that of a man who fell from a height upon his sacrum, urine voided five hours later showed hyaline as well as blood-casts.

characteristic symptom. It often begins in the face, about the eyelids, even before it affects the ankles. Its distribution is not independent of the influence of gravitation; for one may often notice that whereas the face is œdematous when the patient rises in the morning, this subsides towards the latter part of the day, and the ankles are swollen when he goes to bed. But it is not limited to the dependent regions of the body, as cardiac dropsy often is, and it is not accompanied by dyspnœa or lividity. Its favourite seats are the eyelids and conjunctiva, the penis and scrotum (or the labia in women), and the loins, when it forms what Bright called "the renal cushion." We may explain the selection of the two former localities as due to the fact that in the eyelids and genital organs there is no subcutaneous fat.

Often the face, trunk, and limbs swell at the same time, and acquire a peculiar white waxy appearance, which is very characteristic. The occurrence of such general dropsy is frequently the earliest symptom of acute Bright's disease, and the first to draw the patient's attention. Generally the urine is found to be already albuminous; and after scarlet fever, when the supervention of dropsy can be anticipated as likely to happen, albuminuria may be present for several days before œdema can be detected. On the other hand, it sometimes happens that the dropsy precedes the albuminuria by a day or two.

The exudation of Bright's disease is, like all dropsical fluids, of low specific gravity, with but little albumen. In many cases a notable amount of urea is present; and Edelssen once found as much as 1 per cent. in a case of hydropericardium.*

Theory of renal dropsy.—One proposed explanation of the characteristic anasarca of Bright's disease is that it depends on hydræmia, the result of diminished action of the kidneys. Bostock and Rees, who made analyses of the blood for Bright himself, Christison, and many later observers have found that the density of the serum is greatly reduced, being not more than 1020, or even 1013, instead of the normal density of 1030.

Some writers refer this physical change in the blood to the loss of albumen through the kidneys, and the resulting subalbuminous state of the blood. But the amount of albumen which is excreted by the kidneys is after all inconsiderable. In most cases the percentage of albumen in the urine does not exceed 2 per cent.; in exceptional instances it may reach 4 or 5 per cent., but then the quantity of urine passed in the twenty-four hours is always much diminished, so that the total daily loss of albumen cannot be calculated at more than from eight to ten or twelve grammes (two or three drachms). Such an amount of albumen can be very easily replaced by food. Moreover quite as large quantities of albumen are lost, without any dropsy resulting, by patients with large granulating wounds, and by those who have chyluria; and far larger quantities by women during lactation.

* C. Schmidt found in one case that the dropsical fluid from the subcutaneous tissue contained 0·36 per cent. of albumen, that from the meninges 0·6—0·8 per cent., that from the peritoneum 1·13 per cent., that from the pleura 2·85 per cent. Bartels examined fluids taken directly after death from different parts of the body of a person who died of advanced dropsy, and found that the specific gravity of the blood-serum being 1015·60, that of the pericardial fluid was 1009·7, that of the peritoneal fluid 1009·6, and that of the anasarca fluid 1007·65; in each of the dropsical fluids the main part of the solid constituents was made up of inorganic salts. In the fifth volume of 'Allbutt's System' Dr Dickenson gives a valuable tabular statement of the density of dropsical effusions in different organs and under different conditions (p. 668).

Is renal dropsy and its hydræmia due to deficient excretion of water by the kidneys? Rehder is cited by Bartels as having made a very elaborate series of observations, in several cases of Bright's disease, on the relation between the amount of water drunk—omitting, however, that contained in the solid food—and that discharged in the urine from day to day; in one case particularly he found that while the dropsy was on the increase, the water excreted was not more than from 29 to 49 per cent. of that which was ingested; whereas while the dropsy was decreasing the ratio was from 72·5 to 100·5 per cent. But, as Cohnheim remarked, such observations, even if accurate, warrant no conclusion as to the nature of the connection between scantiness of the urine and dropsy. One has just as much right to suppose that the variations in the dropsy caused those in the activity of the kidneys as to take the converse view. The effect on the blood of a deficient excretion of water by the kidneys (supposing it not to be corrected either by diminished ingestion of water, or by an increased loss of water through some other channel) must obviously be to increase the whole bulk of the circulating fluid, while diminishing the percentage of solids in it. Cohnheim expressed this by saying that the resulting state of the blood must be, not mere "hydræmia," but "hydræmic plethora." Moreover, he and Lichtheim ('Virchow's Arch.,' lxi) made a series of experiments upon dogs, in which they found that the injection of enormous quantities of a half per cent. solution of salt into the blood produced not the slightest anasarca, even when the renal arteries were ligatured, so as to cut off the escape of the fluids through the kidneys. So far, therefore, as experiment can settle the question, it appears that an "hydræmic plethora" is incapable of causing dropsy.

But, in fact, there is no evidence that this plethora of the blood occurs in Bright's disease. It is not yet ascertained what amount of water escapes from the lungs and from the skin in these cases, though it must be admitted that the dry harsh state of the surface, and the difficulty with which visible sweating can be induced, render it unlikely that the sweat-glands do much to supplement the renal function. But in one way or other the inactivity of the kidneys is compensated, and the volume of the blood remains nearly unaltered.

Again, there is abundant clinical proof that even complete arrest of the secretion of urine causes no dropsy. Not to mention the anuria of hysterical women, there are the cases of "obstructive suppression" resulting from plugging of the ureter of a single kidney, the other kidney having been previously destroyed by disease (*infra*, p. 685). In animals, again, ligature of the ureters is equally incapable of producing dropsy. Lastly, in many cases of scarlet fever, dropsy sets in before there is evidence of impairment of the renal functions, and long before there has been time for any great change in the density or in the volume of the blood as the result of such impairment.

Such considerations show that some further explanation is needed of the characteristic general dropsy of Bright's disease; and this was sought by Cohnheim in a change which he supposed to take place in the walls of the capillaries, by which they are rendered more permeable to fluids. He supposed that the vessels of the skin and of the subcutaneous tissue become altered by the same cause which sets up the renal affection; and pointed out that whereas dropsy accompanies the nephritis that follows scarlet fever

or exposure to cold, it does not follow a like nephritis in the course of diphtheria, when the skin remains intact.

This explanation only applies so far as anasarca is concerned; and Cohnheim accordingly maintained that dropsy of the serous cavities does not occur in most cases of Bright's disease—at least in an early stage, before failure in the heart's action comes in. But Dr Fagge's experience accorded with that of other pathologists, that in autopsies upon some most acute cases—as, for instance, after scarlet fever—one generally finds some fluid effused into the deeper parts of the body, though not, perhaps, in large quantity. That in more chronic cases of tubal nephritis large effusions in one or both pleuræ or in the peritoneal cavity are very frequent is notorious, and that when the heart is quite unaffected.

Cohnheim's hypothesis of a change in the cutaneous vessels, as the immediate and fundamental cause of renal dropsy, cannot, therefore, be admitted; but the same objections do not apply to another theory he put forth, viz. that the characteristic dropsy of *M. Brightii* is not passive effusion, but acute exudation.

In fact, the inflammatory hypothesis seems to the present writer to be the most probable. If so, the characteristic anasarca of acute Bright's disease, like the pleuritic and ascitic effusion, is really—as it was supposed to be before Bright began his researches—an inflammatory exudation. This may be diluted and increased by watery effusion, proceeding not directly from hydræmia, but from diminished arterial and increased venous pressure. The evidence in favour of this theory is the inadequacy of all hydraulic or osmotic explanations, the rapidity of the effusion, the frequent presence of fibrin with the serous effusion in the pleura and pericardium, and the close connection between acute renal dropsy and inflammatory œdema of the skin, larynx, and lungs. It is impossible to draw the line in every case between urticaria and erythema, between hydrothorax in Bright's disease and pleurisy with effusion, between passive ascites and chronic peritonitis. The chemical difference between the effusion of anasarca and that of ascites or hydrothorax probably depends on the locality and not on the cause, for we find similar differences between the most purely mechanical effusions, those due to cardiac disease, as they affect the subcutaneous tissue or the serous membranes. The great pleuro-peritoneal cavity or cœlom, though divided into pleura, pericardium, and peritoneum, is only a huge lymph-sac lined with endothelium, provided with stomata, and strictly homologous with the subcutaneous lymph-sacs of the *Batrachia*; while the areolæ of the subcutaneous connective tissue are only small lymph-sacs, homologous with the larger ones of the frog. Lastly, the existence of cases of acute anasarca or general dropsy, to be mentioned immediately, exactly like that of Bright's disease in every particular except albuminuria, appears to lend further support to the inflammatory theory of acute renal dropsy, since they are certainly not dependent on changes in the renal epithelium.

The other kind of dropsy, which comes on much later and is most marked in cases of chronic interstitial nephritis, has, no doubt, an entirely different pathology. As Dr Fagge taught, "it is identical in its characters with that seen in heart disease, and depends upon obstruction of the systemic veins. When it appears in the course of Bright's disease, it is only an indirect effect of the primary malady, its immediate cause being failure of the heart to maintain the needful activity of the circulation. It is always more marked in the independent parts of the body than elsewhere, espe-

cially in the lower limbs; and it is associated with dyspnoea, with orthopnoea, and often with lividity. It occurs only in the most chronic forms of Bright's disease, usually when the kidneys are contracted, red and granular."

Dropsy without albuminuria.—Cases are now and then met with in which there is dropsy of precisely the same character as that which is so constantly associated with tubal nephritis, but in which no albumen can at any time be found in the urine. Such cases are sometimes called "*essential dropsy*," because there is no clinical evidence the kidneys are diseased. A patient may come under observation with general dropsy that has lasted several days, or even weeks, yet the urine may yield no albumen, either then or afterwards, while the dropsy more or less rapidly subsides.

The writer has repeatedly seen this acute anasarca without albuminuria.

The first case was that of a child five years old, who came into hospital in August, 1879, with the aspect of acute renal anasarca and a history of scarlatina; but the urine, instead of being loaded with albumen, was perfectly free from it, and remained so until the dropsy had disappeared and the patient was sent home well.

The next case, in a woman about thirty, in 1887, was one of anasarca with the characteristic distribution of renal dropsy but with no albumen in the urine. There was marked anæmia, and a pulmonary murmur, which disappeared under treatment. There was no sign or probability of organic disease of the heart, and she went out cured.

A third case in a boy of eleven was very similar, except that there was no murmur, and it also ended in complete recovery.

A fourth was that of a young married lady with decided anasarca, apparently renal, but with the urine exalbuminous. She was not pregnant nor anæmic, and recovered perfectly.

The fifth case was in a previously healthy young man of twenty, a sailor, who was admitted to the hospital in January, 1895, with swelling of the face, hands, and legs, moderate anæmia, no evidence of cardiac disease, and exalbuminous urine. He was of intemperate habits, and developed mania, which obliged his removal to an asylum.

All these cases were under the writer's care for more than a fortnight and less than six weeks. Other cases of dropsy without albuminuria or evidence of cardiac or pulmonary diseases were noted as "anasarca without albuminuria;" but in one, that of a child, the progress of the disease was much longer, a diagnosis of adherent pericardium became possible and was verified after death. In two other cases, both women, the pulse and the distribution of the dropsy were rather cardiac than renal, and allowed of an alternative diagnosis of cardiac dilatation. They both recovered, and the cases, being doubtful, are omitted from the above list. In another case the patient, a woman of thirty-five, though free from disease of the chest and also albuminuria, was so stout and ruddy that she had not the aspect of Bright's disease, nor was the distribution of the dropsy so characteristic as in the other cases. Two other cases in private patients of dropsy without albuminuria were probably due to non-valvular affection of the heart. Lastly, a case in a man of forty-four, though recorded as "dropsy resembling that of Bright's disease, but without any trace of albumen appearing in the urine," and like the others ending in recovery, is omitted because the notes are too brief to exclude other interpretations.

The sixth case under the writer's care occurred in May, 1891.

The patient was a well-built athletic young man, a policeman, aged twenty-three, and lately married. There was a history of a venereal sore, but no secondary symptoms were present. He was admitted to Philip Ward with anasarca following the distribution of that seen in Bright's disease, and albuminuria was expected. No albumen, however, was found during his stay in hospital. The heart was repeatedly examined, and no sign of disease was discovered. There was considerable pallor; the pulse was good without evidence of high tension, and the lungs were perfectly healthy. He was a temperate man, and had

never suffered like this before. Under treatment with diuretics, laxatives, and afterwards with steel, the dropsy disappeared, and he went home apparently in good health in a month.*

4. *Serous and pulmonary inflammations*.—Among the most severe effects of Bright's disease, and frequently the direct cause of death, must be mentioned inflammation of one or more of the serous cavities, or of the lungs. Of the different serous membranes, the pleura is most apt to be attacked, the pericardium next, and the peritoneum only shortly before death or when paracentesis has been performed. Meningitis is rare, and perhaps, when it seems to be of renal origin, some other cause might be found if carefully looked for. A case, however, occurred under the writer's care (November, 1890) in which a young man died from acute nephritis and purulent meningitis with entire absence of traumatic or other local cause, and of syphilis, tubercle or pneumonia. As a rule, the pleurisy or pericarditis of Bright's disease is not purulent.†

Edema of the lungs is frequently the immediate cause of death in Bright's disease, particularly in the more acute form, and may probably be regarded, like the anasarca and pleurisy or pericarditis, as an inflammatory exudation.

5. *Colitis* is one of the less frequent complications of Bright's disease in each of its main varieties (*cf. supra*, p. 384). It is occasionally met with in the acute cases, but more often in the most chronic; and it occurs both in the mucous and the ulcerative form. In the latter it leads to hæmorrhage, discharge of pus, and sometimes to septicæmia, without bacterial infection (as in a case published by Sir T. G. Stewart in the 'Practitioner' for 1898).

Attention was drawn to this complication by Dr Dickinson many years ago ('Med.-Chir. Trans.,' vol. lxxvii, p. 111; 'Path. Trans.,' 1878, p. 117), and we have several striking examples in the Guy's Museum (Catalogue Nos. 808—811). Dr Hale White gave the clinical history of some of the more recent of these in the 'Guy's Hospital Reports' for 1888 (p. 131).

Cases of lardaceous disease of the kidneys are, as a rule, accompanied sooner or later by uncontrollable diarrhœa, which is due to muco-enteritis with degeneration of the small mesenteric arteries, and this complication is the most frequent immediate cause of death. But it often determines the fatal event in any form of Bright's disease. Besides its prognostic significance this complication should make us cautious in pushing purgative treatment, particularly in long-standing cases.

6. *Dermatitis*.—We may associate with these secondary serous inflammations inflammation of the skin occurring in Bright's disease. Dermatitis is readily excited by irritation more than in other conditions; hence the caution necessary in using acupuncture, and still more a permanent trocar, to draw off the serum; hence paracentesis is a more serious operation in the case of renal than of cardiac or hepatic ascites; or severe erysipelas and sloughs may ensue after pricking the legs.

* On this point see some cases in children recorded by Sir Dyce Duckworth in the 'St. Barth. Hosp. Rep.' for 1883, vol. xix, p. 321, and by Sir George Johnson in his 'Lectures' (1887); also by Dr. Goodhart in the 'Guy's Hospital Reports' for 1884 (vol. xlii, p. 197). See also Dr Herringham's case of acute anasarca in a child without albuminuria, in which the kidneys were found after death in a state of acute nephritis ('Clin. Trans.,' 1901, p. 35).

† Dr Fagge did not remember to have ever seen a purulent exudation in the pericardium, even in the meshes of lymph. On the other hand, the late Dr Sutton, a pathologist of no less experience, regarded purulent pericarditis as common, and almost characteristic of Bright's disease. Empyema is decidedly rare, but peritonitis in renal cases is undoubtedly very often purulent.

The most frequent and until lately the only recognised affection of the skin in Bright's disease is Erythema leve of Willan and Bateman—a roseolous, superficial inflammation with smooth, shining surface, sometimes assuming the character of superficial erysipelas, and even leading to gangrene. It is most often seen on the lower extremities, and always where there is actual œdema.

There are, however, several forms of dermatitis which appear in the course of Bright's disease, but are not the result of wounds or irritation, and are not confined to a swollen surface. Nor are they mere sudaminous eruptions provoked by hot-air baths or other methods of producing diaphoresis.

A diffused roseolous rash chiefly affecting the trunk and proximal part of the limbs, and lasting only a few days, is occasionally seen, most often in cases of tubal nephritis, and occasionally in the acute stage of the disease.

More common, and perhaps most characteristic, is a papular "lichenous" eruption, with rather large, discrete, red pimples on a rough surface, mostly confined to the extensor surface of the limbs, but sometimes seen on the back and trunk generally. It is comparatively chronic, and is sometimes troublesome from the itching it causes.

A moist dermatitis closely resembling, or perhaps identical with, eczema, is now and then found on the limbs, the genitals, or the neck and ears.

Lastly, several cases have been noted in which a diffuse, squamous eruption, beginning on the limbs, may spread until it becomes almost universal so as to simulate pityriasis rubra. This form has always been associated with chronic renal cirrhosis with little dropsy.

These eruptions are usually brief in their duration, and seldom cause pain or irritation. They are generally seen in chronic tubal or chronic interstitial nephritis, and do not appear to be connected with uræmic symptoms except by coincidence. Pathologically they seem to take their place with retinitis and inflammation of the serous membranes. They have no bad prognostic signification except the last-mentioned universal desquamation, but since they usually appear in the course of chronic and confirmed cases of Bright's disease, they occasionally precede a fatal result; but they often pass away before this arrives, and have been seen in cases that have recovered.

Some remarkable cases of dermatitis occurring in the practice of Dr Cavafy and other physicians at St George's were collected and published by Dr Lancaster, now of Swansea, in the 'Clinical Transactions' for 1892 (vol. xxv, p. 49). A short account of them was given by the present writer in the third edition of this book (1891), and a fuller one in his 'Introduction to the Study of Diseases of the Skin' (1893, p. 163), and again in the 'Journal of Dermatology' for 1895 (vol. vii, p. 284). In the eighth volume of the same journal (p. 9), Dr Barry, of Leeds, describes a case of bullous dermatitis in a fatal case of chronic tubal nephritis with uræmic symptoms.

7. *Albuminuric retinitis*.—One of the most characteristic indications of Bright's disease is the presence of changes in the retinae. They are said to have been noticed *post mortem* by Türck in 1850; but their importance in Bright's disease appears to have been first recognised by Heymann in 1856. They occur only in chronic cases; in advanced stages of tubal nephritis after scarlet fever or during pregnancy, or when the kidneys are cirrhotic; very seldom in cases of lardaceous disease, and probably only

when it has long been associated with atrophic nephritis. Nevertheless, the recognition of these changes in the retina by the ophthalmoscope is occasionally the first symptom that leads to discovery of albuminuria. Their frequency is believed by Sir William Gowers to agree with the statement of Mr Eales, who found them in 28 out of 100 cases of chronic Bright's disease, or in about 2 of every 7 cases ('Birm. Med. Rev.,' 1880).

Dr C. R. A. Sutton, among 312 cases of acute and chronic Bright's disease, found retinal changes in 33 ('Guy's Hosp. Reports' for 1895, vol. li, p. 152).

The most common of these lesions is more often seen in a degenerative than an inflammatory stage. It is the appearance of *whitish spots*, sometimes close to the optic disc and sometimes elsewhere; near the macula lutea they often appear as fan-like streaks. They may be so minute as to be only visible by the direct method of examination; or may form irregular patches, which equal the disc in size. A less marked diffuse opacity may spread over more or less of the retina.

Along with these white spots, or independently of them, *hæmorrhages* are frequently observed. They lie, for the most part, in the nerve-fibre layer of the retina; and they therefore are often "flame-shaped" (to use Gowers' expression), following the radiating course of the fibres. They may also run by the side of and parallel to the vessels. When large they are irregular in shape, and may spread into the deeper layers of the retina.

Distinct from these retinal hæmorrhages, the ophthalmoscope sometimes shows larger and irregular patches of hæmorrhage in the choroid, following the vascular changes which we shall see are characteristic of renal cirrhosis. They are seldom seen in early stages, except in very severe scarlatinal nephritis.

Occasionally *optic neuritis* is the most conspicuous change seen by the ophthalmoscope. The appearance of the disc is identical with that produced by a cerebral tumour (vol. i, p. 772), and it may go on to atrophy in exactly the same manner.

Lastly, there may be a general *œdema* of the retina, with complete obscuration of the disc, the arteries narrow and partially concealed; the veins distended and tortuous, with hæmorrhages, forming large streaks in the course of the nerve-fibres, and white spots, large, rounded, and soft-edged. Gowers, from whom the description is taken, says that this form of retinitis is only seen in cases of severe and rapidly fatal Bright's disease.

Anatomically the white spots depend upon a degenerative change in the nerve-fibres, which become greatly thickened, varicose, and filled with fat-globules; compound granule-masses, too, appear in large numbers. The vertical fibres of Müller become swollen and fatty. The fan-like distribution of the spots near the macula lutea depends upon the arrangement of the fibres as they radiate from the *fovea centralis*. The diffuse opacity is due to œdema, which causes, in hardened sections, the retinal elements to appear separated. Even the layer of rods and cones may be thickened where there is a white spot. The albuminuric retinitis always affects both eyes, though not always simultaneously.

The exact *pathology* of these retinal changes is still obscure. Traube believed that they scarcely ever occur except when cardiac hypertrophy is already present; and Cohnheim upheld the same opinion. But later experience has shown that they are often met with in tubal nephritis with no cardiac or vascular changes. Gowers drew attention to the small size of

the retinal arteries, and regarded this as a vital contraction of their coats; but Mr Brailey has since shown that these vessels become affected with an *endarteritis obliterans*, exactly like that of the arterioles of the kidneys and of other parts. Gowers has observed optic neuritis accompany symptoms of cerebral disturbance, such as intense headache, delirium, and convulsions. But these are symptoms of uræmia, and again prove only that optic atrophy belongs to the later stages of Bright's disease.

Albuminuric retinitis may cause no subjective symptoms; or there may be more or less marked amblyopia. Voelcker speaks of cases in which transitory darkening of the field of vision occurs from time to time during excitement or exertion. Even in the most severe forms of albuminuric retinitis central vision is seldom lost.

It must not be supposed that these retinal changes are conclusive of the existence of Bright's disease. Chlorosis, leuchæmia, and idiopathic ("pernicious") anæmia are not infrequently complicated by neuro-retinitis, indistinguishable from that of albuminuria.

As a rule, when albuminuric retinitis has once developed itself, it persists until the patient's death, though the exact appearances vary from time to time. It is in the Bright's disease associated with pregnancy that there is most ground for hoping a permanent recovery from the retinal affection, and in the chronic cirrhotic cases that there is least. The free use of purgatives is believed to favour its subsidence.

The unfavourable significance of albuminuric retinitis is well known, but there are exceptions. In one case of chronic tubal nephritis lately under the writer's care there was well-marked retinitis, yet the patient gradually recovered, and is now, after four or five years, in good health.

8. *Cardiac hypertrophy and arterio-sclerosis*.—In his commentary on the first hundred autopsies on the disease afterwards called by his name, Dr Bright remarked ('Guy's Hosp. Rep.,' vol. i, p. 396), "The deviations from health in the heart are well worthy of observation; they have been so frequent as to show a most important and intimate connection with the disease of which we are treating." Excluding valvular disease, in thirty-four cases there was marked hypertrophy, generally affecting the left ventricle. Since that date (1837) Barlow, Rees, and their successors have always taught that the peculiar renal pulse is "hard," "wiry," "resisting," or "incompressible," or (to use a more modern expression) there is *increase of arterial tension*.*

The hypertrophy of the left ventricle can generally be detected during life by displacement of the apex-beat outwards, by its heaving character, and by alteration in the first sound—often a mere weakening without any change of quality, sometimes a prolongation or reduplication, and occasionally a murmur. That hypertrophy is the result and not the cause of the high arterial pressure seems shown by the fact that frequently the above signs are after a time replaced by those of dilatation: a short slapping impulse, and in many cases a decided murmur—apical in seat, systolic in rhythm, and, except by its being inaudible in the axilla, not to be distinguished from that of primary mitral regurgitation. After death the left ventricle is found hypertrophied or dilated, without any valvular lesion.

* This characteristic feature of most chronic and some acute cases of renal disease was first observed abroad by Traube.

There can be little doubt that dilatation of the systemic ventricle, like the hypertrophy which usually precedes it, is the immediate result of high blood-pressure in the systemic arteries, and comparable to the hypertrophy and dilatation which result on the right side of the heart from obstruction in the pulmonary circulation (*cf.* p. 197).

Bright himself proposed two causes of the ventricular hypertrophy: "That the altered character of the blood [conveyed to the heart in the coronary arteries] affords irregular and unwonted stimulus to the organ immediately; or that it so affects the minute [arterial?] and capillary circulation as to render greater action necessary to force the blood through the distant subdivisions of the vascular system" ('Guy's Hosp. Rep.,' vol. i, p. 396). Each of these explanations has been since expanded into various subordinate theories.

The latter is the more satisfactory. Apart from the anatomical facts stated above, we have no knowledge of alteration in the blood acting as a stimulus to overgrowth; and why should not the same stimulus produce hypertrophy of the diaphragm and other muscles?

But the question remains, what is the nature of the obstruction in the systemic vessels which raises the blood-pressure?

In the first place, hypertrophy of the heart occurs in acute as well as in chronic cases of morbus Brightii.* It may also develop when the kidneys have become atrophied as the result of hydronephrosis, or of some other affection of the renal pelvis, as in cases recorded by Cohnheim.

In association with the lardaceous forms of Bright's disease, however, it is not seen unless the renal cortex is also the seat of advanced tubal nephritis. It is comparatively slight when chronic Bright's disease is complicated by phthisis, cancer, or some other wasting disease, and in those who are very old.

Evidently, therefore, no explanation of the occurrence of cardiac hypertrophy can be valid unless it is applicable to both the principal forms of chronic Bright's disease. The *extent* to which the heart becomes enlarged differs in different cases—partly according to their duration; and it is far greater in renal cirrhosis than in any other form. Thus, whereas in the earlier stages of tubal nephritis its weight may attain fifteen or sixteen ounces, and in the latest granular and atrophic stage of that affection seventeen or possibly twenty-one ounces, there are some instances of (primary) renal cirrhosis in which it reaches twenty-three, twenty-four, twenty-five, or even twenty-eight ounces. Compare the tables of the weight of the heart found by Dr Goodhart in 188 cases of chronic parenchymatous nephritis, in 329 cases of cirrhosis ('Guy's Hosp. Rep.,' xliii, pp. 104, 109, 111), and in 146 cases of lardaceous disease.

The chamber chiefly affected is the left ventricle, the walls of which (and also the papillary columns of the mitral valve) become thick and fleshy, their substance being made up of muscular fibres of perfectly normal appearance. Sometimes the cavity is of normal size, sometimes more or less dilated. In many cases the right ventricle also is somewhat enlarged. This generally indicates that the left ventricle has not been able to maintain the circulation efficiently, or that pulmonary obstruction has arisen from

* This fact was ascertained by Dr Galabin from cases in Guy's Hospital between 1868 and 1872; and Dr Goodhart's further experience of ten years (1873-82) confirms the conclusion ('Guy's Hosp. Rep.,' vol. xliii, p. 104). The left ventricle was hypertrophied in 109 cases of acute nephritis, and normal in only 25.

bronchitis, œdema of lung, or some other cause. But to some extent it is inevitable that the right ventricle should share in the process of enlargement, especially when the left ventricle becomes very greatly increased in size.*

It is important to observe that the state of the left ventricle is one of real, not only apparent, hypertrophy. In some cases fibrous tissue is mixed with the muscular overgrowth (*cf.* p. 210), but in most characteristic examples this is not the case. It may be added that the hypertrophy is not equal to that of aortic (sigmoid) disease, and never produces a "bovine" heart.

With regard to the exact character of the changes that take place in the *arteries* there have been extraordinary discrepancies in the statements of different observers. That in middle-aged or old persons affected with Bright's disease, atheromatous lesions are often found in the aorta, in the cerebral, renal, and other arteries was well known to Bright himself, and also to Wilks, Dickinson, and other writers on the subject. But it was George Johnson who, in the 'Med.-Chir. Trans.' for 1868, stated that the arterioles, not only in the kidneys, but also in the subcutaneous and sub-mucous tissues, in the muscles, and in the pia mater of the brain, become remarkably thickened; and he attributed this change to an hypertrophy of the muscular fibres in their walls. Four years later Sir William Gull and Dr Sutton read before the Royal Medical and Chirurgical Society a paper in which they declared the thickening to be the result of a hyaline fibroid formation, partly outside the muscular layer, in the *tunica adventitia*, partly in the *intima*, the muscular layer itself being often rather atrophied than hypertrophied, and the nuclei of its fibres degenerated. In the course of the discussion which followed, the "hyaline" appearance described by these observers was shown to be due to the action of the acidulated glycerine in which the preparations were placed for examination. Leyden among foreign writers alone lays stress upon this character; but his figures ('Ztschrift. f. klin. Med.,' 1880) represent circumscribed glassy patches lying in the coats of the thickened vessels, unlike the account of Gull and Sutton. Few German pathologists confirm Johnson's statements as to the existence of muscular hypertrophy.

We must, however, bear in mind that this change is most constant in the chronic atrophic form of morbus Brightii, which is undoubtedly less common in Germany, and perhaps on the Continent generally, than in England.

All recent writers agree in considering that an almost invariable condition affecting the intima is that which was originally named by Friedländer *endarteritis obliterans*.

There is, in most cases of renal cirrhosis, both hypertrophy of the media in the smaller arteries, and thickening of the fibrous intima, the adventitia being only slightly affected. The effect of either or both of these conditions is to narrow the passage for the blood towards the systemic capillaries, and thus by increasing peripheral friction (or resistance) to raise the blood-pressure in the arteries, the aorta, and the left ventricle. Thus is

* Traube asserted ('Ges. Abh.,' iii, p. 239) that he had been able clinically to determine the presence of hypertrophy of the heart within four weeks from the commencement of an acute renal affection. Similar cases were recorded by the late Dr Peacock, by Dr Stone in his Croonian Lectures (1879), and by Dr Goodhart ('Path. Trans.,' xxx). A case of recession of an hypertrophied ventricle was recorded by Sir William Roberts ('Glasgow Med. Journ.,' 1884).

explained the hard or incompressible pulse of high tension, which is one of the most characteristic symptoms of Bright's disease.

Since the invention of the sphygmograph this symptom can be estimated much more accurately than before. The first point to be noted in the tracings of a renal pulse is that the pressure applied to the artery while they are being taken is far greater than that which brings out the characters of the pulse most distinctly in health: as registered by the late Dr Mahomed's spiral eccentric it was from four to six ounces, instead of being from one and a half to three ounces. The next points are the breadth or "bluntness" of the tidal wave and the slope of the upstroke; these indicate prolongation of the ventricular systole. The last point is the distance of the dicrotic notch above the base line (*cf.* p. 4).

If next we consider the pulse as it is felt by the finger, we find the following characters. First, it is *persistent*: even in the intervals between the cardiac beats the artery feels full. Next, it is *long*, not falling away as soon as it has reached the finger, but pushing and laboured in character. Lastly, it is *hard* or *incompressible*, requiring much force to empty the artery. Both the last characters are really indications of the state of the left ventricle which is associated with high arterial tension—the slow prolonged systole and the hypertrophy of the ventricular walls.

Examination of the heart often yields valuable corroborative evidence in an enlarged area of percussion-dulness, displacement of the apex outwards, and a heaving, laboured impulse.*

On auscultation the first sound may be faint, or dull and prolonged, or reduplicated, or replaced by a murmur.† A more characteristic auscultatory sign, and one which is the direct result of the increased arterial tension, is the loud, ringing, or metallic quality of the (aortic) second sound, as it is heard at the base of the heart, or over the carotid artery. Occasionally, also, a diastolic shock can be felt by the hand placed over the cardiac region.

The origin of the systemic obstruction which causes the high arterial tension in Bright's disease has been much debated.

Sir George Johnson (1868), relying upon his observations as to the existence of hypertrophy in the muscular walls of the arterioles, propounded the theory that these vessels exert a "stopcock" function, resisting the passage into the capillaries of blood which, as the result of defective elimination by the kidneys, is noxious to the tissues. In fact, he imagined an active antagonism between the heart and the arterioles, as the result of which they each become hypertrophied—a conception quite at variance with probability.

The late Dr Mahomed reverted to the view that the obstruction is in the capillaries, and this view has been supported by Dr Saundby, of Birmingham; but, believing that the high tension in the arteries precedes the development of renal disease, their notion is that the supposed impurity of

* It is, however, important to be aware of the fact that these signs are not seldom absent, even when there is no obvious emphysema or other disease of the left lung to account for it. Thus in cases of renal cirrhosis, when perhaps the patient has been admitted with cerebral hæmorrhage, we have again and again failed to detect any indication of cardiac hypertrophy, although at the autopsy a day or two later the heart has been found enormously enlarged. It is particularly in those cases in which the hypertrophy is unattended with any dilatation that the difficulty arises.—C. H. F.

† Mahomed also maintained ('Guy's Hosp. Rep.,' 1879) that it is sometimes preceded by a short sound resembling the presystolic murmur of mitral stenosis. Surely this is the same sign as what is called reduplication of the first sound?

the blood is due, not to imperfect excretory activity on the part of the kidneys, but rather to over-eating and over-drinking, by which it becomes charged with injurious matters. The objection to all such theories, however, is that there is no proof whatever that any changes in the circulating fluid are capable of retarding its flow through the capillaries.*

Traube, in 1856, argued that destruction of the renal parenchyma would have two results, each of which might tend to augment the pressure in the arteries; one being the accumulation of water in the blood from impairment of secretion in the kidneys, the other the diminution in the amount of blood flowing from the arterial into the venous system as a consequence of obliteration of capillaries in those organs. But the first of these two conditions does not exist in the most marked cases of high pressure, which are accompanied by increased, not impaired, flow of urine; and it is directly contradicted by Dr Bradford's experiments, in which excision of parts of the kidneys caused increase of flow of urine. As to the second, hypertrophy of the heart ought, on this view, to follow on amputation of the thigh or of two limbs more certainly than on the gradual diminution of the moderate area of the renal circulation.

Dr Hamilton, of Aberdeen, has put forward the ingenious theory that the systemic obstruction is due to diminished specific gravity of the blood; this causes the red discs to mingle in the layer of leucocytes, which normally lie close to the vessel wall, and thus friction is increased. Of the physical fact there is no doubt, but why should not the same effect occur in other forms of anæmia?

It is physiologically clear that an hypertrophied heart cannot raise the arterial pressure unless it is supported by a resistance in the systemic arterioles; and experience of the low arterial pressure in cases of incompetence of the sigmoid valve, with far greater ventricular hypertrophy than is ever found in Bright's disease, confirms the conclusion. Surely the fact that the secretion of urine is maintained or even increased shows that considerable changes must have taken place in the systemic arteries generally, as well as in the heart. The means by which the high arterial tension of Bright's disease is kept up may vary. At an early stage of tubal nephritis it can only be by contraction of the muscular walls of the arterioles. Even in the chronic form of Bright's disease this must still play an important part, at least in cases in which a state of low pressure and diastolic tension can be induced by the inhalation of nitrite of amyl, as has been shown by Sir William Broadbent: but the presence of *endarteritis obliterans* no doubt also produces and maintains the permanent peripheral resistance which leads to the high arterial tension of renal cirrhosis.

9. *Hæmorrhages*.—The frequent rupture of arteries in Bright's disease is doubtless partly due to the high arterial tension, and partly to arterial sclerosis. Thus arise retinal and choroidal hæmorrhage, apoplexy, hæmaturia, epistaxis, and hæmatemesis. As regards epistaxis, Mahomed remarked ('Guy's Hosp. Rep.,' 1881) that even when the patient is much blanched by loss of blood, the pressure in the arteries may still remain excessive.

Retinal hæmorrhage is common, and sometimes so extensive as to pro-

* It is true that injection of urea into the blood raises the arterial pressure in animals, yet this is only when the quantity injected is so large as to render the experiment inapplicable to human pathology.

duce hemiopia or amaurosis; but more often it is multiple, and only recognised by help of the ophthalmoscope. Cerebral hæmorrhage is one of the most frequent terminations of cirrhosis of the kidneys, from bad arteries and an hypertrophied left ventricle. Epistaxis in elderly persons should always lead to a careful examination of the urine, the pulse, and the heart: it is often, like retinal hæmorrhage, the precursor of an attack of apoplexy, which may be warded off by purging and low diet (*cf. supra*, vol. i, p. 725). Hæmatemesis is probably the rarest form of hæmorrhage from Bright's disease; but fatal bleeding from a gastric ulcer, whether simple or cancerous, is sometimes due to a diseased artery being opened, and this may be part of general arterio-sclerosis of renal origin. The occasional hæmoptysis of Bright's disease is probably also due to arterio-sclerosis of the bronchial arteries.

10. *Uræmia*.—The importance and danger of cerebral symptoms in Bright's disease was recognised from the first; but there have been wide differences of opinion as to their pathology. These grave symptoms are universally termed "uræmic," although the name uræmia (invented by Piorry) implies that they depend upon an accumulation of urea in the blood, a theory which cannot now be accepted.

a. Acute uræmia.—The symptoms of acute uræmia vary in different cases. The most striking is the occurrence of convulsive fits. These *epileptiform* paroxysms or eclampsia are sometimes seen in patients who are confined to bed with renal dropsy; sometimes in those who are engaged in their daily occupations. The onset may be either sudden, or preceded for a few hours or days by headache, drowsiness, vertigo, or a transient rigidity of the face, or of the lower jaw, or of a limb. Nausea, again, and even vomiting, may be among the prodromal symptoms; or severe dyspnœa.

A description of uræmic eclampsia will be found in the chapter on primary epilepsy (vol. i, p. 913); for the symptoms are identical, even to the biting of the tongue, the foaming at the mouth, the involuntary discharge of urine, and the subsequent sleep, sometimes replaced by an attack of mania. Wagner stated that the pupils are generally dilated, seldom small; but according to the teaching of Addison they are most often contracted. The temperature may rise to 102° or 104°, or even a higher point still—in one case 107°. It slowly falls to normal, or below, during the coma which follows the convulsions; and some days afterwards may remain as low as 94° or 95°. The pulse is commonly accelerated while the spasms continue; afterwards it returns to its natural rate, or may become slower still, remaining perhaps (as in a case of Wagner's) between 44 and 64 for the next fortnight. After the attack the patient is sometimes dull and depressed.

In many cases, before consciousness is regained another uræmic paroxysm sets in; and thus twenty or thirty may occur in succession, as in the *status epilepticus* (vol. i, p. 918). The result is often fatal; but even after a series of fits the convulsions may cease, and the patient regain consciousness. A single paroxysm seldom ends fatally; but in 1862 a woman, aged thirty, died in Guy's Hospital within seven minutes from the commencement of uræmic symptoms. She had just eaten her breakfast, when slight spasms of the arms suddenly set in. She became pale, and her lips and fingers livid; there was foaming at the mouth, and the pupils were dilated. The heart continued to beat regularly till the moment of death.

Sometimes, instead of a uræmic fit having the typical epileptiform character, its symptoms are apoplectic. In the 'Guy's Hospital Reports' for 1839, Addison described this as "a sudden attack of coma with stertor, or, in other words, *apoplexy*." We must be cautious in diagnosing cerebral hæmorrhage in cases of renal disease, notwithstanding the well-known frequency with which it occurs. Although we have not met with fatal cases in which this mistake was made, the following case shows that the caution is not unnecessary (*cf.* vol. i, p. 753).*

A carman was brought into John Ward having been seen to fall unconscious from his seat: there was no history of a previous attack and no one had seen convulsions, so that the case was regarded as one of apoplexy; but the complete absence of hemiplegia, the contracted pupils, and the fact that the urine drawn off, though albuminous, was pale and of low specific gravity, lead the writer to the diagnosis of uræmia, and this proved to be correct by the patient's speedy recovery and his statement of a previous attack of the same kind.

Addison believed that the diagnosis might be made by the character of the stertor; in uræmia, he said, the sound was more hissing, "as if produced by the air striking against the hard palate, or even the lips, rather than against the velum and the throat, as in ordinary apoplectic stertor." He also believed that the respiration was from the first much more hurried than in true apoplexy.

In certain instances uræmia may manifest itself by *delirium*, lasting for days together, or by *rigidity* of one or more of the limbs, or, according to Charcot, by *tremors* like those of paralysis agitans. Bright ('Guy's Hospital Reports' for 1840) related a case in which for two days before death there was distressing and almost incessant twitching of the muscles, which increased until the arms and the legs were forcibly drawn up and the face was distorted by the spasms; yet the faculties of the mind were perfect to the last. In some cases uræmia shows itself by much slighter symptoms; by transitory *trismus* perhaps, or by short attacks of *clonic spasm* in some of the facial muscles, or in those of the eyeballs, or of a limb; the patient retaining consciousness, or being at most a little confused or dull of intelligence. The knee-jerks in uræmic eclampsia are retained, and sometimes increased.

Perhaps the most remarkable of all the effects of uræmia is *amaurosis*. This not infrequently occurs along with eclampsia, and the patient, when he recovers consciousness, finds himself blind; but more often it is the only symptom, except headache. It sets in suddenly, is bilateral, and is almost always complete, the patient having not the slightest perception of light. The pupils generally react to light, but in some cases are insensible and widely dilated. The ophthalmoscope does not show any change in the optic discs or retinæ. This alarming affection rapidly subsides, so that the patient regains sight within twelve or twenty-four hours, or at the longest in the course of a few days.

This kind of amaurosis must be distinguished from that affecting one eye from extensive retinal hæmorrhage.†

* Roberts cites three uræmic apoplectiform cases, but each of them is open to criticism. One patient had had "a few drops" of laudanum given to him for diarrhœa just before the cerebral symptoms set in, so that it may be doubtful whether they were not due to the excessive action which even small doses of that drug are known to exert when the kidneys are diseased; in the second case epileptiform convulsions were present; and in the third case there seems to have been no autopsy, so that the possibility of cerebral hæmorrhage is not excluded.—C. H. F.

† The writer once found a patient with chronic Bright's disease blind from this cause owing to the unaffected eye having not been used for many years.

It is said that in some instances a transitory defect of hearing, or even complete deafness, has been observed as a sequela of a uræmic seizure.

b. Chronic uræmia.—Clinically contrasted with these acute forms of uræmia are those described as chronic. The lungs or the digestive organs may appear to be the parts affected; but there is good reason to believe that in these cases also the starting-point of the symptoms is in the brain.

The *cerebral symptoms* are usually headache, giddiness, or drowsiness, lasting for weeks or months, but often with intermissions. The patient's aspect is dull and expressionless; he lies in bed, indifferent to his own condition, and ultimately may fall into complete stupor. A patient of the writer's died of tubal nephritis of about three months' standing, which was passing from the acute to the chronic stage; and he became increasingly dull and apathetic until at last he lay insensible, but without stertorous breathing, for several days before his death.

Sometimes the symptoms are typhoid, the tongue being dry and brown, and sordes collecting upon the teeth and lips. Such cases might be mistaken for enteric fever.

In other patients the principal indication of chronic uræmia is *dyspnœa*. This is generally paroxysmal, and is apt to come on at night, like asthma. It may be expiratory (again like asthma) or inspiratory, as though there were laryngeal stenosis; or both inspiration and expiration may be free, but unnaturally hurried. The hissing sound of the breath and the great frequency of respiration are very characteristic features of uræmic dyspnœa. There is rarely any cyanosis present. The rhythmical or "phasic" type of breathing described by Cheyne, and afterwards by Stokes, is not infrequently observed in cases of chronic uræmia (*cf.* vol. i, p. 1043).

Another sign of uræmia may be an intense *itching* of the skin; so that patients go on scratching or rubbing themselves, even when they are unconscious. *Muscular cramps* and *hiccough* are also not uncommon.

One of the most characteristic symptoms of uræmia is nausea and *vomiting*. At first this may occur only in the morning when the stomach is empty. Afterwards it may take place whenever any food is taken, and become exceedingly intractable, continuing for weeks, or even for months. The vomited matters, as a rule, are acid. There is no local lesion of the stomach found after death, and the symptom is no doubt cerebral in its seat and toxic in origin.

Diarrhœa is not infrequent, and usually accompanies vomiting. It sometimes seems to be purely functional; but it may also depend upon colitis, or even "diphtheritic" hæmorrhagic sloughing, like that of dysentery. In cases of this kind the evacuations often contain blood, and mucus and pus in large quantity (*cf.* p. 384).

Prognosis.—Epileptiform convulsions and the other symptoms that have been grouped together under the name of acute uræmia may accompany any form of Bright's disease, and are very frequent in the nephritis of scarlet fever and pregnancy; but they are most often seen in the latter stages of renal cirrhosis. The slighter forms of uræmia sometimes pass off; and their occurrence is no certain proof that the renal disease which causes them is so advanced as to be incapable of recovery. But the prolonged stupor, the typhoid symptoms, and the dyspnœa of chronic uræmia almost always end fatally. They are seldom or never seen in the more acute forms of Bright's disease, after scarlet fever, or in association with pregnancy.

Theory of uræmia.—Traube supported the theory, originally put forth

by Rees, that uræmic symptoms were due to cerebral œdema, with anæmia. But often the brain is found after death to be dry; and when it is œdematous, this is probably an accidental result of wasting of the brain.

Nor are they caused by cerebral anæmia, for, as Dr Bradford justly remarks (Allbutt's 'System,' vol. iv, p. 329), although cerebral anæmia can produce convulsions like those of epilepsy, it is very doubtful whether there is provision for a local vaso-motor stimulation or paralysis in the brain. The state of the cerebral vessels is mainly dependent on the state of the vessels at large; contraction of the systemic arteries generally leads to distension of those of the brain; and cerebral anæmia is produced by dilatation of the arteries of the splanchnic area, the limbs, and the skin.

Sometimes minute spots of hæmorrhage are found in the substance of the brain, and Dr Fagge met with two instances of Bright's disease, both with large white kidneys, in which the pons and the bulb were found full of such capillary hæmorrhages. He thought that they are most likely produced by the disturbance of the intra-cranial circulation, which cannot but accompany the uræmic paroxysm. Their occurrence is far too exceptional to admit of their being regarded as its cause.

We are therefore driven to chemical or toxic theories of uræmia. The most obvious supposition, that it depends upon the retention in the blood of urea, is negatived by experiment and by clinical facts.

Voit and Oertel found ('Ztschft. f. Biol.,' 1868) that urea when added to the food of a dog produces no symptoms so long as it can be freely excreted by the kidneys; and more recent experiments show that the mere presence of a large amount of urea in the blood is incapable of producing the symptoms of uræmia, or even of doing harm.

The clinical objections to what may be termed the "urea" theory of uræmia were taken by Frerichs from a work which had shortly before been published by Owen Rees; they were briefly that the occurrence and the severity of the paroxysms bore no necessary relation to the quantity of urine secreted, and that the blood was sometimes loaded with urea without any such symptoms appearing.

In fact, the characters of the urine in cases of Bright's disease, at the time when symptoms of uræmia appear, differ in different cases. As a rule, the amount is much diminished for several days before the symptoms set in; it may even be completely suppressed. But sometimes there is a normal flow of urine, although it contains much less than the due amount of urea; and occasionally the quantity of urine is above normal.*

Fleischer ('Deutsches Arch.,' xxix, 1881) found, as a rule, that the amount of urea excreted by those who had Bright's disease was much diminished; but that when uræmia set in, the amount of urea became increased far beyond the normal, either on the day of the seizure or else a day or two later.

* In a case of Wagner's the patient for three successive days passed seventy ounces daily. In that instance, however, its specific gravity ranged only from 1006 to 1010, so that the excretion of urea and of the other solid constituents of the urine was probably defective. The man had not been œdematous, and there was therefore no reason for supposing that a reabsorption of dropsical fluid had anything to do with the large amount of urine poured out by his kidneys. Wagner, however, remarks that the tissues of the dead body may be found œdematous, when there had been no clinical evidence of it. It seems, therefore, not impossible that the absorption into the blood of such a latent accumulation of fluid may sometimes be the real cause of an excessive flow of urine previous to the development of uræmia, especially as the subsidence of dropsy is known to be frequently followed by uræmic symptoms.—C. H. F.

Although systematic analyses of the urine may show the amount of urea excreted to be only 200 or 150 grains daily, yet in many other cases it is quite as low without any uræmic symptoms arising.

Dr Bradford has found 10—12 grammes of urea in the urine passed in the last twenty-four hours of life by patients dying of uræmia from renal cirrhosis; and this is not a very low output for patients lying in bed and eating little food.

The earliest analyses of the blood seem to have been made by Dr Babington ('Guy's Hospital Reports,' 1836, vol. i, p. 360), who found in a case under the care of Bright himself as much urea in the blood as in the urine! Later observers have found much smaller quantities than this. Wagner says that, instead of the normal proportion of 0.16 or 0.2 part per 1000, there may be 0.4 or 0.6 part, or more. He further cites an observation of Hoppe-Seyler's, who, in the blood-serum of a cholera patient with uræmia, discovered 1.27 parts of urea per 1000. It is true that the quantity of urea in the blood has several times been found to be small. But before we accept this important conclusion, we ought to know exactly at what period of the disease the analyses have been made in which no excess of urea has been detected. If Fleischer's observations are correct, it seems quite possible that in the course of a uræmic seizure, or afterwards, the blood might contain no excess of urea, and yet that a great excess might previously have been present, and have given rise to the attack.

Urea may be discovered in considerable quantity in the gastric and intestinal contents, and in one case of bronchitis and extensive pneumonia, Fleischer found it in the sputum to the amount of about thirty grains in the thirty-seven ounces expectorated during twenty-four hours.

It is a curious fact that in some uræmic patients urea is excreted by the skin. This seems only to occur shortly before death, and scarcely ever without the urine being completely suppressed. Schottin first observed it in 1862 in cholera patients. The 'Guy's Hospital Reports' for 1874 contain a report, by Dr Frederick Taylor, of a patient with Bright's disease, in whom, two days before death, there appeared on the face, neck, and hands white adherent masses which, when removed, were found to be irregularly shaped, with crystalline spiculæ and prisms. They yielded the several reactions of urea. The patient's face is described as having looked as though flour had been sprinkled over it. In some other cases the appearance is said to have been just as though a lather of soap had been allowed to dry on the surface, or as though the beard were frosted.

Frerichs suggested in 1851 that the poisonous agent in uræmia was not urea itself, but carbonate of ammonia formed in the blood by decomposition of urea; and subsequently Treitz amended the hypothesis by supposing that the carbonate of ammonia was produced, not in the blood, but in the stomach and intestine, a vicarious excretion of urea into the alimentary canal first taking place, and the carbonate of ammonia being afterwards reabsorbed into the blood. But this theory of "ammoniæmia," though at one time widely adopted in Germany, is now universally abandoned. For though carbonate of ammonia, injected into the blood of animals, causes symptoms somewhat like those of uræmia, the resemblance is only incomplete, and many other salts produce like effects. Again, many observers fail to detect carbonate of ammonia in the blood of uræmic patients. Frerichs stated that by holding a glass rod moistened with hydro-

chloric acid near the mouth of a uræmic patient the presence of carbonate of ammonia could be recognised in the expired air by the white fumes of chloride of ammonium that were formed. Schottin, however, showed that in many uræmic patients this test completely failed, whereas it often succeeded in other patients who lay in a typhoid state from whatever cause, the carbonate of ammonia being set free from dried secretions within the mouth, and not exhaled from the lungs.*

If neither urea as such nor ammonia carbonate be the poison which causes the symptoms of "uræmia," may we look to lithic acid, hippurates, kreatinin, or any other normal constituents of the urine, as accumulating in the blood when the kidneys are diseased and producing the symptoms in question?

Some have broached the hypothesis that the cause of uræmia is the presence in the blood of products intermediate between urea and the albuminous substances from which it has its origin: such products as kreatin and kreatinin, hypoxanthin and xanthin, or leucin (amido-caproic acid), or aspartic (amido-succinic) acid or tyrosin.

Voit supposed ('Zeitschrift. f. Biol.,' 1868) that uræmia is not due to the poisonous action of any one ingredient of the urine, though he inclined to attribute some share in its causation to the salts of potass. He believed that uræmia may be produced by "any substance which is not a normal constituent of the body if it accumulates in large quantities and is not eliminated."

This vague hypothesis was revived in a more intelligible form by Bomhard, who, in 1887, published experiments on dogs to show that while urea is innocuous, urine is eminently toxic, and sometimes produces convulsions, dyspnœa, contracted pupils, and death by coma.

But there are two objections—the one experimental and the other clinical—which seem to be conclusive against the view that uræmic symptoms are due to poisoning of the brain by urine, or by any of its constituents.

Dr Bradford's remarkable experiments published in the Royal Society's 'Proceedings' for 1892 (vol. li, p. 25) show that the effect of removal of large portions of the kidney in dogs is to produce not diminution, but great increase in the flow of urine and in the excretion of urea; while nevertheless the blood and tissues contain large quantities of urea and other nitrogenous bodies, both crystalline like kreatinin, and amorphous like pigment, and yet no uræmic symptoms are produced.

The other fact, for a clear recognition of which we are indebted to Sir William Roberts, is that symptoms altogether unlike those of uræmia, and holding a different course towards a fatal issue, are presented by cases in which the failure to eliminate urea and the other ingredients of the urine is absolute, but in which the cause of the suppression of the renal secretion is not an affection of the cortex of the kidneys, but obstruction of the ureters. These clinical symptoms are met with in what is called "obstructive suppression of urine" (*v. infra*, p. 685). The absence of uræmia in these cases seems clearly to show that where there is healthy kidney substance, with an active circulation through it, the waste products normally

[* Professor Sée believed that uræmia from urea and from carbonate of ammonia are both real pathological conditions, and that they can be diagnosed from one another during life, but the evidence for each of these beliefs is in almost universal judgment quite inadequate.

excreted in the urine undergo some chemical change that renders them incapable of producing uræmia, notwithstanding that they are retained in the body.

In conclusion, there seems to be no doubt that uræmia is produced by the poisonous action upon the nervous centres of compounds, probably nitrogenous, but not those of the urinary excretion. At present we must refer the condition rather to destruction of the internal metabolism of the kidney than to mere accumulation of urea or other waste products.

In many cases the actual outbreak of convulsions is immediately due to some obvious disturbance of excretion. As Bartels first noticed, the production of profuse sweating in a dropsical patient by a hot bath, followed by hot packing, may at once bring about a series of uræmic attacks. Perhaps the toxic matters, which were safe in the œdematous tissues or serous effusions, are taken up by the lymphatics, and carried in the blood to the cerebral centres. Hence a rapid removal of the dropsy of Bright's disease may bring on fatal uræmia. Possibly the immediate cause of uræmic symptoms is sometimes the sudden failure of the heart to keep up an active circulation through the renal vessels, so that the function of the kidneys, which may for a long time have been more or less impaired, now becomes altogether ineffectual.

11. *Anæmia*.—This is a symptom in so many conditions that it is scarcely a ground for diagnosis of Bright's disease; but next to the blanching caused by actual hæmorrhage, uterine, pulmonary, gastric, or traumatic, and to some cases of chlorosis and essential grave anæmia, there is scarcely any condition which so quickly and profoundly affects the patient's colour. He becomes in acute cases as rapidly white as a patient with rheumatic fever, and in chronic cases as markedly so as a patient with cancer, heart disease, or consumption. In the chronic cirrhotic form of the disease anæmia is less constant and extreme than in tubal nephritis, and loss of flesh is more so: but when the anasarca has disappeared, one is often surprised to find how thin a child is who is recovering from scarlatinal nephritis.

The remarkable symptoms, the pathology of which we have been discussing—albuminuria, dropsy, anæmia, lesions of the optic nerve and retina, cardiac hypertrophy and arterial tension, and the most remarkable and obscure of all, uræmia—are common to all the varieties of Bright's disease, although in very different degrees and frequency.

We now proceed to describe the distinguishing and particular clinical features which belong to the two chief types of Bright's disease, and to their several stages and modifications, as enumerated on pp. 604-5.

I. *TUBAL NEPHRITIS*.*—As remarked before, this name is adopted as the most distinctive and convenient, as well as the one most used in this country and America, but not as implying that the pathological changes in this form of Bright's disease are strictly limited to the secreting tubules of the renal cortex. On the contrary, we shall presently find that the glomeruli, and even the connective tissue, are in many cases markedly affected.

Ætiology.—Parenchymatous nephritis is sometimes definitely traceable to *cold*. Bartels cites three well-marked examples of this: one is that of a patient

* *Syn.*—Parenchymatous nephritis—Tubular nephritis—Desquamative nephritis—Diffuse nephritis—Acute and chronic epithelial catarrh of the kidney.—*Fr.* Néphrite albumineuse.—*Germ.* Croupöse Nephritis.

who was taken ill as the direct result of going to sleep half undressed by an open window on a winter's night, after having spent the evening in dancing; another is that of a man who, while perspiring freely, left his smithy and went out into the open air in his shirt, getting wet through with a sleety rain; the third is that of a skater who broke through the ice, and had much difficulty in extricating himself.

The most striking cases are like those just quoted,—in which acute albuminuria and anasarca follow exposure to wet, not to mere low temperature. The same is true of myelitis and rheumatic fever as compared with bronchitis and pleurisy. The effect of the “chill” is supposed to be a sudden check of perspiration, and consequent congestion of the kidney. But congestion is not inflammation, and the facts show that a chill cannot be the true cause of nephritis: first, because it is as often followed by pneumonia, rheumatism, colitis, or quinsy; and secondly, because acute nephritis often occurs without a chill.

Many cases are undoubtedly the direct result of *scarlatina*; and some in which no definite cause can be found are probably due to a slight attack of scarlet fever during childhood. Cholera, erysipelas, enteric fever, small-pox, measles—all more or less frequently give rise to albuminuria and to some degree of nephritis; but it is very rarely that the renal affection in any one of these diseases leads to permanent albuminuria, to dropsy, uræmia, or any of the symptoms above described, or, lastly, to the anatomical lesions characteristic of any form of Bright's disease.

In one case only has the writer seen the albuminuria of diphtheria followed, and become permanent, with other symptoms of Bright's disease.

In women, *pregnancy* is often a cause of nephritis, as Lever showed many years ago, especially in primiparæ, and, above all, when there are twins. Sometimes the disease recurs in successive pregnancies. It generally appears in the later months of gestation. Probably it is not due to pressure by the gravid womb on the renal veins, nor to the kidneys having extra work thrown upon them in the elimination of effete matters; it is most likely related to the high arterial pressure of pregnancy. Its onset is often insidious, though its course and symptoms are acute; and it is very apt to produce uræmia with puerperal convulsions.

In countries in which *ague* is endemic it is said by some writers to be a frequent cause of Bright's disease; but Osler doubts the fact.

Among the causes of acute nephritis the writer is inclined to regard indulgence in *alcohol* as important; and this was the original opinion of Bright, although it is disputed by many later observers. Dr Fagge held this view, and recorded two cases in point. In one, a solicitor, usually moderate in his habits, acquired temporary albuminuria as the apparent result of drinking sherry in large quantities to induce sleep, at a time when he had a great trouble weighing upon him. Another patient's urine was for many years albuminous during habitual excess in stimulants; he changed his ways, and two years later no evidence of any renal affection could be discovered, and he seemed to have regained his usual health.

The writer has been struck with the rapidity with which albuminuria disappears, when in a severe and recent case of intemperance the patient can be induced to give up his drink. More than once he has seen every symptom of acute tubal nephritis come on during the excesses in liquor of a previously healthy man, and gradually subside when he adopted abstinence.

Hereditary predisposition does not seem to be more frequently observed

than would be accounted for by accident in the case of so common a disease ; but Mr Dickinson recorded one remarkable case, in which it appeared in several generations of the same family ('Path. Trans.,' 1889, p. 144).*

Inhalation of sewer gas has been supposed to be an occasional cause of acute nephritis ; and the list of causes would be incomplete if we did not add that the kidney in some cases contains streptococci, which disappear when the chronic stage is reached. It is doubtful whether the albuminuria of extensive diseases of the skin, particularly large superficial burns, pityriasis rubra, and foliaceous pemphigus should be regarded as septic, or due to suppression of the function of the sweat-glands.

Tubal nephritis is more common in children from three to fifteen years old than in adults, and more common in young adults from sixteen till forty than later in life. It affects male rather more often than female subjects at all ages.

Anatomy.—The appearance of the kidney after death differs considerably, even at the same period of the disease as measured by symptoms ; but three stages are conveniently recognised.

(1) *The large, red, smooth and speckled kidney.*—If the disease has proved fatal during the first two or three months, the kidneys are sometimes found of nearly natural size ; but usually they are enlarged, and occasionally of twice their natural weight. Their fibrous tunic is tense, thin, and easily stripped off. The colour of the surface is dull red, but when incised the medulla presents a much deeper reddish-purple tint than the cortex. If there has been complete suppression of urine, or if death has been due to convulsions attended with great pulmonary congestion, the kidneys are often found gorged with blood, and of a dark chocolate colour (as in Dr Dickinson's 5th plate). In most cases red points are seen scattered over the cortex ; some of them are Bowman's capsules filled with blood, others are punctiform hæmorrhages, between the Malpighian bodies.

In some cases, including those due to scarlet fever, the kidneys show little deviation from their natural appearance ; but, as a rule, the scarlatinal kidney is swollen, bright red, and dripping with blood when cut.

The principal seat of morbid changes is the cortex. The epithelium of the convoluted tubes first becomes cloudy and granular, and afterwards proliferates, so as to fill them with masses of irregular or rounded cells. These are seen in sections blocking up the tubules and greatly increasing the thickness of the cortex. In the acute stage the epithelium and granules are mingled with blood-discs and leucocytes ; as the affection becomes chronic, minute oil-drops appear and render the tubes black by transmitted light (see Grainger Stewart's 3rd plate, fig. 1). Osmic acid shows this change early.

The histology of scarlatinal cases was worked out by Klebs, and in this country by Klein and by Greenfield. It is now agreed that the most constant lesions concern the glomeruli and Bowman's capsules ; not only do the nuclei of the capillary tufts of the glomerulus proliferate, but there is also an abundant growth of nuclei within the capsule, leading to adhesion between it and the glomerulus, and ultimately to compression and atrophy of the latter. The connective tissue around the glomerulus also becomes crowded with nuclei, which ultimately develop into fibroid tissue ; and the

* Is it possible that in this case it may have been habits of living which were hereditary rather than their result ? A similar explanation might perhaps apply to the mortality of the ducal family of Pomerania as described in 'The Amber Witch.'

afferent artery of the glomerulus undergoes a peculiar hyaline change. Sometimes these lesions are limited to a few of the glomeruli only; sometimes they are wide-spread. It is obvious that the obliteration of the space naturally existing between the tuft and the capsule that encloses it must completely abolish the functions not only of the glomerulus itself, but also of the whole length of the convoluted tube that corresponds with it; and the changes in the tufts themselves no doubt obstruct the blood-supply to the convoluted tubes, and so affect the nutrition of their epithelium. Consequently some pathologists were disposed to see in "glomerulo-nephritis," as they term it, the fundamental morbid process that follows scarlet fever, and to regard the lesions of the tubal epithelium as only secondary.

At present a less exclusive view is generally taken than that just mentioned. That glomerulo-nephritis is not peculiar to scarlatinal cases is shown by a typical example Cohnheim met with, in the kidneys of a man who died some weeks after having his skin rubbed all over with petroleum. Moreover, the glomerular lesion is not only present in the early stages of nephritis consecutive to scarlet fever, but also to that which follows cold or pregnancy, or begins without any assignable cause.

That the convoluted tubules are also affected in these cases, and that leucocytes are found between them (interstitially) as well, are well-ascertained facts.

(2) *The large, white, smooth kidney.**—In cases that have lasted three or four months or more, the kidneys present appearances still more obviously morbid. The cortex has now an opaque white or whitish-yellow colour, both on its surface and on section; and this contrasts with the red colour of the medullary portion of the pyramids. It is also much thickened, and the kidneys are sometimes larger than those in the reddish-grey or chocolate-coloured stage. In three cases Dr Fagge found the weight of a pair of such kidneys twenty-eight and a half or twenty-nine ounces, and in a fourth Dr Moxon found them weighing within half an ounce of three pounds. Only occasionally are histologically similar kidneys not above natural size.

The shortest time within which Moxon and Fagge saw the kidneys assume the "large white" character was five or six weeks; more often the disease has lasted several months. In two of the cases just mentioned of great enlargement the patient had been ill for fourteen months. On the other hand, in one instance the kidneys were still of a brick-red colour at the end of six months. It must be remembered that a considerable proportion of cases of tubal nephritis fatal at this second stage are of insidious origin and slow development, so that their duration cannot be fixed.

Probably many cases that used to be classed as examples of the "large white" variety of Bright's disease were lardaceous. Kidneys from the bodies of syphilitic patients, or of those who had phthisis, caries, or other suppurative disease, should never be set down to primary tubal nephritis until the absence of the lardaceous change has been determined by microscopical examination.

Histologically, the most striking appearance in sections of the large white kidney is the accumulation of immense quantities of oil-drops in the epithelial cells of the renal tubes. It is this that gives the opaque yellow colour with reflected light; in thin sections, viewed by transmitted light,

* Including the *pale, marbled, or mottled kidney*, and the *large, granular, smooth kidney* of Bright—granular describing the appearance to the eye, not the feel of the hand. (See his 1st, 5th, and 7th plates in the 'Guy's Hosp. Reports' for 1838.)

the tubes appear dark, or even black. The stroma of the cortex also is closely studded with fat-granules. Hence such kidneys were described by Johnson as "fatty;" but this was a mistake, as he afterwards admitted. They are only "fatty" as in an abscess, or a phthisical lung. Primary fatty degeneration of the kidneys is a totally different condition (*infra*, p. 704), sometimes found in obese persons who also have fatty liver, or in cases of poisoning by phosphorus. Nor must it be assumed that all large white kidneys are fatty even in this sense; for those which are translucent and greyish white are often more lardaceous than fatty.

Dr Greenfield, in his summary of renal pathology in the Sydenham Society's 'Atlas,' speaks of cases, especially in pregnant women, in which the microscope shows that the lesions are almost entirely "interstitial." One marked character of such large white kidneys is their toughness, almost like that of caoutchouc, agreeing with the condition of congested kidneys in chronic disease of the heart, and contrasting with the soft pulpy texture of those in which the tubal epithelium is alone affected. But the fact is that in almost all cases of advanced "parenchymatous" nephritis, interstitial lesions are present to a greater or less extent. This has been insisted on by Mahomed and Saundby in this country, and in Germany by Weigert, who says that he has for years vainly sought for a specimen altogether free from interstitial changes. The leucocytes are not uniformly diffused through the cortex, but form patches of nuclear growth, which afterwards develop into tracts of connective tissue. The glomeruli also have their capsules thickened, and are gradually converted into structureless cysts.

(3) *The contracted white kidney.*—If tubal nephritis runs on long enough, without ending either in recovery or in death, the kidneys at last become shrunken, small, and rough on the surface, though they still retain more or less of the opaque whitish-yellow colour. This continues to distinguish them from kidneys affected with the cirrhotic form of Bright's disease; but the distinction is lost when primarily cirrhotic kidneys become the seat of secondary parenchymatous changes. The granulations are generally more irregular and less minute than in primary cirrhosis.

The occurrence of a granular stage as the ultimate issue of nephritis arising from scarlet fever, or any other cause that commonly produces a large kidney, was at one time denied; but it has now been clearly established by Dickinson, Grainger Stewart, and many other observers. A typical example is figured in plate iii of the Sydenham Society's 'Atlas of Pathology.'

The case was that of a girl aged ten, who, rather more than two years before her death, became dropsical as the result of scarlet fever. After four months she recovered, but a year later the face began to swell from time to time, and she died at last with cerebral symptoms. At the autopsy the kidneys were found to be very small indeed, with thick, opaque capsules, hard, tough, and puckered on the surface, presenting on section yellowish-grey masses.

The records of *post-mortem* examinations at Guy's Hospital contain a certain proportion of more or less similar cases in young subjects, in which the weight of the pair of kidneys was between four and eight ounces. In most of them the history afforded no clue as to the date at which the disease had begun; but in some instances it is recorded that there had been an illness attended with dropsy several years before death.

A case occurring at the London Hospital, in a young woman of twenty-four, is figured in the same 3rd plate of the Sydenham Soc. 'Atlas.' The patient was said to have been

ill for only three months; but her kidneys were found by Dr Sutton to be reduced to about half their normal size, to be "very granular, and of a reddish colour, everywhere mottled with a yellowish or purplish or greyish substance."

The fact that most patients who do not recover die during the "large red" or the "large white" stage of nephritis explains why these cases of a third "contracted" stage are comparatively rare. The writer has, however, met with three in which the clinical characters and history were confirmed by examination after death.

Symptoms.—The parenchymatous or tubular form of Bright's disease varies in its mode of onset in different cases. When due to scarlet fever, or to a definite chill, it may begin with a rigor and pyrexia.

More commonly the earliest indication of the patient's illness is the occurrence of *dropsy* with *anæmia*. This may first appear in the loose tissue round the eyes, and in slight cases it is especially noticeable before the patient gets up in the morning; the lower eyelid is oedematous, and the conjunctiva also. This conjunctival oedema produces the "bright" eye of renal disease, and "the tear that does not run over." Generally the dropsy also affects the limbs, and almost constantly the lower part of the back, as far down as the sacrum—"the lumbar cushion." The external genitals, too, are found swollen, and the prepuce is sometimes so stretched and twisted that we wonder it so seldom interferes with micturition. In severe cases the whole body becomes bloated, while the extreme anæmia, which rapidly develops itself, gives the skin a dead white, wax-like colour.

The temperature and pulse are little affected, but the appetite is often bad, the tongue furred, and the bowels constipated. Vomiting is sometimes a marked symptom, and there may be much headache. Pain in the loins is often entirely absent, but sometimes it is severe, radiating to the groins and down the thighs.

The *urine* in the acute stage is always scanty, and sometimes, though the patient is constantly striving to micturate, a few drops of blood-stained liquid may be all there is to pass. Complete suppression is a very grave symptom, and generally points to a fatal issue. When some ounces of urine are secreted daily it is of high specific gravity (1025—1030), and either red from the presence of blood, or brown like strong tea or porter. This tint is due to acid-hæmatin; but if the blood is so abundant as to neutralise the urine, the colour is red or pink. A small amount of blood in acid urine gives the characteristic "smoky" tint. On standing, a chocolate-coloured deposit is thrown down, containing blood-discs, epithelial cells, and casts—some hyaline, others full of blood-discs or epithelial cells, or brown granules from broken-up red corpuscles.

Albumen is, of course, always present in the urine when there is blood; but it is often not so abundant as it is a little later, when the hæmaturia has passed off. The late Dr Mahomed described, in the 'Med.-Chir. Trans.' for 1874, a *pre-albuminuric* stage in acute nephritis, when no albumen can be detected, but the guaiacum test (not a certain one) indicates the presence of blood, and the sphygmograph shows increase of arterial tension. Bartels also mentions cases in which, when scarlatinal dropsy first sets in, the urine, though exceedingly scanty, was ex-albuminous; and he cites a case of Henoch's, in which no albumen could at any time be detected, except on the day before death, when the patient was cyanotic and almost pulseless, after an attack of convulsions. Commonly the amount of albumen ranges

from 2 to 5 per cent., and the total quantity passed in the twenty-four hours is from 80 to about 400 grains. The excretion of urea is greatly diminished, falling to half the normal amount, or even less.

When tubal nephritis ends in recovery in the course of a few weeks—as occurs in the majority of scarlatinal cases, and in many of those due to other causes—the dropsy and the other symptoms subside, and the urine gradually recovers its normal characters. First it loses its blood, and then becomes more abundant, of lower specific gravity, and paler; the quantity of albumen becomes less and less, until at length there is none.

Chronic course.—When the disease runs on for months, the dropsy continues, or increases, and we see the symptoms of chronic tubal nephritis develop. The patient remains bloated and anæmic, for “large white kidneys go with a large white body.” He lies helplessly in bed, his back propped up with pillows, his legs stretched stiffly out before him, or supported by a pillow under the knees. The swelling of the external genitals is often extreme. The scrotum looks like a bladder full of water, and is so large that there is no room for it between the thighs; while the prepuce is distorted, swollen, and almost translucent. Its twisted shape is compared in Germany to a postboy’s horn. Sometimes the cuticle over some of the distended parts cracks, and the dropsical fluid oozes out in such quantities as to soak through the bedding. This may cause considerable tracts to become excoriated, and ultimately to be covered with pale granulations, which, when they skin over, give the surface a warty appearance. Or inflammation may set in, attended at first with a pink flush (*erythema leve*), like that of the milder forms of erysipelas, but sometimes leading to more or less extensive gangrene of the skin, and even of the subcutaneous tissues. Papular or exfoliative forms of dermatitis are not uncommon, independently of the irritation of urine or exuded serum, or of tension of the integuments (*cf.* p. 623).

A remarkable effect on the skin of the distension caused by the anasarca is rupture of the papillary layer, leading to atrophy of the tissue between the horny cuticle and the deeper layers of the cutis. At first reddish lines appear through the thinned layers, afterwards they become of a dead white. They appear on the abdomen, like the *lineæ gravidarum*, but also on the loins, thighs and buttocks, sometimes in regular rows like the stripes of a zebra. They persist indefinitely, like the atrophic stripes after subsidence of effusion into the knee-joint or after pregnancy; but they are quite visible while the swelling is at its height.

In this more advanced stage the state of the urine is very variable. It may still be scanty; but more frequently it gradually becomes abundant and pale, and its specific gravity falls below the normal, to 1010 or 1005. It is still albuminous, sometimes highly so; indeed, the quantity of albumen sometimes reaches 5 per cent.; but as the disease goes on, the amount often lessens. A tinge of blood may be seen from time to time, but not as a rule. Casts are commonly found in abundance, some of them hyaline, others containing leucocytes, epithelium, or oil-drops, and others opaque with fat-granules.

The characteristic fatty casts are not the only vehicles for excretion of the products of nephritis, for Dr Hamilton gives reasons for believing that the greater part is absorbed into the intertubular spaces and removed by the lymphatics (*Journ. of Anat. and Phys.*, vol. xxv, p. 198).

The excretion of urea is at all periods of the disease much below the normal amount. Even when the flow of urine becomes more abundant, the

total quantity of urea excreted in the twenty-four hours does not increase. Albuminuric retinitis is of frequent occurrence (p. 624); and pleuritic effusion or œdema of the lungs is still more common; sometimes hydrothorax on one side which is free, and œdema on the other, where there is adherence of the pleura.

Event.—Some cases of tubal nephritis prove fatal in the acute or in the chronic stage, as the result of the dropsy or of inflammation; most often from accumulation of fluid in the great serous cavities of the chest, and only rarely from œdema of the larynx. In many instances death is due to pulmonary œdema, and occasionally to pneumonia. In others it is brought about by acute pericarditis. A few cases end by acute uræmia at an early stage; while in those following scarlatina, failure of the heart, from dilatation of the left ventricle, is often the direct cause of death.*

The following case is an example of a rare but fatal complication of tubal nephritis:

A youth of seventeen was admitted into John Ward in November, 1890, with acute parenchymatous nephritis and pleurisy. His temperature was raised and he was delirious, but there was no vomiting, and no sign of pneumonia or of tuberculosis or injury. The urine was scanty, albuminous, and contained blood and casts: soon after it became suppressed, but after bleeding it was secreted again and passed up to death. He died within a week, and pus and lymph were found effused over the vertex and sides as well as the base of the brain. The kidneys, which weighed 15 oz. together, were smooth, chocolate-coloured, and dripping with blood on section. There was no pericarditis, but the left cardiac ventricle was acutely dilated, not hypertrophied.

Complete recovery is seldom seen when the disease has lasted many months, still more rarely when prolonged for years. But it is surprising how symptoms will sometimes subside and disappear, so that even after the patient has been waterlogged and has had alarming uræmic attacks, he yet recovers.

A striking instance of the removal of albuminuria of long standing by treatment is the case of a medical man, aged twenty-six, whose urine after an attack of scarlet fever was continuously albuminous after meals for more than six years. By the advice of Dr Johnson he was strictly dieted, and at the end of nine months the urine became normal, and remained so eighteen months later ('Brit. Med. Journ.,' 1879).

A young man of twenty was under the writer's care with acute albuminuria, excessive anasarca, ascites, and anæmia. He was tapped and continued dropsical for twenty months; yet he finally recovered and was able to go about again.

II. LARDACEOUS DISEASE OF THE KIDNEY.†—From the time of Rokitsansky a lardaceous affection of the kidneys has been described as one of the forms of Bright's disease, and Wilks believes that one of the specimens figured by Bright himself as a "large white kidney" was really a case of this kind. In the liver and in the spleen the lardaceous change is never associated with inflammation; but in the case of the kidney, tubular or diffuse ne-

* See Dr Goodhart's paper in the 'Guy's Hospital Reports' for 1879 (3rd series, vol. xxiv); and, on this and other clinical points in the origin and natural history of scarlatinal nephritis, Dr E. W. Goodall's report, based on a very large number of cases at the London Fever Hospital (*ibid.*, vol. xlv, p. 91).

† *Synonyms.*—Rokitansky's Speckniere—The waxy kidney of the Edinburgh pathologists—Virchow's amyloide Nierenentartung—Depurative nephritis of Dickinson.

phritis accompanies the same degeneration. This gives to lardaceous disease of the kidneys a special clinical importance; and therefore the causes and pathology of this remarkable form of degeneration may be better discussed here than elsewhere.

The lardaceous degeneration.—This morbid condition as it affects the kidneys, liver, and spleen was first described by Rokitsansky under the unfortunate double title of *Speckige Infiltration* (infiltration with material which has the appearance not of bacon, but of bacon rind) and *Colloide Entartung* or colloid metamorphosis, the latter term being used by Schrant. Virchow thought the new material was allied to cellulose, the vegetable carbohydrate then recently discovered in the outer tunic of Ascidians; and believed that by oxidation he could transform the material into starch, and obtain the well-known blue reaction with iodine. He therefore supposed it to be related to the corpora amylacea recently discovered in the brain, and in an unlucky moment perpetuated the memory of almost the only error committed by this great master in pathology, by calling the lardaceous substance of Rokitsansky—already accepted in the form *lardacé* by Cruveilhier and other French writers—*amyloid*, *i. e.* “starch-like.” Meanwhile Meckel had argued that it was chemically closely allied to, if not identical with, cholesterine; the late Dr Sanders and other pathologists in Edinburgh had named it from its physical, not chemical, properties “waxy;” and Budd had (as it turned out, more nearly to the truth) called it an “albuminous” or albuminoid degeneration of the liver. This last term was used by Gull and other clinical teachers in London at and after the time of Virchow’s hypothesis being published. At Berlin and throughout Germany the term “amyloid” was at that time (1856—1866), and still is, almost universally used; and at Vienna, in 1864, the writer found Oppolzer still speaking of the condition as “colloid.”*

The first step in the chemical study of this disease was Meckel’s discovery, that the tissues affected turn a walnut or mahogany-brown colour with iodine, whereas healthy tissues remain pale yellow. Next came Virchow’s statement that by the addition of dilute sulphuric acid before the tincture of iodine, a more or less distinct blue or purple tint can be detected. But most observers agree with Ziegler that this reaction is at best imperfect. Kekulé demonstrated that the “lardacein” (as it is now called in Germany, and by Pavy and Odling in this country) is not a carbohydrate but contains nitrogen; and, finally, Kühne and Rudneff isolated it, and proved that it is really allied to albumin.

Kühne’s plan was very ingenious. Lardacein is insoluble, and unaffected by reagents, alkalies, acids, or digestion. He therefore submitted a lardaceous liver to artificial gastric digestion, and when everything else was dissolved away, analysed the residue of lardacein, and found that its percentage composition corresponded to a proteid.

Tests.—An aqueous solution of iodine and iodide of potassium is the best test for the lardaceous material. The cut surface of the organ to be tested must be first washed free of blood, and is then lightly brushed over with the solution. A considerable degree of lardaceous change becomes apparent in a few seconds, by the formation of brown spots or streaks. If.

* “Lardaceous” is used in France and America, and is adopted in the Nomenclature of the Royal College of Physicians. Moreover it has no misleading meaning, and is distinctive.

however, the change is very slight in amount, it may be perceptible only with the microscope, after application of the iodine to a thin section. In 1875 Jürgensen made known in 'Virchow's Archiv,' and also Cornil in the 'Archives de Physiologie,' the fact that methyl-violet (prepared by the action of iodide of methyl upon aniline) gives a fine microscopic staining-reaction with lardaceous organs. The affected parts slowly become red, whereas the rest of the section is stained blue. The chief advantage of this over the iodine test is the definiteness with which the reaction remains limited to certain elements in a complex structure. Thus it is invaluable in determining whether the secreting cells of an organ, or the adjacent capillaries, are the seat of the morbid change. Another important point is that it enables permanent preparations to be made and preserved in glycerine. On the other hand, it cannot be used as a preliminary routine test in the *post-mortem* room.

Even without staining, lardaceous organs may be recognised by their swollen, homogeneous, glistening appearance, which cannot be mistaken by a practised eye.

Pathology.—The process by which lardacein is substituted for the natural proteids of the tissues is still doubtful. Whether this material is elaborated where it is found, as a "degeneration or metamorphosis" of the normal structures, like fatty or granular or pigmental degeneration of muscle, or whether it is a "deposit" derived from the blood, like the fibrin of serous inflammation, or the earthy salts of atheroma, is still uncertain. The most prevalent theory seems at present to be that it is exuded from the blood, and coagulates between, not within, the elements of the tissues. It cannot, however, be detected in the blood.

Locality.—In every organ the first parts to become lardaceous are the small arteries. In the kidney the change is often very partial, occurring only in certain glomeruli, or in certain loops of a glomerulus. By carefully searching, the lardaceous change may be often detected in other parts than those commonly recognised.*

As an important pathological process, however, lardaceous degeneration is limited to a small number of organs, these being the kidneys, the liver, the spleen, the intestine, the thyroid, and the adrenals; and in the last two, so far as is at present known, it gives rise to no symptoms and has no clinical significance.

Dr Goodhart found that of 150 cases at Guy's Hospital the kidney was affected 110 times, the spleen 99, the liver 73, and the intestine 63. At the London Hospital Dr F. C. Turner found, among 58 cases, the spleen affected 48 times, the liver 30, the kidneys only 15, and the intestines 10 ('Path. Trans.,' vol. xxx, p. 517).

Ætiology.—Many writers speak of scrofula, chronic tuberculosis, cancer, malaria, rickets, and syphilis, as being all alike constitutional diseases which predispose to the development of lardaceous degeneration.

The vague ætiology given by Rokitansky, and even by Bartels and by Eichhorst, has been replaced by a strictly limited and defined view of the

* As examples may be cited Burow's case of lardaceous degeneration of laryngeal tumours, Ziegler's case of lardaceous nodules at the base of the tongue, Birch-Hirschfeld's statements as to the presence of lardaceous material in mesenteric glands after enteric fever, and the curious examples of a lardaceous change in the vessels of the conjunctiva recorded by Sämisch and by Leber. References to these observations may be found in a paper by Kyber in vol. lxxxi of 'Virchow's Archiv.'

necessary antecedents of lardaceous disease, and this is chiefly due to Dickinson and Fagge.

In the first place, it is more than doubtful whether "scrofula" (in other words, the presence of tubercle in the lymph-glands or joints) has any influence, apart from its tendency to cause suppuration. In the second place, it is certain that syphilis leads to the lardaceous change, even when formation of pus has occurred only to the most insignificant extent, if at all. We may therefore reduce the known causes of the lardaceous change to two—chronic *suppuration* and *syphilis*; sometimes combined, but capable of acting separately.

In many instances it is only by the discovery after death of fibrous degeneration of the testes, or gummata or cicatrices in the liver, that the syphilitic character of the case is made out. In this respect there is a wide difference between the two great causes of the lardaceous change. For the occurrence of suppuration can seldom be overlooked during the patient's life, except perhaps when it follows intestinal ulceration (as in dysentery), or when there is no external discharge of pus at all, but merely a large deep-seated abscess.

Wilks, who published 96 cases of lardaceous disease in the 'Guy's Hospital Reports' for 1856 and 1865, referred the condition to long-standing and deep-seated cachexia, particularly from tuberculous or syphilitic disease of bones. Grainger Stewart published 27 cases in the 'Brit. and For. Med.-Chir. Rev.' for 1866, most of which occurred after caries or necrosis, phthisis or syphilis. Dickinson, in the 'Med.-Chir. Trans.' for 1867, published sixty cases from St. George's Hospital, 46 of which were certainly, and four more probably, the result of chronic suppuration. He believed that the draining away of alkaline salts in the pus transformed fibrin into lardacein, and proposed the adjective depurative, instead of amyloid, waxy, or lardaceous.*

In 1876 Dr Fagge brought before the Pathological Society a tabulated statement as to what appeared to have been the ætiology of 244 cases of lardaceous disease of the viscera, collected for him by Mr H. F. Lancaster from the records of autopsies in Guy's Hospital over a period of twenty-one years (1855—77 inclusive).

In 154 there had been *prolonged suppuration*. Of these 67 were cases of *phthisis*; in 51 there was disease of some joint (generally the hip- or knee-joint), or caries of the spine, or of some other bone; in the remaining 36 there were a variety of affections, amongst them chronic abscess, empyema, dysentery, and tuberculous ulceration of bowels, calculous and tuberculous pyelitis, cystitis from stricture, and bedsores of long standing as the result of disease of the spine, and a small residue of malignant ulcers.

In 5 other cases suppuration had been present, but only in small amount. Thus in one instance there had been chronic discharge from one ear as well as from the nose; in another, one testicle had been inflamed and suppurating as the result of a blow two months and a half before death, but with open discharge only for a fortnight; and in a third there was merely tuberculous peritonitis with caseous disease of the mesenteric glands.

In five or six of the 154 cases that were clearly due to prolonged suppuration, some idea could be formed of the length of time required for the development of lardaceous lesions. One patient had a carbuncle eight months; another had pelvic suppuration for the same period; a third had a

* See, however, his larger table of 201 cases on p. 268 and his withdrawal of the term depurative on p. 266 of the 3rd volume of 'Allbutt's System.'

bedsore *seven months*; in a fourth a sarcomatous growth had been discharging for *four months*. In a fifth case there had been fracture of the spine three months before death, bedsores *two months and a half*, and also a double empyema; but syphilis was probably also present, inasmuch as the testes presented fibroid changes and there was a scar in the groin. A sixth patient had suffered amputation of the leg *three months and a half* before death, on account of a compound fracture with abscess; in that instance it is particularly noted that both in the liver and in the spleen the lardaceous change was just beginning.

Next, among the 244 cases of lardaceous disease there were 76 in which there was satisfactory proof (either from the history, or from appearances after death, or from both together) of *syphilis*; and in 3 others there was at least a suspicion of its presence. In about 34 of these 76 cases there was evidence of there having been caries or suppuration, leaving 42 to be ascribed to syphilis alone. In no instance is it stated that the syphilis had been inherited; but Bartels speaks positively of having seen lardaceous affections in cases of ulceration of the skin or bones from inherited syphilis.

Of the 244 cases, there are thus left only six examples of lardaceous disease that were not accounted for by the presence either of syphilis (79) or of suppuration (159); and in some of them the notes of the autopsy are incomplete, the state of the testes, in particular, being unrecorded; so that possibly in each of these six cases the cause of the lardaceous change was really syphilis.

Since the first edition of the present work was published, the writer has obtained a series of statistics in continuation of those just given. They were collected by Dr H. J. Campbell, now of Bradford, from the *post-mortem* records of Guy's Hospital, and extend from 1876 to 1889 inclusive. The total number of cases of lardaceous disease recorded in these fourteen years was 302. Prolonged *suppuration* had been present in 124; there were 35 cases of (probably tuberculous) disease of the joints, usually the knee, hip, or sacro-iliac synchondrosis; 31 cases of vertebral disease, often associated with psoas abscess; 5 cases of caries or necrosis of other parts of the skeleton; 5 of empyema; 11 of pelvic abscess; 2 of chronic caseous disease of the kidney; and the 35 remaining cases were of suppuration from chronic abscesses, bedsores, ulcers, &c. There were 121 cases of pulmonary *phthisis*, together with one of acute general tuberculosis, and another of tuberculous disease of the uterus and of the peritoneum. There were 43 cases of *syphilis*, shown by anatomical changes after death, whether or not by symptoms observed during life.

These three chief causes, therefore, account together for 289 cases out of the total of 302. The remaining 13 cases were very miscellaneous, as is shown by only two coming under the same denomination. One was a case of acute Bright's disease, one of chronic tubal nephritis with cirrhosis of the liver, and one of valvular disease of the heart with a large white kidney. In another case there was found a renal calculus and shrivelled kidney, which was probably once the seat of suppuration. Two were cases of chronic non-tuberculous disease of the lung without any history of prolonged or excessive purulent expectoration. In two an adherent pericardium was found, one cut short by diphtheria, the other with a history of chyluria seven years before. One was a case of hobnail liver, and one of glio-sarcoma of the brain and lung. There remain only three cases, two of multiple lymphomata (Hodgkin's disease), and one in which lardaceous degeneration appeared in cancerous glands.

In many of the thirteen cases just enumerated the lardaceous change was slight, and limited to the liver or kidney or lymph-glands. Probably some of them might have been brought under the head of suppuration or of syphilis; but it is best to leave them as the conditions stand recorded by independent observers in the records of autopsies.

The result is strongly to corroborate the conclusions derived from Dr Fagge's series. Putting both together, we find that in 546 cases of lardaceous disease occurring consecutively in the same hospital during the thirty-six years from 1854 to 1889 inclusive, there were 216 which could be ascribed to suppuration, 190 cases to phthisis, and 122 to syphilis, leaving only 19 unaccounted for by the anatomical evidence of previous disease.

Dr F. C. Turner referred 42 of his 58 cases ('Path. Trans.,' xxx) to prolonged *suppuration* (including 20 cases of *phthisis*), 8 or 9 to *syphilis*, and 6 to *malignant disease*, while the remaining 2 were unaccounted for. Of the six cancerous cases, in one there had probably been lues and certainly considerable suppuration, in another there had been six months' ulceration, in a third there was history of syphilis and ague, in the fourth a history of ague; and one was a case of Hodgkin's disease, with lardaceous change in the enlarged lymph-glands. One of the six cases, it must be confessed—epithelial cancer of the tongue—is difficult to explain away.

Age and sex.—The period of life at which lardaceous lesions due to syphilis are most apt to occur is from thirty to forty. As the result of protracted suppuration, on the other hand, the degeneration is rather more frequent from twenty to thirty than from thirty to forty. It occurs, too, in adolescents, and even in children; there has been one case at Guy's Hospital in a boy only four years old. Above the age of fifty lardaceous disease is decidedly uncommon, but we have had one instance of it in a man of sixty-five.

Among our cases, male patients were more numerous than females in the proportion of two to one; probably because suppuration from injuries and tertiary syphilis is more common in men than in women.

Anatomy of the lardaceous kidney.—Sometimes the lardaceous degeneration of its arteries is the only change in the kidney discoverable, whether by the naked eye or with the microscope. This, however, is rare, for in a collection of more than sixty cases of lardaceous kidney taken from our records at Guy's Hospital, Fagge found only three or four uncomplicated.

A kidney with no other lesions than this peculiar degeneration is of the natural size, and looks smooth and healthy, except that a keen eye may perhaps perceive the glomeruli to be somewhat more distinct and more translucent in appearance than is natural. On the addition of iodine the change is most marked in the glomeruli, often confined to a few, and even in the same glomerulus to one or two of its coils. Not infrequently the afferent arteries are involved as well, their middle coat being the first part to suffer. Sometimes the reaction is obtained, not in the cortex, but in the straight vessels of the pyramids, or in both together; and when the change is far advanced, the vessels which surround the renal tubes are also affected.*

Far more frequently, as above stated, lardaceous kidneys are the seat of

* Dr Dickinson's 8th plate gives an excellent picture of the appearance of the kidney to the naked eye, and his 9th and 10th of its histology; the latter is also well illustrated by Sir T. G. Stewart's 5th and 6th plates.

other lesions also, by which they are greatly increased in size. In about a quarter of the sixty cases the weight of the two organs together was from twenty to twenty-seven ounces. The appearance of such kidneys is peculiar, and justifies the term "waxy" or, in German, *Butternieren*. Their surface is smooth and pale yellow, with conspicuous stellate veins; their section is shining and polished, of a semi-translucent pale yellowish colour, sometimes showing the grey swollen glomeruli, sometimes mottled with creamy opaque spots and streaks, where fatty granules are present in abundance. The enlargement of the kidneys is undoubtedly due to accumulation of inflammatory products, not only in the tubes, but also in the interstitial tissue. But why this diffused nephritis should arise is difficult to say.

Cohnheim inclined to believe that it is an independent result of the same cause which produces the lardaceous change. But diffuse nephritis, apart from the lardaceous change, is not a common result of syphilis or of suppuration. Moreover, in the liver and spleen no secondary effect is produced.

The ætiology of the lardaceous change, as now ascertained, clearly excludes the view of Cornil and Ranvier that it is preceded by the nephritis.

We have therefore no alternative but to suppose that in some way lardaceous degeneration must cause the nephritis; as we see lardaceous degeneration accompanied by enteritis.

Sometimes lardaceous kidneys are found after death to be smaller than natural. This occurred in about ten of the sixty cases collected from our *post-mortem* records. As a rule the loss of size was not very obvious, the two organs weighing together not less than seven and a half or eight and a half ounces; but in one instance the weight (of what happened to be a "horseshoe" kidney) was only three and a half ounces. There was always, however, much irregularity and shrinking of the surface—a more or less markedly "granular" condition; and the loss of substance was no doubt far more considerable than was indicated by the scales, inasmuch as the kidneys still retained inflammatory exudation, as well as the lardaceous material itself. Such wasted organs clearly represent the most advanced stage of the lardaceous affection, and it is fair to assume that its course has been more than usually slow and protracted. But there is no evidence that kidneys that ultimately become thus reduced in size have passed through an earlier stage in which they were enlarged. Some pathologists account for such cases by supposing that the kidneys were already contracted and granular before they began to be affected with the lardaceous change, as the result of syphilis or protracted suppuration. There was, in 1866, at Guy's Hospital an instance of this accidental association of the two morbid processes in a woman who died at the age of forty-five of cerebral hæmorrhage, and whose kidneys, besides being lardaceous, were granular and full of minute cysts. The hypothesis of previous interstitial nephritis could, however, hardly apply to two of our cases, each of which occurred in a young man of twenty-four; in one of these the two kidneys weighed seven and a half, in the other six and a half, ounces.

Symptoms.—The characters of the urine secreted by lardaceous kidneys were first studied by Traube in 1858; and his account has been confirmed by Grainger Stewart and others who have since taken up the subject. No doubt cases occur in which the state of the urine alone may suggest that the kidneys are lardaceous; but such cases are rare. We have seen how seldom the lardaceous change is found without other lesions in the kidney, and it

is therefore unlikely that the urine will always present the same characters. There is much force in Cohnheim's remark, that the existence of "simple" lardaceous kidneys can scarcely ever be determined, except in patients who have fatal disease of other organs which causes them to be wasted, to be anæmic, and to suffer from pyrexia, diarrhœa, and other symptoms that must in themselves disturb the health. Bartels appears to be right in declaring that there is no evidence that the lardaceous change in itself interferes with the excretion of urea; if this is diminished, it is probably due to concomitant tubal or interstitial nephritis. The effect of the lardaceous change in the glomeruli appears only to render their walls more permeable than before to water, salts, and urea.

With regard to the *quantity* of urine secreted by lardaceous kidneys, different writers make very different statements. According to Grainger Stewart, unless an extreme degree of nephritis is present, it is excessive, ranging from fifty to two hundred ounces daily. He also maintains that an increased flow of urine is in many instances of great clinical importance as an early symptom of the lardaceous affection, preceding albuminuria. George Johnson supported him in both these statements, and so does Dickinson, who puts the daily amount at from fifty to ninety ounces. Bartels, however, gives much smaller average amounts—from fifty to sixty or seventy-six ounces; and he believes that there is never such a degree of polyuria as occurs in some cases of granular disease of the kidneys. He mentions a case in which the average daily secretion amounted to less than seventeen ounces. Wagner found that in the majority of cases of lardaceous nephritis—almost constantly in the last few weeks, but sometimes in the whole course of the disease—the urine is more or less scanty, sometimes with intervals of a few days when it is normal or increased in quantity.

Perhaps these discrepant statements depend partly upon the care taken by different observers to recognise an underlying lardaceous change in the kidneys that would formerly have been set down as examples of tubular or interstitial nephritis. Wagner says that in the *post-mortem* room the lardaceous form of Bright's disease is seen "much more frequently than chronic parenchymatous nephritis or than cirrhosis of the kidney;" and Dr Fagge's experience at Guy's Hospital accorded very much with this. But in this country and abroad there is no doubt that, in the last twenty years, the frequency of lardaceous disease has greatly diminished, owing to the increased safety and success of surgical operations.

The specific gravity of the urine varies with the quantity; it may be as low as 1003 or 1005, or it may occasionally reach 1025. For several days before death the urine is often very scanty, and of high density.

In almost every case *albumen* is present. Lecorché maintained that, so long as the lardaceous change is uncomplicated with nephritis, the urine is ex-albuminous; but one at least of our cases at Guy's Hospital afforded proof to the contrary. Bartels goes too far when he declares that albuminuria is always present, except perhaps at the very commencement of the affection; for many observers have noted that even in cases in which albumen is sometimes to be detected, it may at other times be absent from the urine; and Cohnheim found lardaceous disease after death in several cases in which albuminuria was said to have been altogether wanting.

The quantity of albumen varies. There may at first be only a little; but the rule is that the quantity is large, as large as in tubal nephritis.

Blood is very rarely present, even in small amount. The statement

once made by Senator that the presence of paraglobulin in large quantities is characteristic of this rather than of other forms of Bright's disease, has not been confirmed since the introduction of Hammarsten's method of separating paraglobulin from serum-albumen.

In some exceptional cases there may be found in the urine glistening epithelial cells, which turn reddish brown with iodine; and it has been supposed that their presence is conclusive as to the nature of the renal affection; but the writer has frequently observed this reaction with iodine, not only without any symptoms of lardaceous disease of the kidney, but in spheroidal and transitional epithelium of cystic origin.

The urine is, in the least complicated case, pale and transparent, and throws down no deposit, or at most a few hyaline casts and epithelial cells. When, however, there is much nephritis, the urine may be high-coloured, and may give an abundant precipitate containing lithates, as well as numerous hyaline and granular casts, to which epithelial and fatty cylinders may be added (see Dickinson's plate vii, fig. 2).

The other symptoms which accompany lardaceous disease of the kidneys are variable and uncertain. *Pallor* is the most constant, and is often extreme; but cachexia and wasting may sometimes be absent. One of our cases at Guy's Hospital was that of a man, aged thirty-six, who died soon after being admitted with a fractured spine, from a fall while he was carrying a sack of barley on his shoulders; lardaceous changes were found in the liver, the spleen, and the adrenals, as well as in the kidneys, which weighed sixteen ounces.

An important and often fatal complication of this form of Bright's disease is uncontrollable diarrhoea, due to lardaceous degeneration of the mesenteric arterioles. The ulcerative colitis common in cases of renal cirrhosis is seldom seen.

Dropsy is sometimes wanting as a symptom of lardaceous disease of the kidney, especially when there is diarrhoea from a coincident affection of the intestinal mucous membrane. But, as a rule, anasarca is present, and frequently in an extreme degree, affecting the face, the arms, and the whole body, exactly as in cases of the "large white kidney," for which those really lardaceous have often been mistaken, both at the bedside and after death.

The left ventricle is seldom found hypertrophied. Of more than sixty cases observed at Guy's Hospital, there were only four in which the heart weighed from eleven to thirteen ounces; and in two of these the kidneys also were enlarged, weighing fourteen and eighteen ounces respectively. The absence of cardiac hypertrophy was ascribed by Dr Fagge to the cachectic and anæmic condition of the majority of those suffering from lardaceous degeneration; but the pulse in these cases shows none of the characters of high arterial tension which cause the cardiac change. Primary cirrhosis of the kidneys is not infrequently complicated by lardaceous degeneration, and in such cases cardiac hypertrophy and dilatation, with œdema of the legs, may follow.

Retinitis and uræmia are seldom observed in cases of lardaceous affection of the kidney; but in three cases in Guy's Hospital death was preceded by eclampsia.

One patient, who had been in hospital nine months previously with dropsy, was readmitted four days before death in a state of collapse, with cold and blue extremities, the result of severe diarrhoea and vomiting. In several instances acute pericarditis was the immediate cause of death.

Diagnosis.—Neither the characters of the urine, nor any other symptoms, can be relied on to distinguish lardaceous affection of the kidneys from the other forms of Bright's disease. What in practice decides our diagnosis is either that we find enlargement of the liver, or intractable diarrhoea without products of ulceration—or else that the case is one of prolonged suppuration or of tertiary syphilis.

The liver is always enlarged when the seat of this disease; but infiltration of the spleen or of its Malpighian follicles (the "sago-spleen") often does not increase its bulk. Knowledge of the causes of the affection is therefore all-important.

Prevention and treatment.—In order to prevent lardaceous affections, it is clearly most important that all sources of suppuration should be removed as speedily as possible; and in cases of syphilis it is desirable to continue treatment long after the symptoms have been removed.

Experiments on animals have shown that after suppuration has been established and kept up (by the *Staphylococcus pyogenes aureus*), lardaceous disease follows as in man; but, what is more important, they have also shown that by stopping the suppurative process, the lardaceous change has disappeared. (See Lubarsch in 'Virchow's Archiv,' 1897, ii, 471, quoted by Dr M. Murray in the 'Encycl. Med.')

It might be thought that the clinical diagnosis of lardaceous degeneration from other forms of Bright's disease is not of practical importance, since it is to the presence of diffuse nephritis that most of the symptoms are really due. But as regards both prognosis and treatment it is important not to overlook the presence of lardaceous complication; for when general dropsy sets in, the downward progress of cases with lardaceous kidneys is more rapid than in other forms of Bright's disease. Again, it not infrequently happens that albumen is detected in the urine of syphilitic patients several years before other symptoms of renal disease appear; and there is evidence that such cases, even when dropsy occurs, may end in recovery, and the urine gradually resume its normal characters.

In all cases of Bright's disease, where there is reason to suspect previous lues, we should at once put the patient on a course of iodide of potassium.

Two instances of this are recorded by Dr Dickinson in vol. xxx of the Pathological Society's 'Transactions.' One was that of a patient who contracted syphilis in 1861, and who in 1874 began to suffer severely from periosteal nodes; soon afterwards his legs became cedematous, and his urine was loaded with albumen. Under specific treatment, assisted by a residence on the Riviera during four winters, the disease slowly subsided, and by June, 1879, the urine was normal and the patient apparently in perfect health.

The other case was that of a young man with secondary syphilis, caries, albuminuria, and dropsy. After two years' treatment, chiefly with iodides of potassium and of iron, the urine became normal, and all his symptoms disappeared.

What changes take place in the kidneys when recovery occurs we do not know. It seems not impossible that the glomeruli and other vessels that were lardaceous may return to a normal condition, if the albuminuria has only lasted a short time. But in protracted cases it is far more likely that the affected parts of the kidney shrink and undergo atrophy, and that parts which had escaped the disease supply their place, with possibly some compensatory hypertrophy.

III. CIRRHOSIS OF THE KIDNEY.*—The morbid process which produces the

* *Synonyms.*—Chronic interstitial nephritis—Red granular atrophy—Granular degene-

“small red kidney” is the most frequent, the most insidious, and in some respects the most important of all. The general characters of this form of morbus Brightii have been already defined (p. 605).

Those cases of renal atrophy which are secondary to tubal nephritis should be distinguished from those which are the results of a primary interstitial nephritis. One proof of the reality of this distinction is afforded by the difference in the appearance of granular kidneys at different periods of life. In autopsies on children, and even in adults under the age of twenty, twenty-five, or perhaps thirty years, we do not meet with the red or brown contracted and granular kidneys which are so frequent in middle-aged and in old people. The kidneys in young subjects are full of opaque whitish-yellow spots or patches. It is true, doubtless, that these become fewer and less conspicuous as the process goes on; so that it is quite possible that in some exceptional instances they may disappear, and leave the organ in a condition like that which is usually met with at a later age. But even if the means of distinguishing them should thus sometimes fail, it would not diminish the obvious differences between the two conditions in the majority of cases.

On the other hand, in older patients the criterion afforded by the appearance of the kidneys not infrequently ceases to be applicable. The reason is that when a part of the renal substance has been destroyed by cirrhosis the remainder is apt to become affected with the parenchymatous change. Thus the records of *post-mortem* examinations at Guy's Hospital furnish no fewer than thirteen cases in which the kidneys of patients who had been the subject of gout, and whose joints contained urate of soda, were wasted and granular, but at the same time whitish yellow in colour.

In 1873, a woman aged forty-four, a gin-drinker, died after an illness of seven weeks' duration, which she said began one day with pains in the loins while she was working in a cold washhouse. On the following day the face was swollen; vomiting then set in, and afterwards the legs and the abdomen became swollen. The urine was albuminous, of sp. gr. 1002. After death the kidneys were found mottled with yellow; but they weighed only eight ounces, they were granular on the surface, and their cortex was much narrowed. The arteries were rigid. The heart weighed twelve ounces, the left ventricle being hypertrophied.

No doubt the pathologist, if he has no clinical history to guide him, is unable to distinguish such “mixed” cases as this from cases of primary parenchymatous nephritis in its most advanced or contracted stage; though even without any history he may be guided to the right conclusion if he discovers the gouty origin of the disease from the presence of urate of soda in the joints. But in this country the “mixed” cases after all form only a minority compared with those in which the kidneys are purely cirrhotic.

Ætiology.—What proportion of cases of renal cirrhosis is associated with *gouty* deposits it is impossible to say, for the joints are still too often forgotten in an autopsy; and urate of soda may be found in the toe-joints with no history of a gouty attack. At the meeting of the International Congress, in 1881, Dr Ord stated the results of autopsies made at St Thomas's Hospital, on twenty-four cases of granular disease of the kidneys; in sixteen gouty deposits were present in the joints, in eight they were absent.

ration, producing the small red kidney of Bright—Chronic gouty nephritis—The gouty kidney of Todd—Desquamative nephritis of Johnson—Contracted kidney—Raspberry kidney—Cirrhosis of the kidney.—*Fr.* Maladie de Bright, forme chronique aux reins ratatinés.—*Germ.* Schrumpfniere (the contracted kidney).

It is not unlikely that the rarity of gout in Germany brings with it a corresponding rarity of renal cirrhosis, and this may be the chief reason why some German writers fail to recognise the latter affection as distinct from other forms of Bright's disease. They speak of a "senile atrophy," which they regard as devoid of clinical importance; but few pathologists in this country would admit that the kidneys are naturally liable to any purely senile change, except slight shrinking corresponding with the loss of weight in all the tissues, that comes with advancing years. At the same time it is true that granular degeneration of the kidneys is associated with vascular and other changes which belong to the later periods of life, and that in the exceptional cases when it occurs under thirty years of age there are often other indications of premature old age.

Next to gout, a well ascertained cause of cirrhosis of the kidneys is chronic poisoning by *lead*. The disease is common in painters, in printers, and in type-founders. But lead-poisoning also produces gout, and therefore it is difficult to prove the extent of its direct effect in leading to renal cirrhosis.

Indulgence in *alcohol*, apart from its tendency to set up gout, is probably a common cause of renal cirrhosis. Although Dr Dickinson gives reasons for doubting this conclusion, the general impression is strongly in its favour.* The reports of autopsies at Guy's Hospital do not bear out the statement sometimes made that the renal affection is very frequently found in association with cirrhosis of the liver. Dr Pitt found granular kidneys in only one fourth of the cases of cirrhosis of the liver which he examined there,† and Dr Hadden in only one sixth of those at St Thomas's Hospital. Moreover, if drink produces gout, so does lead, and therefore gouty nephritis may be mistaken for directly alcoholic nephritis. Perhaps one of the best arguments in favour of intemperance leading to the more chronic form of Bright's disease is that it has been so generally assumed without argument by men of wide clinical experience, including Bright himself. There is no belief, as there is no evidence, that syphilis is a cause of interstitial nephritis.

Renal cirrhosis often accompanies atheroma of the larger arteries, and *endarteritis obliterans* of the smaller (arterio-sclerosis), as well as hypertrophy of the left ventricle and apoplexy (*cf. supra*, p. 625).

Chronic interstitial nephritis, secondary to obstruction of the urine, will be treated separately as *consecutive* cirrhosis (p. 662).

Age and sex.—The cirrhotic form of Bright's disease is almost unknown in youth and early adult life. Out of 121 cases at Guy's Hospital, death occurred in eighteen between 31 and 40 years, in thirty-nine between 41 and 50, in thirty-six between 51 and 60, in twenty-four between 61 and 70, in four between 71 and 80. Below the age of 30, typical instances are very rarely met with, but several of the patients between 35 and 40 had suffered from gout, and renal cirrhosis appeared in its characteristic form. These figures correspond pretty closely with those given by Dr Dickinson. They differ, of course, from those of Wagner, who mixes together all forms of "contracted kidney." Eichhorst, however, gives 40 to 60 as the usual age in Germany. Exceptional cases of renal cirrhosis may be met with in chil-

* See Sir Wm. Roberts's criticism on Dr Dickinson's statistics ('Brit. Med. Journ.,' November 4th, 1871).

† 'Guy's Hosp. Rep.,' 3rd series, vol. xx, p. 196.

dren under puberty. The writer once had a fatal case in a child of only $2\frac{1}{2}$ years old.

A little girl in her third year was admitted to Miriam Ward, December 14th, 1893, with signs of slight bronchitis, and a frequent pulse which was not hard. Very little urine was passed, high-coloured and full of albumen, but without blood or casts. There was no retinitis, nor retinal hæmorrhage, nor optic neuritis. She died a few hours after admission. She had always been weakly, but beyond a cough for a fortnight before her death, no serious symptoms had appeared before admission.

After death the bronchial tubes contained pus and mucus; there was no pleurisy or pneumonia. The left ventricle was hypertrophied, the peri- and endo-cardium healthy, as were the brain, liver, and other organs, except the kidneys. These were both shrunk, with adherent tunic, granular surface, and wasted cortex, "typical small red raspberry kidneys."

The proportion of males to females in the Guy's Hospital cases was almost exactly as two to one; and in Dr Dickinson's cases it was 165 to 85.

*Anatomy.**—In its earlier stages, renal cirrhosis is by no means a conspicuous morbid change. The kidneys may be of natural size and colour; and beyond the fact that the normal arrangement of the pyramids of Ferrein is no longer visible upon the cut surface of the cortex, and that the capsule is thick and too adherent, there may be nothing to suggest disease, so that one is sometimes surprised to find with the microscope to what an extent the cortex has been destroyed.

In advanced stages the case is very different. The capsule is greatly thickened, and cannot be stripped off without the cortex tearing and coming away with it. The surface of the kidney is covered with little projections or granulations, which, in uncomplicated cases, are of a red or reddish-grey colour. Hence the phrase "raspberry kidney" applied to this condition. They no doubt consist (like the granules of a cirrhotic liver) of portions of the cortex which are less altered and wasted than the rest. Kelsch and Charcot maintained that they correspond in position with the summits of the "medullary rays" or columns of straight tubes that traverse the cortex, the depressions between them answering to the intervening tracts of convoluted tubes; but Weigert and Wagner dispute this statement. The reduction in the thickness of the superficial parts of the cortex is often so extreme, that the bases of the medullary pyramids are separated from the surface of the kidney by a layer of tissue not the tenth of an inch thick. The inter-pyramidal portions of the cortex are not generally wasted to the same degree; perhaps they undergo a compensatory hypertrophy.

The weight of the two kidneys is often reduced from eight or nine ounces to five, four, three, and sometimes even to two and a half ounces. The smallest kidneys we have seen at Guy's Hospital weighed together thirty grains short of an ounce and a half. Yet neither the diminution in size, nor the loss of weight, gives an adequate idea of the destruction of the renal cortex; for the pelvis is proportionately wider than in the healthy organ, and is filled with fat, over which the remaining renal tissue is, in extreme cases, spread out as a thin shell.

Histologically, the tubular structure of the renal cortex is replaced by connective tissue in various stages of development. At first there is merely a "small-cell infiltration," in scattered foci, especially round the capsules of the glomeruli, but with prolongations between the tubes adjacent. Gradually the beautiful pattern of the renal cortex is disturbed. The

* The following account is based upon notes of considerably more than 100 examples of primary and uncomplicated renal cirrhosis, inspected by Dr Fagge, and entered by him in the volumes of *post-mortem* records at Guy's Hospital.

tubules are squeezed here and dilated there; they lose their epithelium and become obliterated, or converted into minute cysts. The glomeruli are at first less affected than the rest of the vessels, and the Malpighian capsules less than the tubules, so that they may sometimes seem little altered, though crowded together by the atrophy of the tissue between. The intertubular blood-vessels and lymphatics, with remains of shrunken tubes, form the elements of the hard and almost bloodless new tissue. Ultimately the exuded leucocytes undergo conversion into new tissue, in which there are generally very few blood-vessels, though these are sometimes remarkably wide. Relics of tubes are usually to be seen embedded in this tissue, and are filled with altered epithelium or with hyaline casts. There are also areas in which the tubes are comparatively unaltered, except that they are too wide and patent, with flattened epithelium, and here the glomeruli are often dilated.

Sometimes hardly a trace of renal structure can be detected over extensive tracts. The glomeruli and Malpighian capsules are destroyed; or the capsules become enlarged and thickened by the formation of concentric layers, while the tufts degenerate into a structureless material, containing only a few scattered nuclei. By the shrinking of the intervening tissues these "glassy globes" or cysts are often drawn close together, so that a large number of them are seen in the same microscopic field. Ultimately nothing may be left but round translucent masses, of which the diameter is not more than one half or one third of that of the normal glomerulus.*

It is held by some German pathologists that degeneration of the glomeruli is the primary change in the tissue of the kidney in this disease, and that it again is dependent upon an *arteritis obliterans* affecting the smaller branches of the renal artery in the cortex, and the afferent vessels of the tufts. Thoma has shown ('Virchow's Archiv,' 1877) that, when a glomerulus becomes obsolete, its afferent artery may remain pervious, and may open straight into the efferent vessel or into the Bowman's plexus. The thickening of the larger arterial branches is undoubtedly one of the most obvious morbid appearances in a red granular kidney; they stand out upon the cut surface of the organ with patent mouths, like so many little quills. But it is very doubtful whether the arterial lesion is the starting-point of the whole process. The theory just stated approaches very near to Gull's and Sutton's of primary arterio-capillary fibrosis, of which the granular kidneys are only one symptom. It seems, however, more probable that the two processes of renal cirrhosis and arterial disease are concomitant effects of some primary condition.

Johnson held that the disease begins in the convoluted tubules, which shed their epithelium (instead of retaining it, as in the large white kidney), and accordingly proposed the name of "desquamative nephritis" for granular degeneration or cirrhosis of the kidneys ('Med.-Chir. Trans.,' vol. xxx); but this view has been now generally abandoned.

A study of the early stages of the disease shows that it begins as sub-acute intertubular inflammation. In response to repeated but slight irritation (from lead or alcohol or uric acid or other unknown cause), leucocytes are exuded between the tubules, and gradually form a granulation-tissue. This contracts, squeezing the tubules, destroying their epithelium,

* For instructive figures illustrating renal cirrhosis see Dr Dickinson's 5th plate, Dr Conts's fig. 274, and figs. 65 and 66 in Dr Woodhead's 'Practical Pathology.' Also Dr Saundby's and Dr Greenfield's plates in the 'Path. Trans.' for 1880, with their comments.

and thus reproducing Virchow's type of chronic interstitial inflammation with cicatrization, fibrosis, and contraction, as we have seen it exemplified in sclerosis of the cord, fibroid pneumonia, and cirrhosis of the liver.

In many cases a contracted kidney is pervaded by innumerable minute *cysts*; cases of this kind have been separately described as "micro-cystic" kidneys. The great majority of the cysts are invisible to the naked eye, but some may be of all sizes up to that of a pea or even larger. They may make up by far the larger part of a microscopical section. Their contents are often a yellowish-brown jelly-like substance, which can be turned out of the larger cysts as a solid mass. With regard to their nature there was at one time some controversy. It was thought that they might be overgrown epithelial cells, or dilated Malpighian capsules. But the former mode of origin seems impossible (though it was once advocated by Sir John Simon), and the latter only accounts for a fraction of the numberless cysts that are often present. Probably they consist of portions of tubes that have become cut off, and have assumed a spherical shape as "retention-cysts." Not only are they sometimes arranged in rows, like beads in a necklace, but intermediate forms are often met with—cylindrical cavities with constrictions here and there in their course.

Another appearance that is observed in cirrhotic kidneys is due to the deposition of *lithic acid* or of its salts in the renal tissues. It consists in the presence of minute whitish-yellow grains scattered through the cortex, or arranged in lines in the pyramids. Some of these deposits are amorphous, some are made up of bundles of acicular crystals. They appear to lie partly between the tubes, partly in their interior. It has been supposed that such deposits are the results of gout, and that, acting as foreign bodies, they produce albuminuria and set up the renal disease in association with which they are found after death. But they are found in cases in which there is no other evidence of gout, and they are frequently met with in Germany, where gout is rare.

Symptoms: the urine.—In the slighter degrees, or in the earlier stages, of cirrhosis of the kidneys, the excretion of urine may be normal. Sir Grainger Stewart, for instance, relates a case of a man of sixty-five, who died of phthisis, and who passed forty ounces daily, the specific gravity being 1020; yet the kidneys were found to be granular after his death. But when the disease is advanced the quantity of urine is increased, and its specific gravity is low.

The *quantity* amounts to a total of 70 to 200 ounces or more daily; in a case recorded by Bartels it was measured on a single occasion from 8 p.m. to 8 a.m., and was found during that period to reach 210 ounces. It is commonly secreted more abundantly at night than in the day. Bartels had the urine of one of his patients measured for twenty-six days, that passed from 10 p.m. to 7 a.m. being separated from that passed from 7 a.m. to 10 p.m.; during the nine hours of night the average quantity was found to be seventy-seven ounces; during the fifteen hours of day only forty-eight ounces.

The specific gravity varies from 1004 to 1010. The urine is faintly acid, pale and clear, depositing no sediment, or only a very slight cloud.

It usually contains only a small quantity of *albumen*, less than .5 per cent.; the whole amount of albumen in the twenty-four hours is said by Wagner to be not more than from twenty to ninety grains. Indeed, unless care is taken in applying the tests, the presence of albumen may be alto-

gether overlooked ; after pouring the urine upon nitric acid one must allow two or three minutes for the opalescent zone to appear, and, after adding a drop of acetic acid, heat must be gradually applied until the boiling point is reached.

It may happen that albumen is entirely absent for days together ; or the night may be constantly free from it. That the urine is ever ex-albuminous throughout the whole course of the disease is extremely improbable. When albumen is more or less constantly present, it is often more abundant in the day than in the night ; and in the urine passed after meals than in the *urina sanguinis* of the early morning, which the patient is often told to bring.

Casts are often entirely absent ; when any are found they are commonly hyaline and narrow, but sometimes opaque and granular.

From time to time the urine may become scanty and the albumen in it abundant or even tinged with blood. This might be due to the supervention of intercurrent nephritis, as bronchitis may occur in the course of chronic phthisis.

Another source of the hæmaturia which not infrequently appears during the course of the most chronic cases of renal cirrhosis, is probably the degenerated renal arterioles, in fact hæmorrhage of the same kind as the epistaxis and apoplexy of Bright's disease (p. 630).

The amount of *urea* secreted daily seems, according to recent observations, to be not so deficient as used to be supposed. It is true that the proportion in a given quantity of urine is generally not more than 1 or 2 per cent., but so abundant is the urine that Bartels found in four of his cases that the daily average output of urea ranged from 296 to 522 grains.

Even when the quantity of urine passed is not noticed to be excessive, the patient may be obliged to get up out of bed three or four times every night to micturate. It is often the first symptom complained of, but one has sometimes to ask whether it is so. If nocturnal irritability of the bladder has existed for a considerable time, it may be fairly inferred that the renal affection is of at least as long standing.

Patients who have suffered from gout and are accustomed to pass high-coloured urine, becoming thick with urates, often flatter themselves that the kidneys are working better than before, when the secretion becomes pale and clear as the result of developing renal cirrhosis.

Again, when the flow is much increased, with consequent thirst, patients sometimes take alarm and seek advice for diabetes ; and if the urine is tested and no sugar is found, the diagnosis of *diabetes insipidus* may be wrongly given.

Other symptoms.—The character of the *pulse*, and the state of the *heart*, are important elements in the diagnosis of cirrhosis of the kidney, especially in cases where albumen is not constantly found in the urine. The hypertrophy of the left ventricle, the thickening of the smaller arteries and the high tension of the pulse, common in some degree to most forms of Bright's disease, reach their fullest development in the typical cases of chronic interstitial nephritis. Frequently, however, towards the end of a case, when cardiac dilatation takes the place of hypertrophy, the "renal" character of the pulse disappears, and it becomes soft and irregular like that of primary mitral disease.

Pleurisy and pericarditis are both common, and after paracentesis the peritoneum may inflame. Dropsy is, like albuminuria and anæmia, less

striking than in the tubal form of Bright's disease, and when it supervenes is most often due to cardiac dilatation. Retinitis and retinal hæmorrhage are not uncommon, and uræmic symptoms are very frequent.

Clinical forms.—Chronic renal cirrhosis presents itself in several different ways, which are all important to recognise:

1. Some patients only complain of *weakness and exhaustion*; and the most conspicuous physical change discoverable in them is that they are anæmic and wasted, with flabby muscles. A man, aged fifty-nine, with what proved to be chronic Bright's disease, was so pallid that his disease was set down during life as idiopathic ("pernicious") anæmia.

2. In certain cases the chief symptoms are *gastric or intestinal*; uncontrollable vomiting or diarrhœa, or both together. It is especially in such cases that the breath is sometimes horribly foetid, as mentioned by Bartels. Vomiting is sometimes of a uræmic character, and of serious import. Diarrhœa towards the end of a chronic case is often uncontrollable, and depends upon intestinal catarrh with œdema of the mucous membrane. Intestinal inflammation is more common in this than in the more acute form of Bright's disease; and occasionally ulcerative colitis is the immediate cause of death.

These ulcers are not very deep, but numerous, mostly oval and transverse, sometimes large and spreading. The absence of sloughs distinguishes them from Enteric ulcers, absence of thickening, and serous implication from tuberculous ulcers, and freedom of the mesenteric glands from both. Lastly, they are not so large and numerous, or sloughing or hæmorrhagic, as the ulcers of Dysentery.

Often catarrh is present without ulceration, and sets up an uncontrollable and fatal diarrhœa.

3. In many instances the patient comes under medical observation owing to the occurrence of *acute pneumonia, pleurisy, or pericarditis*. Peritonitis is scarcely ever seen as a complication of renal cirrhosis. Acute pleurisy is often rapidly fatal, and pericarditis is particularly dangerous; but occasionally it seems to cause little disturbance of the general health, and subsides after a while. Even when it is still active at the time of the patient's death the amount of lymph exuded is often but small, so that it is not always clear whether it has brought about the fatal issue. Purulent pericarditis is, in our experience, very rare.

Inflammatory œdema of the lungs is another fatal complication in cases of renal cirrhosis, though less frequent than in those of tubal nephritis.

4. *Cerebral hæmorrhage* is often the cause of death, and in many cases it is not preceded by any serious symptoms that are recognised. Sometimes the occurrence of hemiplegia is due, not to extravasation of blood, but to local softening of the brain, as the result of arterial atheroma.

5. *Uræmia* is probably less frequent than in tubal nephritis. Among 120 cases at Guy's Hospital only fifteen or sixteen ended thus. Three of them occurred in patients between thirty-one and forty, six between forty-one and fifty, two between fifty-one and sixty, two between sixty-one and seventy, two between seventy-one and eighty. Dr Fagge thought that the common belief that this form of Bright's disease is the one in which uræmia is most apt to occur, was due to the advanced stages of tubal nephritis being often mistaken for it.

6. Sometimes the patient comes under observation with *renal dropsy*, having the characters described above (p. 641). Such cases are, as a rule,

examples of the supervention of parenchymatous nephritis upon antecedent cirrhosis, and at the autopsy the renal affection is found to be of the "mixed" kind. But, unless the patient has been under observation previously, it may be impossible to diagnose the presence of any but recent changes in the kidneys; for the state of the urine is indistinguishable from that of primary tubal nephritis. In all cases of renal dropsy occurring after middle age it is very important, with a view to prognosis, that the high probability of there being chronic as well as acute lesions should be borne in mind.

7. The last and perhaps the most important clinical group of cases of cirrhosis of the kidneys is that in which the main symptoms are *cardiac*, the patient coming under observation with dyspnœa, palpitation, and dropsy of the dependent parts of the body. Dyspnœa, especially, is often an early and important symptom. Dr Mahomed ('Guy's Hosp. Rep.,' 1879) stated that such "cardiac" cases make up 17 per cent. of all those in which granular atrophy of the kidneys is found after death, and Dr Fagge's analysis of a somewhat larger number of cases yielded an even higher figure. After death the left ventricle is usually found dilated as well as hypertrophied. Apart from this dilatation, degeneration of the muscular tissue of the heart is probably sometimes the cause of its failure; for its substance is often soft and flabby, and under the microscope the fibres may be seen to be granular, and to break into short fragments when an attempt is made to separate them with needles. A fibrous change in the papillary muscles of the mitral valve is also sometimes seen; they taper gradually into the tendinous cords, instead of appearing as blunt fleshy columns.

A systolic apex-murmur, having some of the characters of the mitral regurgitant bruit, but not usually audible in the axilla, may be heard in many cases. Dilatation of the left ventricle, without any lesion of the mitral valve, is probably the cause of this murmur; but it is not often that positive proof of regurgitation is afforded at the autopsy by the presence of "ripple lines" on the posterior wall of the left auricle, as in a case recorded by Dr Mahomed ('Guy's Hosp. Rep.,' 1879). Sometimes the edge of the anterior flap of the mitral valve is thickened, and can be readily bent inwards. Endocarditis secondary to Bright's disease is very rarely acute or ulcerative; but thickening, with contraction of the mitral curtains, is not uncommon.

At one time cases of cardiac failure secondary to cirrhosis of the kidneys were regarded during life as examples of morbus cordis, but frequently the diagnosis of mitral incompetence has not been sustained after death; though the left ventricle was hypertrophied and dilated, the mitral valve was healthy. Sometimes, however, one may be fairly in doubt as to the correct interpretation of the morbid appearances. The kidneys, perhaps, are of nearly average size, although hard, red, and glistening on section. Even the microscope, while revealing a certain amount of fibrous change, with degeneration of some glomeruli, may leave one in doubt whether this is more than an accidental feature of the case, especially if the patient was advanced in years. Chronic venous congestion of the kidneys from valvular disease may end in tubal nephritis, and it is possible that, as the late Dr Mahomed believed, mitral stenosis may be more than an accidental sequence of cirrhosis of the kidneys.

If when the organs are before us it is occasionally difficult to decide whether the heart or the kidneys were first affected, it is no wonder that the

clinical diagnosis of the cardiac dropsy secondary to renal disease from that which depends on primary disease of the heart is sometimes more difficult. The state of the urine does not always help us. In either case it may be scanty, high-coloured, and loaded with lithates, and may contain more or less albumen as well as casts. Wagner says, however, that in cases of renal cirrhosis, even when the urine is scanty, its specific gravity is seldom above 1012; and he cites an observation of Traube's that in extreme instances it may remain pale and of low specific gravity even when there is great obstruction to the venous circulation or (on the other hand) when some febrile disease, such as acute pneumonia, develops itself. In two of our cases, however, at Guy's Hospital, the specific gravity of the urine ranged from 1020 to 1025, although at the autopsy the kidneys weighed only seven and a half or eight ounces, and were very granular; and in a foot-note to Dr Southey's translation of Bartels' work on 'Bright's Disease' a case is mentioned in which in spite of very great wasting of the kidneys the urine had a specific gravity of 1028, and deposited lithates on cooling. Indeed, whatever may be the rule as to the more advanced stages of the affection, so long as the kidneys are not much shrunk, although the microscope may afterwards show that they have undergone extensive fibroid changes, they commonly yield a high-coloured secretion, of great density, during the time that cardiac dropsy is present.

In all such cases it is to the pulse that we must look for guidance in our diagnosis. But a visible and tortuous condition of the temporal arteries is often due to local senile changes in the coats of these vessels. Moreover, in cases in which there is much cyanosis from emphysema, it is not safe to rely even upon a hard and persistent pulse at the wrist as proof of renal disease. Probably the occurrence of high arterial tension in such circumstances is comparable with the rise of blood-pressure in the arteries that is observed at a certain period of asphyxia in experiments on animals.

Recognition, course and events.—Renal cirrhosis is an insidious disease, obscure in its origin and extremely slow in its course. The only exceptions are cases which develop as the result of acute scarlatinal nephritis, and probably some of these are examples of secondary atrophy—"the small white kidney" described above (p. 640).

We suspect the presence of granular degeneration when a patient complains of "rheumatic" pains, lumbago, and muscular weakness, of having to rise in the night to pass water, of troublesome headache or of slight nausea while dressing in the morning, particularly when brushing his teeth. The suspicion is strengthened if he has had gout, or is exposed to lead-poisoning, or is intemperate in liquor. A pale, sallow face, a hard pulse, a weak or muffled first and a ringing or reduplicated second sound, with a little œdema of the eyes in the morning and of the ankles at night, make the diagnosis almost certain, before we examine the urine; and if this is abundant, pale, and of low density we may be sure that we are right, even if at first neither albumen nor casts are to be found.

Such patients, judiciously treated, often go on for many years with little or no aggravation of their symptoms, and it may be very long before severe cardiac symptoms come on, or an attack of uræmia or of apoplexy.

But they are always in danger. Exposure to cold is likely to bring on bronchitis or pleurisy, or perhaps a subacute attack of tubal nephritis with general dropsy. If they are attacked by pneumonia, the prognosis is serious; and if an injury befalls them or a surgical operation is proposed,

the case assumes unusual gravity. If they escape these accidents, and are not cut short by intercurrent diseases, the hypertrophied heart will probably sooner or later dilate, and they will slowly die, with cardiac symptoms.

Nevertheless, with care and good management, the renal degeneration often seems to cease to advance, and we may see patients live on with chronic Bright's disease for ten or twenty years, and even to the full term of life.

Two remarkable cases have been recorded by Dr Francis Hawkins in the 'Clinical Transactions' for 1893 (p. 216), one of a patient of Sir William Gull, who was living twenty-five years after albumen was first found; the other a patient of Dr Bright himself in 1849, who lived for forty-three years, and died of cerebral hæmorrhage at 88 in the year 1892. In both these cases albuminuria was found present at various times for twenty years consecutively.

Consecutive renal cirrhosis.—Under this name may be appropriately described an affection of the kidneys which is proved by its histological characters to be a form of Bright's disease, but which is secondary to morbid changes in the renal pelvis, or in some part of the lower urinary passages.

The mechanical effects of pressure on the renal pelvis have long been recognised in the flattened pyramids and thinned out cortex of hydro-nephrosis.

Consecutive interstitial nephritis also may occur as the result of urethral stricture, prolapse of the uterus, or other cause of urinary obstruction. It is often cut short in its course by the supervention of rapidly fatal suppuration of the kidneys ("ascending nephritis"); but it is quite capable of destroying life without any aid. An excellent account of this form of renal cirrhosis was given by the late Mr Marcus Beck in the fifth volume of 'Reynolds' System of Medicine' (1879).

Anatomy.—In these cases the appearance of the kidneys varies. Occasionally they are found swollen, mottled, with red and white patches, and of a soft consistence; but, as a rule, they are very tough and hard, of a dull white or opaque waxy yellow colour. The surface is generally smooth. But sometimes there is as marked granulation as in ordinary cirrhosis of the kidneys. The capsule is thickened, and very adherent to the kidney and indurated adipose tissue around.

In 1879, in an autopsy on a boy aged twelve, we found an hypertrophied and contracted bladder with great dilatation of the ureter and of the renal pelvis on each side; the two kidneys together weighed less than two ounces, and the cortex was in most places shrunk to a thin red line; but, contrasting with the rest, there were some raised, soft, pale yellow, rounded nodules.

Even when the seat of obstruction is in the bladder, or in the urethra, the two kidneys do not always suffer equally; and when it is in the renal pelvis on one side only, that kidney will suffer alone. Thus, in 1876, in the case of a woman aged forty-seven, who for twelve years had suffered from calculous pyelitis, we found the cortex of the affected kidney reduced to a thin shell of white fibrous material; the other one weighed nineteen ounces, and had undergone hypertrophy, though it was also affected with recent lardaceous and other changes. She had died from uræmic coma. In another case a calculus lay in one calyx of a kidney, and the corresponding part of the cortex was narrow while the rest was healthy.

Even when consecutive cirrhosis affects the whole of both kidneys, it often happens that the change is far more advanced in some parts than in others, and thus deep puckered cicatrices are produced. This is one of the points of difference between the consecutive and the primary or "medical" form of renal cirrhosis, where the difference between the two kidneys is seldom marked. The former process affects the kidneys anatomically, the latter physiologically.

The morbid process, as described by Beck, is identical in its histology with that of Bright's disease in which the interstitial tissue is mainly affected. There is first an accumulation of immense numbers of leucocytes in different parts of the cortex, chiefly round the Malpighian capsules, but also between the tubes. The tubal epithelium is but little altered; but sometimes small extravasations of blood occur within the tubes, as well as into the intertubular tissue. The glomeruli become crowded with nuclei, and gradually shrivel into transparent bodies surrounded by thickened capsules. Ultimately the small-cell growth develops into fibrous tissue.

Consecutive Bright's disease occurs in various surgical affections of the urinary organs, such as *stricture*, disease of the prostate, villous disease of the bladder, stone in the bladder. But it is also seen by physicians as a result of any of those diseases that will be enumerated in the next chapter as causes of *hydronephrosis*. It occasionally affects one kidney as a consequence of pyelitis; and in cases of tuberculous disease it has sometimes an important part in completing the work of destruction. Lastly, *prolapsed uteri* and other affections of the female genitalia, which drag upon the ureters or obstruct the urethra, are important causes of consecutive cirrhosis affecting both kidneys.

The clinical *diagnosis* of this form of renal disease is beset with peculiar difficulties. The urine is often so altered as the consequence of cystitis or pyelitis that its characters lend scarcely any assistance. When it is free from blood and pus, it may perhaps contain neither albumen nor casts, though sometimes it is albuminous, and occasionally a few hyaline casts may be discovered in it. It is generally rather excessive in quantity, and of low specific gravity. But no conclusion must be drawn from the density of a single specimen, especially soon after surgical interference with the urinary passages. Mr Beck mentions a case of lithotripsy in which the specific gravity of the first sample of urine passed was only 1003, whereas that of the whole twenty-four hours' urine was 1018. The quantity of urea excreted may be little, if at all, below normal; certainly it is often as great as can be expected if one takes into account the small amount of food which the patient can eat.

Nor is the presence of consecutive Bright's disease clearly indicated by any marked general symptoms. In subacute cases there is often an evening rise of temperature to 100° or 101°, whereas the morning temperature may be constantly normal or even subnormal. The patient feels weak and languid, and steadily loses flesh. The tongue may be foul, with a thick white or dirty fur, and may in bad cases be dry and brown. There may be much thirst, with little appetite for food, and more or less nausea. The skin is usually moist and clammy. There is neither tenderness nor pain in the loins. The patient's mental state is often placid; and he may be drowsy, like a person slightly under the influence of opium. Death sometimes occurs by exhaustion, sometimes by the supervention of some acute disease, sometimes by uræmic coma. But if the primary disease can be relieved by

surgical treatment, it is surprising how all the symptoms may subside that had appeared to indicate grave renal mischief, so that after all one may be left uncertain whether consecutive cirrhosis really existed. Unfortunately, restoration to a fair state of health is no proof that the kidneys have not been damaged; and in the most chronic cases of all, in which the organs become yellowish white and tough, there are often for a long time no symptoms at all: the patient does not waste, he is not anæmic, and he eats, drinks, and sleeps as usual.

It has been doubted whether this form of Bright's disease is liable, like the others, to produce cardio-vascular changes. Beck stated that the heart does not become enlarged, but that the renal arterioles show hypertrophy of their muscular coats. Wagner, however, found the heart was hypertrophied in each of five cases that came under his observation; and in one there was a well-marked albuminuric retinitis. In two cases secondary to stricture of the urethra, Dr Fagge found the heart weigh sixteen and nineteen ounces respectively.

Cystic disease of the kidneys.—We stated that in many cases of renal cirrhosis there are found in the kidneys immense numbers of cysts, both microscopic and visible to the naked eye. Such kidneys have been termed "micro-cystic" (p. 657).

There are, however, other cases in which the cysts are much larger, sometimes as large as oranges, and these have been described separately under the name of "cystic disease of the kidneys." The organs then look as if they were each made up of a mass of rounded cavities, separated by an abundant fibrous matrix. There still exist remnants of secreting tissue, by which the renal functions have, however imperfectly, been kept up. The calyces and the pelvis are little, if at all, altered, an obvious distinction from hydronephrosis. The cysts have walls of varying thickness, and are filled, some with clear yellow fluid, others with red or brown, or with a gelatinous substance. The fluid always contains albumen, and sometimes blood-discs, leucocytes, or plates of cholesterine; but urea and uric acid are said to be generally absent.

There seems to be no doubt that the cysts are formed out of the tubes of the cortex, exactly like those which occur in cases of renal cirrhosis, and this supports the original opinion of Wilks and Moxon that the megalo-cystic, as well as micro-cystic, kidneys belong to Bright's disease.

An important clinical difference, however, between the megalo-cystic kidneys and those in which minute cysts complicate cirrhosis is the fact that the former can often be felt during life through the abdominal walls. Bright himself recorded such a case, in which a distinct tumour was detected in the left loin some months before death, and afterwards another was discovered in the right loin. Sir William Roberts recorded a case in which he successfully diagnosed not only the renal nature of the two tumours that he discovered, but also the exact character of the disease by which the kidneys were affected; the tumours appeared to be soft, but not fluctuating, about as large as cocoa-nuts, but disproportionately long. After death one kidney weighed twenty-eight ounces, the other twenty-six. In many instances they have been larger still. Thus, there was at Guy's Hospital in 1867 a case in which the right kidney weighed eighty-four ounces, and the left fifty-three. In two other published cases the weight of the two kidneys together was six and a half pounds in one, and eight and

three quarter pounds in the other. Perhaps the most remarkable case is one brought before the Pathological Society by the late Dr Hare (vol. iii), in which the left kidney alone weighed sixteen pounds, the right being in a comparatively early stage of the disease, so that it was only of about twice the natural size. During life a tumour filled the whole left side of the abdomen.

Such monstrous cystic kidneys are sometimes congenital, and may form a serious or insuperable obstacle to delivery of the fœtus (Virchow, 'Ges. Abh.,' p. 864). The origin of this foetal condition is very obscure; it is supposed to depend on inflammation of the straight collecting tubes of the kidneys *in utero*.

In some adult cases there is reason to suspect that the affection is of intra-uterine origin, and apparently it has ended its course, for there may be no symptoms of renal disease; moreover, microscopical examination shows plenty of healthy secreting tissue. In such cases compensatory hypertrophy has probably taken place after birth. In other cases, as in one observed by the writer in a man of fifty-three, there were symptoms which led to the diagnosis of renal cirrhosis during life (compare Mr Eve's case, 'Path. Trans.,' vol. xxxi, p. 164).

F. S—, twenty-seven, was admitted into Guy's Hospital March 4th, 1894. He had enjoyed good health till a fortnight ago; then vomiting appeared, followed by diarrhœa also. Stupor with stertorous breath supervened. A small amount of albumen was present and casts. He died comatose without convulsions. We found one patch of retinitis in the left eye. After death the body was well developed, and no anasarca was present. The brain weighed 48 ounces, and was not œdematous or otherwise unhealthy. Lungs ditto; right 41 ounces, left 51 ounces. Heart 15 ounces; left ventricle hypertrophied; light atheroma of aorta. Stomach and intestines normal. Liver 41 ounces, normal; adrenals and pancreas also. Spleen 7 ounces. Kidneys: right $10\frac{1}{2} \times 5 \times 2\frac{1}{2}$ inches, left $9 \times 4\frac{1}{2} \times 2$ inches; right, natural tissue; left, some healthy secreting tissue.

What is most remarkable is that these hypertrophic cystic kidneys are sometimes found in association with cysts of the liver. Bristowe twice recorded this coincidence ('Path. Trans.,' vols. vii and x), and in the same seventh volume Wilks related a similar case. Rindfleisch met with one, and Frerichs with another. A marked example was brought before the Pathological Society in 1881 by the present writer ('Path. Trans.,' p. 117, vol. xxxii) with histological figures. As there stated, microscopical examination "proves that this extremely remarkable form of cystic degeneration is histologically the same as the ordinary microcystic form of kidney associated with the later stages of chronic Bright's disease." Rindfleisch's suggestion that hypertrophic cystic kidneys may be examples of cysto-sarcoma seems to be far less probable.

The cysts in the liver seem to have a different origin—not to be retention-cysts, but to result from vacuolation of the hepatic cells; at least, so Dr Beale concluded in his report on Dr Bristowe's case in 1856, and the same result was independently reached from a study of the case in 1881.

Drs Savage and Hale White have since described two cases of general paralysis in which cystic degeneration affected not only the liver and kidneys, but also the brain, the lungs, and the heart ('Path. Trans.,' 1883, p. 1, with plate). In the same volume Dr Mahomed recorded another case of cystic liver and kidneys (xxxiv, p. 182). See also Dr Paterson's paper ('Brit. Med. Journ.,' Sept. 27, 1890, p. 735), and Rolleston and Kanthack's ('Virchow's Archiv,' Bd. cxxx, S. 488).

The *symptoms* of cystic disease of the kidneys, when not latent, are like those of other forms of chronic Bright's disease. The urine is often excessive in quantity, pale and of low specific gravity. It generally contains albumen, and occasional hæmaturia has been observed. There has sometimes been marked emaciation with great prostration of strength. But many patients have not been known to be ill until they were attacked with uncontrollable vomiting, or with uræmic convulsions and coma, or (as has happened in two instances at Guy's Hospital) with cerebral hæmorrhage.

It is obvious that, in the absence of the characteristic abdominal tumours, the clinical recognition of this affection must depend upon the extent to which the renal cortex is destroyed. A few scattered cysts give no symptoms, and even when the kidneys are enormously enlarged there may be enough secreting tissue left to carry on the functions of the kidneys.

Both clinically and pathologically this cystic disease of the kidneys must be carefully distinguished from the presence of one or more apparent cysts in cases of hydronephrosis (*v. infra*, p. 688). The former condition is bilateral, the symptoms are those of Bright's disease, and beside the visible cysts, there are numberless others of microscopic size; whereas the latter affects one kidney only, the cysts are very few in number, and the symptoms are those caused by local pressure.

Mutual relation of the several forms of Bright's disease.—At present there is general concurrence as to the broad divisions of the group of affections first recognised by Bright. The supposition of Frerichs that all the various anatomical forms present different stages of the same pathological process is universally abandoned. The eight species of morbus Brightii once described by Rokitansky, and the thirteen admitted by Rayer, are now scarcely remembered. The three anatomical forms recognised by Bright's successors at Guy's Hospital, Barlow, Rees, and Wilks, viz. the large red kidney of acute nephritis, the large, smooth, white kidney of subacute and chronic nephritis, and the contracted granular kidney of cirrhosis, are now generally accepted. To these Virchow added the lardaceous form of disease as a fourth variety of morbus Brightii.

The first and second forms are closely related, and may be regarded together, as acute and chronic stages of the same process. The fourth may either be considered, as we have considered it in this volume, independently, or may be regarded (with Bartels and Lecorché) as part of a general degeneration affecting other organs beside the kidney, and when attacking the kidney producing or complicating either the tubal or the interstitial form of Bright's disease.

Subject to these differences (which only affect nomenclature and classification) the division into acute and chronic tubal (or parenchymatous) nephritis, cirrhosis or chronic interstitial nephritis, and lardaceous disease has been recognised by Johnson, Stewart, Roberts, and Dickinson in this country, by Wagner, Niemeyer, and Eichhorst in Germany, and by Charcot in France.

The origin, sequence, and exact relation of the several pathological processes remain a difficult question. Some of the chief points still disputed are the following:

a. Does the "coarse," hard, congested kidney (the *Stauungsniere* of German authors), often seen in cases of chronic valvular disease of the heart, go on to become either a large, rough, white, or a contracted granular

kidney? Probably the true answer is that it may end in interstitial cirrhosis, but that this result is very rarely reached because the primary cardiac disease goes on more quickly.

b. Is acute glomerular nephritis distinct from the first stage of tubal nephritis, and may it go on to produce the large white kidney? It does not appear to be more than a frequent complication of scarlatinal nephritis, and such cases may end in the chronic form.

c. Does the renal affection of diphtheria, erysipelas, enterica, pneumonia, and other febrile diseases ever lead to a chronic structural lesion with the clinical symptoms of any form of Bright's disease? There is reason to believe that it very seldom does: the nephritis which follows scarlatina is probably a distinct infective process throughout.

d. Is the large white kidney always preceded by an acute stage, or may it be produced by a process of tubal nephritis which begins insidiously? While the possibility of such a process cannot be denied, the more carefully cases of renal dropsy are investigated the less frequent it appears to be.

e. Does the large white kidney ever become contracted and granular? This question, of the existence of a third atrophic stage of tubal nephritis, may now be considered as definitely decided in the affirmative. It is less frequent than recovery and less frequent than death in the second stage, but it undoubtedly occurs, and can often be distinguished from primary cirrhosis during life.

f. Is the contracted granular kidney always the result of a slow and insidious process from the beginning, or does it ever originate in an acute form of nephritis, such, for instance, as that which follows scarlatina? Although the latter pathological sequence is rare, such cases no doubt occur.

g. May lardaceous disease of the kidneys produce symptoms of Bright's disease without tubal catarrh or interstitial inflammation? This is very dubious, for slight lardaceous degeneration without secondary changes in the kidneys is often found where no symptoms of Bright's disease have been present during life. The rule is for a lardaceous kidney to become a large white one; when the lardaceous change coincides with cirrhosis it is probable that the former is subsequent in date to the latter. Just as a contracted granular kidney is often complicated by tubal nephritis, so it may be complicated by lardaceous changes.

h. Is consecutive cirrhosis of one kidney pathologically identical with chronic Bright's disease? We have seen reason to affirm that it is.

i. Are hypertrophic cystic kidneys to be ranked with microcystic specimens as exceptional cases of chronic Bright's disease? If we exclude congenital cases, this question also may be answered in the affirmative.

Geographical distribution.—The acuter forms of Bright's disease are most common in temperate Europe, in Australia, and in the United States. They are more frequent in the colder districts of New England and the middle States, and less so in the Gulf States and the dry, though cold, districts of the western plateau. They are rare in severe climates, as Canada and Iceland (according to Dr Hjaltelin, quoted by Dickinson), and also in the south of Europe, at the Cape, in India, and in the tropics generally.

Lardaceous nephritis, being almost always secondary to suppuration or syphilis, is found wherever these conditions occur; hence it has greatly diminished in frequency since modern surgery has rendered suppuration so much more rare, and since syphilis is earlier recognised and more efficiently

treated. It is said to be, relatively to other forms of Bright's disease, more frequent in India than in Europe.

Cirrhosis of the kidneys is decidedly more common in England, Scotland, and America than on the Continent of Europe. Its prevalence is probably connected with that of gout, and both directly and indirectly with intemperance.

Occurrence in animals.—Acute tubal nephritis with hæmaturia is well known as "red water" among lambs and horned cattle.

Chronic interstitial nephritis is, according to Mr Bland-Sutton ('Path. Trans.,' vol. xxxvii, p. 579), a well-known disease in veterinary medicine, and its association with arterio-capillary fibrosis and cardiac lesions is recognised. He reports, from his own observation, changes in the medium-sized arteries and hypertrophy of the left ventricle concurring with interstitial nephritis in horses.

Treatment of Bright's disease and of its complications.—The plan of treatment depends on the stage of the malady, and on the symptoms present at the time.

Acuter cases.—In the early period of parenchymatous nephritis the patient should be kept strictly in bed. It is often well to put him between blankets, and he should always wear a flannel gown with sleeves down to the wrists, so that the arms may not get cold when they are put outside the bedclothes. The diet should consist mainly of milk, but farinaceous food may be allowed. Beef-tea and meat extracts are probably injurious from the kreatine and other stimulating nitrogenous compounds they contain.

The routine prescription at Guy's Hospital, since the days of Addison, has been the diaphoretic liquor ammoniæ acetatis, with or without small doses of antimonial wine.

Where there is severe lumbar pain, it may often be relieved by leeches or cupping-glasses; and this local bloodletting sometimes relieves threatening suppression of urine. Bleeding from the arm is indicated in the most acute and formidable cases.

The benefit that has sometimes followed exploratory operations on the kidney has led Mr Reginald Harrison to propose exposing the organs in cases of acute nephritis with suppression of urine, and dividing the tense fibrous capsule, or puncturing the kidneys and so directly relieving their congestion (Brit. Med. Assoc., Cheltenham, 1891).

It is important to keep up a flow of water through the glomeruli, so as to wash out the tubes and empty them of the cell-masses and casts by which they are blocked up. Dr Dickinson recommends the patient to drink water freely, calling it very justly a most efficient but unirritating diuretic. Many physicians give the diuretic salts of potass, or the spiritus ætheris nitrosi, or inhalations of oil of juniper; but no one would prescribe broom, squills, copaiba, digitalis, or mercury.

Sir William Roberts believed that the acetate or citrate of a fixed alkali is of benefit, because the salt is converted into a carbonate, which tends to diminish the acidity of the urine, and so to prevent its irritating the kidneys as it passes through them. There seems to be no objection, in the later stages of the disease, to the use of such mild vegetable diuretics as horse-radish, juniper, and uva ursi, but not much good is to be expected from them. Of late years the writer has prescribed the salts of strontium, but has not been convinced of their usefulness.

The importance of setting the skin freely in action is generally recognised.* It is best effected by *baths*. One plan, advocated by Liebermeister, is that of placing the patient in a bath at about 100°, and then gradually adding more and more hot water until it reaches 104° or 106°; he is left in the bath for half an hour, or even an hour, and is afterwards closely packed in a sheet and warm blankets for two or three hours longer, during which time profuse sweating occurs. It is said that after such a bath the weight is often reduced by from two to four pounds, and even in children by half a pound or a pound. Many experienced physicians advise packing the patient in a sheet wrung out of hot-water, and covered with one or two well-warmed blankets. Others employ "hot air baths," a lamp being placed in the bed in which the patient lies, while the bedclothes are raised by means of a cradle so as to keep a confined space of air around his body. One of these methods may be repeated every day or every other day, especially in cases attended with severe dropsy. Their effect upon this symptom is often very striking, and may be followed by an increased secretion of urine as well as of urea; but if there is prostration or great dyspnoea they may be dangerous. Sometimes they cause faintness or headache, and a feeling of oppression, or a rise of temperature, and they may apparently bring on uræmic convulsions. Occasionally one has seen these grave symptoms, probably of toxic character, follow the use of a hot-air bath, so that it must be used with caution. It may be, as Bartels suggested, that the effect of rapid absorption of dropsical serum may be to transfer soluble poisons from their safe position in the subcutaneous tissues and serous cavities to the lymphatic and venous circulation, and thus to bring them rapidly to bear upon the nervous system and the heart.

One of the most powerful diaphoretic drugs is *pilocarpine*, the active principle of *jaborandi*, of which from one sixth to one third of a grain may be injected subcutaneously, or about twice that quantity taken by the mouth. It gives rise to a great flow of saliva and also to abundant sweating, but the latter effect is not always so well marked in persons who have Bright's disease as in those who are healthy. In some cases, moreover, it produces unpleasant symptoms, such as nausea, vomiting, or even collapse. To obviate them Wagner recommends the administration of a little brandy, or wine, or coffee, before the pilocarpine is injected.

The regular administration of *purgatives* is of equal importance. The usual practice is to give a dose of compound jalap powder twice or three times a week. If this causes sickness, saline laxatives must be substituted, or, if needful, elaterium. Calomel should never be employed, except in a single dose, for comparatively small quantities are apt to set up salivation in persons who have diseased kidneys. This caution was given by Osborne and by Bright himself ('Guy's Hosp. Rep.' for 1836, p. 375).

When dropsy cannot be otherwise got rid of, it becomes necessary to resort to *acupuncture*, or draining with Southey's tubes. The legs should be placed in a dependent position first, and the surface should be then cleansed and carefully oiled, in order that inflammation of the skin may be as much as possible prevented. As the serum trickles away, it should be soaked up by blankets or conveyed to a vessel. There is often a great diminution of the anasarca and even of ascites. By antiseptic precautions we can now prevent the danger of erysipeloid inflammation, which used occasionally to cause sloughing, and thus prove fatal.

* It was strongly advocated by Dr Osborne, of Dublin, as early as 1835.

During *convalescence* from an acute attack of Bright's disease the greatest care must be taken to prevent the occurrence of a relapse. The patient should be warmly clad with flannel next the skin. He must avoid exposure to cold, as well as muscular fatigue and long railway journeys. The diet must still be carefully regulated, as regards both solid food and alcoholic drinks. Johnson published some remarkable cases of recovery under a purely milk diet continued for weeks or months.

Chronic cases.—The *anæmia* of Bright's disease is best met by tincture of steel, but not while the urine continues scanty and smoky. In treating pleurisy or pericarditis, blisters are best avoided.

When the *tinctura ferri perchloridi* is not well borne, some milder preparation must be substituted, such as the *ferri et ammoniæ citras*, or the *pilula ferri*. If there is headache, the bromide of potassium is very useful. It is often of great advantage to the patient to spend the winter in a warm dry climate, such as that of the Riviera, or of Egypt, but often the Canaries or Cornwall or the Channel isles are quite as favourable. In renal cirrhosis the first indication is to relieve the arterial tension. No doubt this is part of a system of compensation; but it must be desirable to relieve the blood of any substances which the kidneys are unable to remove, and the accumulation of which renders the excessive arterial pressure necessary. As a matter of experience, the regular administration of *purgatives* is of great value, and seems to be capable of warding off cerebral hæmorrhage for a time. *Nitro-glycerine*, again, is sometimes serviceable, especially in relieving uræmic dyspnoea; or recourse may be had to inhalations of nitrite of amyl, although its effect is only temporary. When sedatives are needed, chloral hydrate is safe, and usually efficient. Tincture of henbane and chloralamide and sulphonal deserve the same praise.

There is no indication for the administration of water as a diuretic, and when there are signs of cardiac dilatation it is better to limit the amount of liquid taken by the patient to as little as he can take with comfort.*

For acute *uræmia* venesection is the most effectual remedy; the abstraction of ten or fifteen ounces of blood, *pleno rivo*, may be followed by a striking and rapid subsidence of the symptoms. A couple of leeches on each temple is one of the most efficient modes of relieving uræmic headache, and probably of warding off more dangerous symptoms. In other cases the inhalation of chloroform proves effectual, and bromides relieve a less severe headache.

It is generally advisable to give a purge; and we may prescribe gamboge or elaterium or croton oil—drugs which are better avoided in ordinary cases. Sometimes cold affusion to the head may be employed with advantage. If further experience should confirm the belief that the occurrence of uræmia often leads to a greater activity on the part of the kidneys, it would be well to abstain from diaphoretics, such as pilocarpine or hot air baths.

In cases of cardiac dropsy, secondary to renal cirrhosis, when the left ventricle is dilated, and the pulse soft, frequent and irregular, *digitalis* is often invaluable. By it the disease can often be kept at bay for a considerable time, and the patient may even be restored to a state of apparent health.

It is important to remember that in cases of Bright's disease (particularly

* This treatment was long ago taught by v. Ziemssen, and has lately been strongly advocated by Ewald and von Noorden.

when the renal cortex is much wasted) even a small dose of opium or morphia may produce fatal cerebral symptoms. Thus in one case a grain of opium, prescribed for pain in the head, appeared to be the cause of convulsions and stupor that ended in death; and in another case like results seemed to be due to the administration of a third of a grain of morphia for lead colic. A patient under the writer's care, who was suffering from cancer of the throat, died comatose from subcutaneous injection of a fourth of a grain of morphia, and after death the kidneys were found to be cirrhotic with wasting of the cortex. This caution was given by Dr Bright himself, and has generally determined practice since; but lately Dr Stephen Mackenzie has published cases in which uræmic symptoms were apparently relieved by morphia. It is possible that there may be exceptions to the general rule, but at present we do not know under what peculiar circumstances the danger may be absent. Even if we admit that morphia is admissible, we must not shut our eyes to its possible danger.

Prognosis.—By such treatment with drugs, combined with strict diet, and, if possible, removal to a warm climate during winter and spring, cases of Bright's disease, both parenchymatous and interstitial, may be greatly alleviated and not infrequently cured. The statement made thirty years ago, that parenchymatous nephritis, when once established, is as hopeless as tuberculous phthisis, is certainly untrue now, even with the better prognosis of phthisis which pathology and clinical experience alike have taught us.

Neither uræmic convulsions, nor Cheyne-Stokes breathing, nor dermatitis, nor retinitis are so fatal as these symptoms have been thought to be. Grave they undoubtedly are, but the writer has witnessed recovery follow after each one of them. On the whole, his prognosis is much more hopeful in cases of chronic Bright's disease generally than it was thirty years ago.

A considerable proportion of acute cases recover completely under judicious treatment: many of the chronic cases last until they can no longer be counted a disease (*cf. supra*, p. 662); and in one case which the writer watched for more than 25 years the albuminuria and the signs of cardiac hypertrophy gradually disappeared and the patient is now in good health, the only difference being that he golfs instead of hunting.

RENAL CALCULI AND THEIR EFFECTS

“I had a while talked with him, first of his diseases, both in his brest of olde and in his reynes nowe by reason of grauel and stone, and of the crampe that diuers times grypeth him in his legges.”—SIR THOS. MORE: *Letter to Lady Alington*, fol. 1434.

Structure and formation of calculi—Lithic or Uric acid and Lithates—Oxalate of lime—Cystine, Xanthine, Indigo, and other rare calculi—Phosphate of lime—Carbonate of lime—Fusible calculus—Ætiology of calculi.

Symptoms—haematuria: its other causes—nephralgia—vomiting—treatment.

Effects—obstruction and suppression of urine—atrophy of one kidney—hydro-nephrosis—anatomy and causes—diagnosis from hydatid and single cyst—Prognosis and treatment.

Hydropyeltis and renal abscess, with other causes of pyuria.

Treatment of calculi—solvents of lithic acid—Nephrotomy, nephrectomy, and nephrolithotomy.

THE great emunctory organs of the body are adapted for removal of its excreta in different physical conditions. Carbonic dioxide passes off in a gaseous state from the lungs; water from the lungs, skin, and kidneys; and insoluble solids from the bowels; while the soluble excreta are removed by the kidneys alone. Under certain physical or chemical conditions these last products are no longer held in solution, but form urinary precipitates, chiefly “red gravel” consisting of lithic acid and the lithates, and “white gravel” consisting of the earthy phosphates—both fully discussed in a former chapter (p. 578). Sometimes, however, instead of forming crystalline or amorphous deposits, these products form larger aggregations which are known as calculi or stones, and produce characteristic and very serious symptoms.

The formation of calculi by concretion of insoluble mineral products in the urinary passages is exactly comparable to that of gall-stones, of salivary or of prostatic calculi, and of rhinoliths; and we shall find that there is a remarkable likeness in the effects of biliary and renal calculi.

Formation of calculi.—It has long been known that most calculi are made up of concentric layers which differ in composition, and that the nucleus or central part may be quite distinct in character from the rest of the stone. Most calculi are formed chiefly of lithic acid, according to Sir William Roberts five out of six; and lithic acid was once thought to be the most frequent constituent of their nuclei. This, however, was a mistake; even when the apparent nucleus consists of uric acid, the microscope generally shows that the very centre is either an aggregation of spheroids

or dumb-bells of oxalate of lime, or else hedgehog-crystals of urate of soda. In countries where *Bilharzia hæmatobia* is endemic, its ova frequently form a nucleus; and in other cases it consists of inspissated mucus, or a small blood-clot, or casts of the renal tubes. Ebstein once found in the urine a deposit of epithelial cells from the renal pelvis beautifully encrusted with uric acid. As regards the spheroids of oxalate of lime which form the nuclei of most calculi, Dr Beale maintains that they often have their origin within the tubes of the kidney; not only has he found dumb-bells in the substance of casts, but he describes and figures microscopic calculi, already laminated, which he says he has many times seen in the renal tubes after death.* Lastly, vesical calculi are sometimes moulded on foreign bodies introduced into the bladder from without.

In the laminæ which generally make up the body of a calculus the materials are often laid down in a definite manner. Thus lithic acid appears in the form of rods or columns, piled one upon another or arranged side by side; the lithates appear as globules, with concentric rings; oxalate of lime may form spheroids, and dumb-bells of this substance are sometimes embedded in laminæ consisting mainly of urates. Clear crystalline layers of uric acid or calcium oxalate make up a large part of some concretions.

Although few calculi consist entirely of one substance, there are conspicuous chemical distinctions between them; and corresponding differences in their physical characters.

1. *Uric acid* ($C_5H_4N_4O_3$).—These are the commonest of all stones. They occur as extremely hard bodies, of round or oval shape; smooth on the surface or tuberculated; of an ochrey, fawn, or reddish colour. They are formed in the pelvis of the kidney; and sometimes are passed in enormous numbers while still small, from the size of poppy seeds up to that of mustard seeds or split peas. Not infrequently several lithic acid stones, perhaps as large as marbles, are found in the pelvis of the kidney, or in the bladder. These multiple calculi often have flat surfaces or smaller facets, produced by contact with one another, and the presence of such surfaces is important as an indication that the concretion is not solitary. Calculi of uric acid sometimes weigh three or four ounces.

2. *Urate of soda* ($C_5H_3NaN_4O_3$).—These are soft concretions, of rare occurrence, which appear never to reach a large size, except by the addition of lithic acid, or some other substance. Like crystalline deposits of lithates they occur chiefly in children.

3. *Oxalate of lime* (CaC_2O_4).—These are the only common renal calculi beside those of uric acid. They are characterised by extreme hardness and a rough irregular surface, whence the name of "mulberry calculi." They are, however, often passed safely while still small, as smooth, rounded, grey or brown bodies, like hemp seeds; or they may be covered with glistening crystals. As a cause of hæmaturia, pyelitis, or renal colic in middle-aged people, such calculi are probably more frequent than those of uric acid. The larger "mulberry" stones are generally of a blackish-brown colour, and irregularly rounded form; Roberts, however, says that stones of the same composition are sometimes oval, smooth, and bluish-grey.

* Dr Beale met with a remarkable instance in which a smooth, oval urethral calculus, two inches and a quarter in length, was composed entirely of minute concretions of calcic and triple phosphates, united by a whitish material; this, however, was altogether an exceptional specimen, for it is said to have lain in the urethra for fifty years before it was extracted. In vol. xiii of the 'Pathological Transactions' is a full account of this case by Mr. Haynes Walton.

When crushed, they break into sharp, angular pieces. Mulberry calculi are usually solitary, and are never present in large numbers. It seldom happens that more than two of the small hemp-seed concretions are passed by the same patient, and then only at long intervals.

4. *Cystine* ($C_3H_5NSO_2$).—Calculi of this substance are very rare. They are usually egg-shaped; their surface is granular, and glistens with minute crystals; they are of a honey-yellow colour, and on section look semi-translucent, like beeswax, and show indications of a radiating structure. It is curious that when exposed to daylight for a long time they slowly acquire a delicate green hue, which is therefore the colour of museum specimens. They are of rather soft consistence, so as to be marked by the nail—a distinction from the uric acid calculi, which they somewhat resemble. They are soluble in ammonia. They may reach a considerable size, weighing as much as three or four ounces, notwithstanding the low specific gravity of cystine. Roberts describes a specimen, passed *per urethram*, which was cylindrical, an inch and a quarter long, and weighed twenty-seven grains. They usually consist of pure cystine; but one mentioned by the same author had a nucleus of uric acid, with an outer layer made up of a mixture of uric acid and cystine. In the 'Path. Trans.' for 1880 (p. 182) Mr Shattock described a cystine calculus which contained a minute proportion of oxalate of lime throughout, beside a thin layer consisting entirely of that salt: the nucleus was of cystine. In the Guy's Hospital Museum there are ten cystine calculi, successively passed by the same patient (Nos. 2144, *seq.*). Mr Jacobson once removed from the kidney of a patient of the writer's a calculus of cystine weighing 387 grains.

5. *Xanthine* ($C_5H_4N_4O_2$).—This substance was discovered by the elder Marcet, about the year 1817, in a calculus given him by Dr Babington; and only five instances of its occurrence are on record altogether. Its chemical composition is that of uric acid, less one atom of oxygen; hence it was at one time termed "uric oxide." The characteristic test for it is analogous to the well-known murexide test for uric acid: when moistened with nitric acid it dissolves without effervescence, and on evaporation there is left a bright yellow residue; this, when cool, becomes violet-red (not purple-blue) if treated with a solution of caustic potass. It is insoluble in cold, and sparingly soluble in hot water (1 in 1000 parts at $100^\circ C.$), but is readily dissolved by liquor ammoniæ or liquor potassæ.

Hitherto xanthine calculi seem never to have been found in the renal pelvis, only in the bladder. In their physical characters they resemble uric acid calculi. One removed by Langenbeck from a child weighed 339 grains ('Guy's Hosp. Reports,' vii, 202; Museum. 21459^o).

6. *Indigo* (C_8H_5NO).—This substance, like xanthine, scarcely ever occurs as a urinary deposit, though it occasionally colours lithates. As a concretion it has only once been found in the renal pelvis of a middle-aged woman, by Dr Ord ('Path. Trans.,' 1878). It formed a flat broad cake, like a lozenge in shape and size, and weighed forty grains; its surface was partly dark brown, partly bluish black; its section grey and polished. On paper it left a blue-black mark.

7. *Urostealith*.—This name was given by Heller in 1845 ('Heller's Arch.,' Band ii) to certain soft, elastic concretions, like india-rubber, which were passed by a young man. Sir John Moore, of Dublin, has since ('Dubl. Quart. Journ.,' xvii) met with similar specimens, and in the Museum of the College of Surgeons there are two which belonged to Hunter's collection.

The latter were taken from the bladder, and perhaps the fatty salts of lime, of which they are entirely made up, were formed by the decomposition of a solution of soap, which might have been injected for therapeutical purposes. This theory, however, seems not to apply to the other cases.

In the 'Med.-Chir. Trans.' for 1872, Mr McCarthy described certain calculi of peculiar form, eleven in number, which were taken from the left kidney of a woman after death. When first removed they felt soft and greasy, and they each consisted of a central globular body, with long tapering spines projecting from it. None of these appear to have been analysed, but a somewhat similar concretion from the right kidney was found to contain 36.6 per cent. of fat and cholesterine, the other chief ingredients being lithates (35 per cent.) and oxalates (9 per cent.). Similar specimens in the Museum of the College of Surgeons are said to consist of oxalate of lime. Mr Benjamin Duke, of Clapham, once removed several similar calculi after death.

8. *Phosphate of lime, or bone earth* ($\text{Ca}_3\text{2PO}_4$).—All the varieties of calculi hitherto described appear to occur in acid urine; certainly this is the case with the two of clinical importance—uric acid and calcic oxalate. This is an important distinction between them and the three remaining kinds, for the latter can only be formed when the urine is alkaline. One, which is very rare, consists of phosphate of lime. Concretions formed of this substance are described by Roberts as being white and chalky in appearance, and rather smooth on the surface, with an earthy fracture. Their texture is sometimes loose, sometimes very compact; and they vary in size from a pea to a hen's egg. In the Museum of the Manchester Infirmary there is a laminated specimen in which bone earth alternates with uric acid. A peculiarity of calculi composed of pure phosphate of lime is that while they require alkaline urine for their production, it must not be alkaline from ammonia, since, if it were, the triple phosphate could not fail to form, and to make up a large part of their substance.

9. *Carbonate of lime* (CaCO_3).—Concretions of this substance are very rarely seen in the human subject. Roberts says that when they do occur they are generally small very hard bodies, varying from the most minute size up to that of a hazel nut; grey, yellowish, or bronze-coloured; smooth on the surface, sometimes with a metallic lustre. Haldane, of Edinburgh, once found many small calculi consisting of carbonate of lime in the renal pelvis of a man who had died from spinal abscess. Some years before, Roberts had met with a case in which immense numbers of precisely similar bodies were passed during life. The largest of them were the size of mustard seeds, they were translucent and of an amber colour, and showed a laminated structure under the microscope. They were probably also of renal origin. The urine in which they were found was ammoniacal.*

10. *Mixed calcic and ammonio-magnesian phosphates* ("fusible calculus").—We saw how the decomposition of urea into carbonate of ammonia during the alkaline fermentation of urine inevitably leads to the formation of the "triple" phosphate of ammonia and magnesia; and how this salt and the amorphous phosphate of lime come down together as a precipitate, which has a strong tendency to agglomerate into a mortar-like mass (p. 589).

* In vol. xix of the 'Pathological Transactions' a large renal calculus is described as composed of carbonate of lime, but in vol. xxviii it is stated that on a re-examination of the specimen it proved to consist of other materials.

It sometimes collects on the surface of the inflamed mucus membrane of the bladder, or on any foreign body exposed to its action, particularly upon calculi, of whatever nature, which are washed by putrid urine. It rarely forms the starting-point of a stone, whether in the kidney or in the bladder; but it often converts a small nucleus into a concretion of enormous size. It is a soft friable substance; in the blowpipe flame it melts into a kind of enamel, whence the term "fusible calculus." In the bladder it may form stones weighing as much as twenty, thirty, or even forty ounces. In the kidney it often takes the shape of the pelvis and calyces, each branch with an expansion at its end. This is the only calculus, beside those of lithic acid and calcic oxalate, that is not rare.

Dr Gee has recorded ('Med.-Chir. Trans.,' vol. xxxix) a case in which such a concretion weighed thirty-six and a quarter ounces; it, or rather the remains of the kidney stretched over it, had been felt during life as an abdominal tumour of stony hardness. As is often the case, its surface was covered with brilliant crystals of pure triple phosphate, but these were of exceptional size and beauty, some of the prisms being half an inch long. The whole of the calculus was very hard and dense; and it had a dark brown nucleus, which consisted mainly of oxalate of lime. Such "coral-like" masses, sending prolongations into the several renal calyces, are only found as a result of ammoniacal decomposition of the urine in the renal pelvis, and are probably always composed of mixed phosphates. Dr Ord and Mr Wagstaffe have published similar cases in the 'Pathological Transactions.'

Ætiology.—It is apparent that, in acid urine, the only calculi likely to form are composed of lithic acid, or of oxalate of lime, or of alternating layers of lithic acid, lithates, and oxalate. Phosphatic calculi very rarely occur except when the urine is ammoniacal, and only by deposition on a pre-existing nucleus, which (when not a foreign body) is either lithic acid or oxalate of lime. The ætiology of primary calculus, therefore, resolves itself into the causes that favour the precipitation of uric acid or calcic oxalate from the urine.

The eastern counties of England, and especially Norfolk, are well known to yield a much larger proportion of cases of stone—at least of stone in the bladder—than any other district. But, in his address on surgery delivered before the British Medical Association in 1874, Mr Cadge, of Norwich, estimated that lithic acid and the lithates make up nine tenths of the calculi observed there, whereas for the whole of England a lower proportion (usually five sixths) is generally given. It seems therefore probable that the excessive number of cases in the eastern counties is due to calculi of uric acid, the frequency of calculi formed of oxalate of lime being no greater there than elsewhere. Mr Cadge could only call to mind three cases in which he had removed an oxalate of lime stone from an adult. In one of these cases, only the outside shell consisted of oxalate, the central part being uric acid; the patient had been recently living in North Wales, having previously left Norfolk, probably with a uric stone already in his bladder. Another case occurred in a soldier who had only been for a short time in this county. The third was in a Norfolk man, but he also had resided elsewhere.

Why deposits of lithic acid should occur so frequently in East Anglia is unknown. It is generally believed that hardness of drinking water is one cause of calculus, and the soil of Norfolk and Suffolk is mostly creta-

ceous; but the chalk formation is not confined to the eastern counties, and limestone soils make water "hard."

At first sight it seems difficult to understand how this "hardness," which means abundance of earthy salts, can affect the concretion of calculi, since calcic oxalate and lithates do not occur as minerals, and chalk is the rarest constituent of calculi. But distilled water will dissolve lithates, oxalates, or other more or less "insoluble" substances better than water already holding other salts in solution. Hence if the blood and urine of patients, who habitually drink well-water from a calcareous soil, contain more salts in solution than in the case of those who drink rain-water—a supposition which has not yet been verified—it would follow that the small quantity of oxalate of lime or lithic acid, which was held in solution in the urine in the latter case, would begin to be deposited in the former.

Calculi are said to be less common in Germany than in France and England, and to be remarkably rare in Sweden. In India, calculus is extremely common in the Punjab, not uncommon in Bengal, Oudh, and the North-west Provinces, less common in Bombay and Central India, and exceedingly rare in the Madras presidency and in Assam (Dr A. E. Roberts, the 'Lancet,' 1895, vol. i, p. 381).

Hereditary influence sometimes shows itself in a marked way; but persons who come to live in Norfolk are said sometimes to form a calculus very rapidly, while others who leave the district lose the proclivity.

In children stone is almost entirely confined to the poor, and Mr Cadge was inclined to think that a deficient supply of milk as food had to do with it. The late Sir William Fergusson is said to have complained that he only once received a fee for cutting a child for stone. The only child of well-to-do parents whom he had treated for this disease was said by the mother to have differed from all her other children in having never been able to take milk.

Sex and age.—Stone is far more frequent in males than in females; but this applies to stone in the bladder rather than in the kidney. In fact, the difference is probably not in the less frequent formation, but in the earlier and easier escape of calculi from the female bladder.

As regards age, vesical calculi are well known to be much less common in young adults than in children and in old people, but it is not certain that the same rule applies to renal calculi.

Symptoms and effects of renal calculi.—The morbid conditions produced by the presence of gravel or calculi in the kidney may be classed together as "nephrolithiasis." They depend upon irritation of the renal pelvis, or upon mechanical obstruction of the ureter, and may be arranged as follows,—the first three are symptoms, the rest pathological effects:

1. *Pain in the loins*, or renal lumbago.
2. *Hæmaturia*, the most constant effect of calculi.
3. *Renal colic*, consisting of nephralgia with vomiting, and produced by the passage of a stone into the ureter.

i. *Obstructive anuria*, or *suppression of urine*, caused by impaction of a calculus in one ureter, when the opposite kidney is from any cause already incapable of secreting urine.

ii. *Unilateral atrophy of a kidney*, from obstruction of its ureter.

iii. *Hydronephrosis*, from the same cause.

iv. *Pyelitis*, with *pyonephrosis* and *perinephric abscess*.

1. *Renal lumbago*.—In the mildest form of nephrolithiasis the principal symptom is a dull aching pain in the loins, *lumbago*. Such a pain is often the result of irritation of the kidneys by the urine, or by something deposited from it. It is practically difficult to distinguish such cases from those of myalgia (or, as it is vaguely called, muscular “rheumatism”) affecting the lumbar muscles, for in both cases the back may feel stiff, and stooping may aggravate the pain. When, however, the urine is strongly acid and deposits lithates, and relief is given by making it alkaline, we are justified in ascribing a renal origin to the attack. Often the pain is at once removed by a few full doses of the citrate of potass, repeated at short intervals. It may be a question whether lumbago is ever due to an over-acid state of the urine alone, or whether there is not an actual sediment of uric acid in the renal pelvis, which is re-dissolved when the secretion becomes alkaline. The dull aching sensation is sometimes experienced only when the patient first wakes in the morning, and ceases after he gets up, and “the alkaline tide” sets in after food (p. 574).

2. *Hæmaturia*.—The presence of blood in the urine is an almost invariable symptom of renal calculus. When hæmorrhage is profuse, the urine may look like pure blood. From this every gradation of colour may be observed down to the palest pink or the faintest brown tinge. The pink and red shades are due to the urine being neutral or alkaline, and are seen as a rule in cases of hæmorrhage from the bladder; the brown and smoky tints are caused by the hæmoglobin being changed into acid hæmatin, and therefore appear with an acid state of the urine. The red colour seen in profuse renal hæmorrhage depends on the alkaline serum neutralising the acid urine. In many instances, when the colour is not in itself distinctive, there is a sediment which cannot be mistaken: on tilting the chamber vessel from side to side, a granular-looking reddish-brown substance may be seen lying just within the edge of the fluid, and following its movements.

In any case the microscope will at once reveal the presence of blood in a drop of urine taken from the bottom of a vessel in which it has been standing for a little while, or sometimes from the bulk of the urine directly after it is voided. It is impossible to insist too strongly upon the importance of microscopical examination in all cases in which hæmaturia may be suspected; without it the absence of blood should never be asserted. The only cases in which hæmaturia may fail of detection by the microscope are when the urine is of very low specific gravity or in a state of ammoniacal decomposition, for in such cases the blood-discs may be rapidly destroyed. In urine of acid reaction and a normal density they remain visible for days.

Albumen must always be contained in urine in which there is blood. But the ordinary tests often fail to show it, when the discs are at once seen with the microscope.

Blood-corpuscles do not always retain their form in urine: they often have crenated edges, and sometimes they are shrivelled; in dilute urine they often, however, become delicate globular corpuscles slightly larger than natural, due to endosmosis, from urine being of much lower specific gravity than blood. Minute discoid forms of oxalate of lime might be mistaken for blood-corpuscles in urine, but they are more refracting and have no yellow tinge. Dr Beale speaks of cases in which spirilla looked so like blood-discs that great care was required to distinguish them. The microscope at once distinguishes hæmaturia from hæmoglobinuria (*supra*, p. 593)

by discovering the presence of blood-corpuscles. But the two conditions may co-exist or may each replace the other in the same patient.

Beside the microscopic test, the spectroscope may be used or chemical tests applied. One of the latter is known as Heller's; it consists in rendering the urine alkaline by the addition of caustic potass or soda, and then boiling, so as to precipitate the earthy phosphates, which carry down with them any blood-colouring matter that the fluid may contain. Salkowski says that this reaction is very delicate, but that it also occurs with the colouring matter of rhubarb or senna. Another test much employed in this country is adding to the suspected urine a drop of freshly prepared tincture of guaiacum, and shaking it up with a few drops of ozonic ether; if blood is present, a bright blue appears in the layer of ether that collects on the surface of the fluid when it has stood for a minute or so. The chief sources of fallacy lie in the fact that saliva, nasal mucus, and iodide of potassium give the same reaction. According to Dr Mahomed ('Med.-Chir. Trans.,' lvii) this guaiacum test is even more delicate than that of the spectroscope; but it is too uncertain to be of much practical value.

It must not be forgotten that blood may be added to urine for purposes of deception. In one such case the microscope showed that the corpuscles present were the oval blood-discs of a bird.*

Causes of hæmaturia generally.—Blood in the urine may be a symptom of a general disease: purpura, scurvy, smallpox, black-water fever, yellow fever, leuchæmia, or any other form of grave anæmia. Whether hæmaturia is ever vicarious to menstruation, or to a hæmorrhoidal flux, or to asthma is doubtful; such cases do not occur now as they used to. It may certainly be caused by congestion or embolism in valvular disease of the heart; and more frequently renal hæmaturia is a symptom of calculi and gravel, acute nephritis, and also of tuberculous ulceration, and new growths.

Bleeding of the bladder may be caused by stone, acute cystitis, *e. g.* from gonorrhœa, tuberculous or cancerous ulceration, villous growths, or the presence of *Bilharzia hæmatobia*.

Blood in small quantity is present in healthy women's urine during the catamenial flow.

Hæmaturia may appear when the kidney has been lacerated by violence, in cases of poisoning by oil of turpentine or by cantharides, and in very rare cases in which quinine, by a curious idiosyncrasy, produces a like effect ('Brit. Med. Journ.,' Jan., 1870). The renal congestion which in some patients is set up by the application of a blister sometimes produces hæmaturia; sometimes only fibrin is exuded, but in such quantity as to form transparent gelatinous clots in the bladder, and obstruct the outflow of urine. A case of this kind occurred in the writer's practice in 1886, and two are related by Bartels.

In some instances hæmaturia has been caused by undue sexual indulgence; and occasionally by hard riding on horseback or on a bicycle.

When blood is poured into the urinary passages in sufficient quantity to coagulate, symptoms of various kinds may result. Thus a clot in the ureter may produce an attack of "renal colic" just like that caused by an impacted calculus; and clots in the bladder may give rise to dysuria, or to complete retention of urine.

* I once checked an attempt of this kind by remarking that it was necessary for me to see the urine passed. The patient, who had had a railway accident, subsequently confessed that he mixed with his urine blood that came from a cut upon the wrist.—C. H. F.

Seat of hæmorrhage.—When blood-clots are passed in the urine, they often clear up all doubt as to the seat of the hæmorrhage. Sometimes they are discoid, and were evidently formed on the floor of the bladder; sometimes cylindrical, having come from a ureter. Microscopic blood-casts of the uriniferous tubes prove of course that the hæmorrhage is derived from the renal cortex. They are rarely found except in acute tubal nephritis.

We may be helped in discovering the seat of hæmaturia by the way in which the blood escapes during micturition. When bleeding occurs from the urethra, the blood precedes the stream of urine, and is washed out by it. On the other hand, when the bladder is the seat of disease, it is towards the end of micturition that the urine is most deeply discoloured. Dr Beale speaks of cases in which persons, apparently healthy, day after day pass small quantities of blood, just as micturition is ceasing; it would seem that "the effort required to expel the last drop of urine causes the rupture of a few capillaries about the membranous part of the urethra or the neck of the bladder." When there is hæmorrhage from the renal pelvis, the blood is always intimately mixed with the urine; but so it is likewise in many cases in which its origin is from the bladder.

When the urine is of red or pink hue this is due to the reaction being alkaline, and hence the seat of hæmorrhage is most likely the bladder.

If the urine, instead of being bright red, has a brownish hue ("like tea"), this shows that the blood-corpuscles have been acted on by acid, and their hæmoglobin turned to acid hæmatin; the probability is that it came from the kidney. If, however, the hæmorrhage is profuse, the urine remains red; for the blood-serum then neutralises the smaller amount of acid urine.

After all, it must be confessed that in certain cases, including some of the most severe, there are no certain indications as to the seat of the hæmorrhage, except from other symptoms, such as pain or dysuria.

In practice almost the only *vesical* affections that give rise to profuse hæmaturia, as their sole symptom, are villous tumours and other forms of new growth, generally malignant. The late Sir George Humphry believed that the occasional cessation of hæmaturia for a long period favours a diagnosis of villous disease of the bladder. Such diseases occur chiefly in patients who have reached middle age.

Thus in 1865 a child, only eighteen months old, died in Guy's Hospital from the effects of a polypoid growth of the neck of the bladder. The writer had once sent him, from Mr. Bryant, a minute clot which was passed with the urine of a little girl in apparent health, and only five years old; on microscopic examination a beautiful tuft of villous growth was apparent, and she soon afterwards died. In 1877 an autopsy was made in the case of a man aged thirty-four, who said that ever since he was twelve years old he had suffered from hæmaturia, recurring at intervals of weeks or months, with greater or less severity. There was found a soft spindle-cell sarcoma, growing as a flat, slightly lobulated tumour from the base of the bladder on one side.

Occasionally, as in a case of Murchison's ('Path. Trans.,' 1869), villous growths from the pelves of both kidneys are associated with a like affection of the bladder. Mr Davies-Colley once succeeded ('Clin. Soc. Trans.,' 1881) in removing through a perinæal incision a villous growth from the bladder of a man aged thirty-two, who had suffered for eight years from hæmaturia, and was completely cured by the operation. This patient had sometimes passed blood at the beginning of micturition, sometimes at its close. Sir Henry Thompson and several other surgeons have published successful cases of the same kind.

Hæmorrhage from the *renal pelvis* may be due to various causes. When a patient passes blood in the urine without there being other symptoms to throw light upon the nature of the disease, the presumption is generally in favour of the presence of a renal calculus; but the possibility that tubercle or cancer of the kidney may be developing itself must never be left out of consideration.* It is, however, surprising how often one meets in practice with profuse hæmaturia, which causes great alarm, and ceases after two or three days without any clue as to its source, leaving the patient apparently as well as ever. The late Sir William Gull reassured a student who was subject to this symptom by calling it epistaxis of the kidney.

In 1881 a man aged sixty-three was admitted into Guy's Hospital with extreme wasting and cachexia. After a few days he was attacked with severe hæmaturia. This, however, quickly subsided, though afterwards pus appeared in the urine, and three weeks later he died. At the autopsy it was found that the cause of the wasting was cancer of the œsophagus. In one of the calyces of the left kidney a calculus was impacted. The lining of the renal pelvis was much thickened, œdematous, and of a deep purple colour from ecchymosis, looking like velvet. Probably a like condition is generally present when hæmaturia is the main symptom.

It is a question whether bleeding ever occurs as a result of granular deposits of uric acid or oxalate of lime, or whether the presence of a calculus is necessary. Whenever even small calculi are present in the kidneys or their pelves, hæmaturia is apt to occur after horse exercise, bicycling, or riding in a railway carriage. Even when no blood is obvious to the naked eye, a microscopic examination of the urine passed afterwards may decide the presence of a renal calculus. In most cases of this kind there is lumbar pain, or pain referred to the front of the abdomen on one side, or to the groin. A fact to which Brodie drew special attention is that the symptoms are sometimes referred mainly to the bladder. Micturition may be frequent, and accompanied by a cutting pain in the neck of the bladder and in the urethra, so that the presence of a vesical calculus is suspected.

Treatment.—Whatever the cause of hæmaturia, rest in bed is essential. If the seat of hæmorrhage is the kidney, ice-bags are often applied to the loin; if the bladder, to the hypogastric region. It is doubtful whether cold applied to the surface has any decided effect in checking local hæmorrhage, either in the lungs, the brain, the stomach, or the kidneys; but there is better warrant for introducing injections of iced water into the rectum or into the bladder itself. Prout found the injection into the bladder of a solution of alum (twenty to forty grains in a pint of water) effectual when the hæmorrhage was vesical. As internal styptics, gallic acid, acetate of lead, ergot, and alum may be employed; and in some cases the tincture of perchloride of iron is particularly serviceable. Oil of turpentine, too, may succeed when all other drugs have failed; it would probably be injurious if the blood came from the renal cortex, but in cases of hæmorrhage from the pelvis of the kidney there is no objection to its use. The hæmaturia caused by cantharides is treated by cupping over the loins, warm poultices, diaphoretics, and purgatives.

On the whole, drugs have less effect on hæmaturia than on most other

* As Dr Mussen well observes ('Philadelphia Med. Journ.,' April 16th, 1898), if hæmaturia persists after a patient is put to bed, it is more likely to be due to cancer or tubercle than to calculus.

forms of hæmorrhage,—less, for example, than ergot on hæmoptysis, or opium and lead on bleeding from the bowels.*

Hæmaturia, though so important as a symptom, is not a serious occurrence in itself. It is far less dangerous than intestinal, gastric, or pulmonary hæmorrhage; indeed, few physicians have seen a fatal case of bleeding from the kidneys.

In the treatment of recurrent or persistent hæmaturia attributed to renal calculus the first thing is, if possible, to get rid of the cause of the disease. This will be considered at the close of the present chapter (p. 701).

3. *Nephralgia*.—The passage of a calculus down the ureter into the bladder is attended with symptoms which are commonly known as *renal colic*. The term is etymologically absurd; but probably the pain of impaction of a ureter is essentially the same as that of intestinal obstruction before inflammation sets in, or of so-called “biliary colic;” for in each case the cause of pain is the spasm of a muscular tube to overcome an obstacle. We may also compare the pain to that of urethral stricture and of childbirth.

The attack often sets in with extreme suddenness and violence, and is one of the most painful of diseases. The patient is sometimes awakened from sleep by an attack; sometimes it is brought on by the jolting of a vehicle, or by some muscular effort, such as sneezing, coughing, running, jumping, or riding on horseback.

The *pain* is generally felt to run from one loin downwards towards the bladder; but it may spread over the whole of the abdomen, or radiate to the chest or to the shoulder-blade, or appear to run along the costal cartilages or the iliac crest. Very often it shoots down into the corresponding testicle, which is drawn up towards the inguinal canal, and is distinctly swollen as well as tender. There may be pain, too, along the inner side of the thigh, with numbness and tingling of the skin. The suffering is often intense; the patient grows faint and cold, and breaks out into a profuse sweat; the pulse becomes frequent and small,† the breathing is quickened, and the temperature may presently rise.

Vomiting and nausea are marked symptoms, and the vomited matters often become bilious after a time. Epileptiform convulsions have occasionally been observed. In pregnant women abortion frequently takes place; cases are recorded in which successive pregnancies have been brought to a premature termination by the supervention of renal colic. The writer had a case of this kind in a patient sent into hospital by Dr Cressy, of Carshalton, in January, 1899.

In the hope of assuaging the pain, patients adopt various and strange positions. One found relief from kneeling with the head bent over, so as

* “I do not lay any great stress upon the use of internal astringent remedies (for hæmorrhages), because it does not appear likely from reasoning that they should do any service, and I am far from being convinced by experiment that they ever do, except perhaps in hæmorrhage of the primæ viæ. They may sometimes have appeared to be attended with success, because there is but a small proportion of hæmorrhages, not owing to internal violence, which would prove fatal though no means were used to stop them” (Heberden). A recent expression of opinion agrees with that of this wise physician more than a hundred years ago: Dr Saundby says of the use of drugs in hæmaturia, “My experience has been that they are all very untrustworthy, and I hesitate to give the preference to any one” (‘Brit. Med. Journ.,’ Dec. 17th, 1887).

† Traube, however, recorded a case in which, during repeated attacks of renal colic, the pulse was slow, full, and remarkably tense. Heberden had remarked the same fact.

to touch the ground; and others lie on the side with the knees drawn up. Movement generally increases the pain, yet the restlessness is so great that it is often impossible to remain in one position. The paroxysm, if it lasts long, is generally interrupted by remissions of the pain, which soon, however, returns as bad as ever. The duration is very variable; it may be over in the course of a few hours, or it may last for several days. Its termination is sometimes quite sudden; the patient, perhaps, during a violent fit of retching, may experience a sensation as though he were stabbed, and from that moment the acute suffering ceases, for the stone has slipped into the bladder.

Micturition during an attack of renal colic is frequent, sometimes with severe strangury, and burning pain in the urethra. As a rule, only a few drops of urine are voided at a time, and they are deeply tinged with blood; but if the kidney on the opposite side is healthy, it may go on pouring out a normal secretion. Ebstein remarks that in cases of calculous pyelitis, in which the urine is habitually discoloured by blood and pus, the fact that it becomes normal when a stone is impacted in the ureter affords valuable evidence that the other kidney is not affected in the same way. But sometimes calculi are present in both kidneys, so that the urine from that which is unobstructed is purulent or blood-stained, or perhaps pale and albuminous—from consecutive Bright's disease (p. 663).

Those cases in which at the time when an attack of renal colic occurs the opposite kidney is absent, or so atrophied as to be unable to secrete any urine, will be separately described in the next section (p. 686).

It is doubtful whether the passage of a calculus is dangerous to life when the other kidney is healthy.*

The subsidence of an attack of renal colic is not a complete proof that the stone has passed into the bladder; for possibly it remains permanently impacted, while the kidney undergoes atrophy or passes into a state of hydronephrosis, as will be presently described (p. 688).

The fact that the pain may cease while its apparent cause is still there, is a strong argument in favour of an opinion expressed above that nephralgia really is due, not to the direct irritation of the mucous membrane by the calculus, but to the peristaltic movements which take place in the over-distended ureter above.

A ureter which has given passage to a stone may be found considerably wider than natural when death occurs from some other cause, after the lapse perhaps of many years; the vesical orifice of the tube may be large enough to admit a thick probe. Such dilatation of the tube explains how when attacks of renal colic are frequently repeated, the later ones are often comparatively slight and of much shorter duration.

Sometimes an attack recurs at more or less regular intervals. Sometimes a calculus is passed by a patient who never before showed any symptoms of urinary disorder; particularly when the stone consists of oxalate of lime. An attack of renal colic occasionally puts an end to pyelitis which had existed for a considerable time; in such a case one may infer that the renal pelvis contained a solitary stone, which was the cause of the previous

* Ebstein speaks of the possibility of its ulcerating through the walls of the ureter and escaping, so as to set up fatal peritonitis. But the case which he cites from Allan Webb ('*Pathologia Indica*,' 1846) was not uncomplicated, for "the vermiform appendix and the ureter were found ulcerated and adherent to one another and to the surrounding structures, and a large amount of pus had escaped from the ulcerated ureter into the abdominal cavity."—C. H. F.

symptoms, and which has now escaped into the bladder. Prout, however, remarks that after the passage of an oxalate calculus, painful sensations often continue to be experienced for some time, so that he had found difficulty in convincing his patient that other calculi did not remain behind.

In making a *diagnosis* we must not suppose that renal colic is in itself proof of the impaction of a stone in the ureter. In some cases (as, for example, in one recorded by Traube, in which the pain for some time returned every night, lasting about five hours) it seems more likely that the attacks are due to a stone in the renal pelvis sticking fast before it can enter the ureter, just as hepatic colic seems sometimes to arise from a gall-stone which has never left the gall-bladder. Moreover, precisely similar seizures may arise from the ureter becoming blocked in other ways; for instance, by a small hydatid, or by a clot of blood, or a fragment of new growth. Again, in a remarkable case recorded by Dr Dickinson in the 'Pathological Society's Transactions' for 1875, an abdominal aneurysm over which the ureter was stretched gave rise for a long period to paroxysms of pain exactly like those of renal colic, and once with swelling of the testicle. In the immense majority of cases, however, renal colic is a symptom of a calculus.

The *treatment* of renal colic consists mainly in the administration of anodynes, though something may perhaps be done to facilitate the release of the calculus. Sir James Simpson had the patient held head downwards in two cases with success, the concretion apparently falling back into the pelvis of the kidney. Venesection was formerly used, and seems to have relieved pain, but it has long been out of fashion. A hot bath is often of signal service, or hot fomentations to the loins; but our chief reliance is on the free use of opium. The hypodermic injection of morphia is to be preferred on account of the rapidity and certainty of its action, and also because vomiting may prevent a draught being retained; or a morphia suppository may be used. The inhalation of chloroform often answers better than anything else, and a hot hip-bath is always a useful adjuvant.

4. *Suppression of urine*.—In the chapter on cholera (vol. i, p. 275), the fact was mentioned that the secretion of urine may, for a time, be completely suppressed, the patient voiding none, and none being found in the bladder when a catheter is passed. Poisoning by turpentine may produce a like effect; and in some remarkable instances it has been observed after an operation upon the urethra, or even after passing a catheter. It is also a symptom of suppurative nephritis, and of the most acute form of Bright's disease. Such cases of "*non-obstructive suppression*" (as Roberts termed them) end fatally in a few hours or in a day or two, unless the kidneys resume their function. In the latter case, the urine that is first passed is scanty, high-coloured, and generally albuminous or even bloody. The best treatment appears to consist in the use of the hot bath, or the application of hot mustard poultices to the loins, and the injection of hot gruel into the rectum.

In other cases, a temporary suppression of urine, without obstruction of the ureters, occurs as part of the general *shock* or collapse produced by a large dose of an irritant poison, or by sudden lesions, such as perforation of the stomach, intestine, or gall-bladder, strangulation of the gut, or rupture of the uterus. The suppression does not add to the danger, and passes off of itself if the patient rallies.

According to Charcot, hysterical women are liable to suppression of urine, which may continue for a long time, without seriously disturbing the health. He describes one of his patients as voiding less than a tea-spoonful of urine on an average each day for weeks together, while in the matters which she vomited urea was present. It is difficult to believe that fraud was not practised in this and like cases of *hysterical ischuria*, although Charcot was convinced to the contrary.

A very different condition is *obstructive suppression* of urine. The patient, instead of dying within a day or two, goes on for seven or eight days without obviously grave symptoms or appearance of danger. In this condition, to follow Sir William Roberts's description, the patient is calm and free from distress, with an unclouded intellect, and with natural pulse, respiration, and temperature. He may be able to take food, the tongue may be clean, and there may be neither nausea nor vomiting. The muscular strength, however, begins to fail, and there is often marked sleeplessness. There is no desire to micturate, and sometimes no urine at all is voided; but generally, at irregular intervals, the bladder discharges a few ounces, or sometimes a pint of urine. This is always pale and watery and of very low specific gravity; and, unless tinged with blood, it is usually free from albumen. At the end of about a week symptoms appear, which, as a rule, lead to a fatal termination, and this within two or three days at the latest. The most distinctive of these are muscular twitchings and contraction of the pupils. The muscular weakness now rapidly increases; and, as a result, the breathing is slow, panting, and laborious. The appetite is entirely lost, and the tongue and the palate become dry. There is increasing drowsiness, with short snatches of sleep, and a little rambling delirium. Convulsions and coma rarely set in, the intellect being commonly preserved to the last, so that the patient has in more than one instance spoken sensibly shortly before his death. Diarrhœa is quite exceptional; and so is severe vomiting. The skin is moist, and often sweats profusely; there is never any ammoniacal or urinous odour from the surface of the skin or with the breath. In one instance slight general anasarca was observed when the suppression first took place, but it passed off entirely on the third day.

The duration of life is stated by Roberts to be, as a rule, from nine to eleven days, and he remarks that the passing of a few ounces, or even of two or three pints, of a dilute urine does not seem to prolong it by more than a few hours. He knew of only three instances in which the patient survived beyond the eleventh day. In one of those cases, that of a man aged sixty-four, recorded by Rayer, death did not occur until the lapse of twenty-five days; another, in a man aged seventy-three, recorded by Sir James Paget ('Clin. Soc. Trans.,' vol. ii), did not prove fatal for twenty-one days; the third, observed by Roberts himself, in a woman aged fifty-six, ended in death on the fifteenth day. The age of the patient does not appear to have any influence in accelerating or retarding the progress of the affection. Recovery has been known to occur in two or three cases in which there had been nearly complete suppression of urine for nine or ten days; in one of them the pupils had become contracted, and there was some mental confusion, but muscular twitchings had not made their appearance.

It was to Sir Wm. Roberts that we owed the first complete account of the symptoms and causes of "obstructive suppression" of urine. But such cases had, of course, been observed before, although their characters had

not been distinguished from those without obstruction. The case recorded by Sir Henry Halford, and cited in 'Watson's Lectures,' must have belonged to this category, although it was much more rapid in its course, having apparently proved fatal in about three days.

An instance of obstructive suppression of urine occurred at Guy's Hospital in the year 1876. A man aged forty-six received a blow on the left side of the abdomen, which was followed by hæmaturia; two days later the urine became entirely suppressed and remained so until he died, seven days after the injury. In the course of the last twenty-four hours the muscles of his face were noticed to twitch, a profuse sweat broke out, and he became unconscious. At the autopsy one unusual feature was observed, namely, suppurative nephritis; and probably this accounts for its having reached a fatal termination more rapidly than usual. But the cause of the suppression of urine was found to be exactly what is stated above, viz. obstruction of the ureter of one kidney by a calculus, when the other one is incapable of secreting urine, owing to some antecedent lesion.

It is conceivable that both ureters might simultaneously be plugged with calculi, or they might both be obstructed by pressure from without, as by cancer of the uterus or by some other disease of the pelvic organs, such as we shall find to produce hydronephrosis. Roberts relates a few cases of this kind which ended fatally, and one in which, after no urine had been secreted for seven days, it flowed again naturally during the remaining four weeks of the patient's life. But in most cases obstruction due to *external* compression, the renal cortex becomes atrophied or destroyed by hydro-nephrosis or consecutive Bright's disease before complete obstruction of the ureters occurs, so that the symptoms and course of obstructive suppression are seldom typical.

The more common cause of obstructive suppression is blocking of the ureter of the only useful kidney possessed by the patient. In such cases the renal pelvis does not become much dilated, and the quantity of urine it contains is by no means large. The kidney in one of Roberts's cases was much congested, but in another it was rather anæmic, though dotted on the surface with numerous blood-spots. In animals, œdema of the kidney, and a deeply ecchymosed state of its pelvis, were observed by Cohnheim after ligature of the ureter. The kidney in the human subject is generally of about twice the normal size, from hypertrophy, owing to the overwork thrown on it by the obsolescence of the other kidney.

As already remarked, whatever urine is formed by a kidney of which the ureter has been blocked, is pale, of low specific gravity, and contains but a small percentage of urea. This is, perhaps, contrary to what one might have imagined to be the probable effect of such an occurrence, but it accords perfectly with the results of the experiments of Hermann upon animals. He showed that in dogs the secretion of urine appears to cease entirely under a pressure of 2·4 inches of mercury, and that when the pressure is removed the result is that a large quantity of watery urine is poured out, in which very little urea is present. Bartels relates the case of a young man who had suffered from previous attacks of renal colic, and who in one such attack had suppression of urine for five days. When this passed off, he voided in twenty-four hours more than 3000 c.c., having a specific gravity of 1001, and containing numerous hyaline casts as well as albumen. Possibly the cessation of the activity of the kidney as soon as the pressure in the ureter and renal pelvis reaches a certain point is more apparent than real, the urine being really secreted, but reabsorbed as fast as it is formed. We can see what an important bearing this view has upon the theory of

uræmia, when taken in connection with the absence of the usual uræmic symptoms in obstructive suppression.

In the *treatment* of suppression of urine, when it appears to be due to impaction of a calculus in one ureter, recourse should be had to those measures which we have seen to be sometimes effectual in aiding its expulsion downwards into the bladder, or its return upwards into the pelvis of the kidney, when there is renal colic. The abdomen in the course of the ureter may be well rubbed and kneaded, while the patient is in various positions,—standing, or lying, or inverted, with his head downwards. But probably the absence of pain in such cases means that the peristaltic movements of the ureter itself have ceased; so that there is little chance of success from such means. In two of Roberts's cases it is expressly noted that soon after the secretion of urine ceased, the pain of which the patient had been complaining disappeared entirely. Consequently it does not appear hopeful to employ hot baths, or chloroform inhalations, or anodynes of any kind, for the purpose of relaxing spasm.

Surgical treatment, however, has met with some success. By cutting down upon the kidney in the loin, and incising the ureter in the renal pelvis, whatever fluid may be collected there will escape, and the removal of pressure is followed by an abundant secretion. Moreover the surgeon may be able to remove an impacted calculus from the ureter. In a case that occurred at Guy's Hospital in 1876, the stone was found after death impacted four inches from the kidney; in two cases recorded by Roberts it lay just within the vesical orifice of the ureter.

A stone has been removed from the latter place, after dilatation of the female urethra, and from the upper part of the ureter either by lumbar nephrotomy, as in probably the earliest successful case by Dr Kirkham, of Downham Market, or by laparotomy, as practised by Mr K. Thornton, or by Langenbeck's method, of which Mr Lane published a successful case (see Jacobson's '*Operations of Surgery*,' and Morris's '*Surgical Diseases of the Kidneys and Ureters*').

A remarkable case of obstructive suppression of urine occurred at Guy's Hospital in 1885, under the care of Mr R. C. Lucas. The patient, a woman of thirty-seven, was first admitted in June, 1885, with a history of frequent hæmaturia, and pain and swelling on the right side of the abdomen. Mr Lucas removed the right kidney, which proved to be a mere shell filled with calculi, with excellent results. The following October the patient was seized with violent left nephralgia and suppression of urine, followed by vomiting, muscular weakness, and at last somnolence, with feeble pulse and temperature of 99°. On the fifth day of suppression Mr Lucas cut down on the left kidney and removed a large impacted calculus. The patient recovered, and was well five years afterwards ('*Med.-Chir. Proc.*,' Jan. 13, 1891).

5. *Unilateral atrophy of the kidney*.—It is not uncommon in the dead-house to find one kidney shrunk to a mere thin flat relic, scarcely larger than the adjacent adrenal, and weighing about an ounce or an ounce and a half. Twenty cases of this kind were collected by Dr Fagge from the records at Guy's Hospital; and the number has since considerably increased. In several instances the cause of death was some disease entirely unconnected with the urinary organs. The secretion of urine, in fact, goes on as before, because the opposite kidney undergoes compensatory hypertrophy, and becomes as heavy as the two organs were together.

The nature of the process by which this enlargement is effected has been studied by different observers with discrepant results. Perls ('*Virchow's Arch.*,' vol. lvi) found increase in size of the convoluted tubes and epithelium, but not of the glomeruli. Beumer (*ibid.*, vol. lxxii) could find no

demonstrable increase in size, whether in the glomeruli or in any of the tubes, in a case in which one kidney was congenitally absent, so that, according to the strict terminology of Virchow, the compensatory change would be a *hyperplasia* rather than a *hypertrophy*, *i. e.* a multiplication rather than an overgrowth of the individual parts. There can be little doubt that the latter view is correct. There is more renal tissue, but the renal tissue is not composed of gigantic elements.

Such a condition is so far dangerous that everything depends on the hypertrophied organ. If its ureter should from any cause become obstructed, the result is suppression of urine, with the symptoms described by Roberts, instead of a mere attack of renal colic. Again, laceration of the region by violence may be followed by fatal results, as in the case of a boy admitted into the accident ward of Guy's Hospital, although during life it appeared a mystery why a unilateral injury should cause death. Possibly, also a kidney enlarged by compensating hypertrophy is particularly liable to Bright's disease; for in about one fourth of our twenty cases there was chronic nephritis of the hypertrophied kidney. And among forty-eight instances of congenital absence of one kidney, collected by Beumer from different sources, there were no fewer than twenty in which the opposite kidney was found diseased—most often from "chronic inflammation," but in many instances it contained calculi in the renal pelvis.

Acquired atrophy of a kidney is due to various causes. In three of the above twenty cases, a calculus was found impacted in the ureter, and in two other cases calculi were in the renal pelvis. In none of the remaining cases was any concretion found, nor was there any obstruction to the outflow of urine from the renal pelvis; yet the pelvis and the calyces were dilated in no fewer than nine of them, and in two the ureter was dilated and thickened all the way down to the bladder. Probably the patient had once suffered from a renal calculus, which escaped through the natural passages after it had deranged the kidney sufficiently to cause it to waste. This conclusion is strengthened by the fact that in two other cases there was a history of lithotomy many years previously; in one of them the operation had been performed by Sir Astley Cooper when the patient was aged thirteen, forty-five years before his death. Lastly, in one instance in which neither the renal pelvis nor the ureter was enlarged, the vesical orifice of that tube was considerably lower than that on the opposite side, and lay nearer to the prostate, as though it had been forced downwards in the expulsion of a concretion.

A very rare effect of the presence of a calculus in the renal pelvis is the replacement of the substance of the kidney by a mass of adipose tissue having the shape of the healthy organ, and of about the same size, as in a case described and figured by Dr Rickards, of Birmingham, in the 'Brit. Med. Journ.' for July 7th, 1883.

6. *Hydronephrosis*.—We have seen that plugging of a ureter by a calculus, or partial obstruction of both ureters as the result of morbid processes of various kinds, does not always lead to any considerable accumulation of fluid in those tubes or in the renal pelvis. There are, however, cases in which such an accumulation occurs, and to these applies the term *hydronephrosis*, originally proposed by Rayer.

Anatomy.—The earliest indication found after death, that a kidney has been subjected to pressure from within, is that the mammillary apices of

the pyramids become separated from each other, and ultimately flattened, or even hollowed out. With the microscope it may be seen that the tubes in the remains of the pyramids are bent into a series of wave-like curves. Meanwhile, the calyces and the whole renal pelvis undergo dilatation. Sometimes the calyces stretch out of the hilus of the organ, so that the pelvis forms a sac situated nearer to the middle line of the body than the kidney; in a case that occurred at Guy's Hospital in 1876, such a sac lying internally to the kidney was found to hold a pint of fluid. Much more frequently, as the calyces and pelvis yield before the pressure of their contents, each calyx forms an oval cavity, communicating with the pelvis by a smooth orifice, and separated from the adjacent calyces by a tough fibrous membrane; the surface of the organ acquires a lobulated appearance, the lobules corresponding in number with these cavities; or, if the sac is very large, the septa between them may become perforated, so that they at last break down and form a single huge loculated cavity.

In the meantime the secreting substance of the organ passes into the condition described as consecutive Bright's disease (p. 663), or undergoes atrophy, until at length no trace of secreting tissue can be discovered, or at most only a few scattered relics here and there in the walls of the sac. If the obstruction is low down, the ureter may be dilated until it is as large as the finger of a glove, or a coil of small intestine.

Secretion.—The nature of the fluid contained in the sac of hydronephrosis varies in different cases. When the enlargement is but slight, as in most instances in which both kidneys are affected, it is still more or less dilute urine, which, however, may contain albumen, or be mixed with pus or blood. In those extreme cases which are generally unilateral the fluid is sometimes pale and clear, sometimes stained with blood. It is usually of lower specific gravity than normal urine, being in this respect like the fluid secreted in cases of "obstructive suppression." But in a remarkable case operated on by Czerny it must have had the characters of healthy urine, for that passed by the patient was in all respects natural; yet extirpation of the hydronephrotic organ was followed by complete and fatal anuria, and on *post-mortem* examination it turned out that the opposite kidney had undergone atrophy, and that its ureter was obliterated.

The solid matters dissolved in this fluid are generally urea, uric acid, and salts of the same composition as those that are found in urine. But in a case that Dr Fagge saw in 1876, neither urea nor uric acid could be detected in the fluid removed by tapping from a tumour believed to be hydronephrotic; and Sir Spencer Wells and Mr Cooper Rose ('Lancet,' 1868) have also met with instances in which urea has been absent. Mr Henry Morris ('Med.-Chir. Trans.,' 1876) cites cases in which the contents of hydronephrotic sacs in the foetus have been devoid of urea. Albumen is commonly present in greater or less quantity. In some cases the fluid has been purulent, as in one described by the writer ('Path. Soc. Trans.,' xxiii) in which six and a half pints of an opaque reddish fluid were drawn off by a trocar; the disease may then be called *pyonephrosis*, if a special name is needed. Dr Dickinson ('Path. Soc. Trans.,' xiii) has recorded a case in which a very large sac contained a gelatinous or colloid substance. In a case under the writer's care, a large renal cyst, when tapped, was found to contain a clear yellowish liquid, which speedily solidified into a uniform gelatinous coagulum.

Etiology.—The causes of hydronephrosis often affect *both kidneys* at once. Double congenital hydronephrosis is not very rare, and depends on malformation of the ureters, or imperforate urethra, often associated with harelip, and seldom compatible with life. The causes of acquired hydronephrosis are stricture of the urethra, enlargement of the prostate, villous and other diseases of the bladder; also pregnancy, prolapsus, or retroflexion or cancer of the uterus, and other pelvic tumours. Cohnheim recorded a remarkable case in a rachitic boy of eleven with contracted pelvis, in whom double hydronephrosis was produced by the pressure of an enormously dilated rectum and sigmoid flexure.

In such cases the hydronephrosis is usually more marked on one side than the other. But there is so much interference with the secretion of both kidneys that death occurs before the sac has become large enough to be recognised as an abdominal tumour during life. Mr Morris, however, relates a case of villous disease of the bladder in which a rounded swelling, of the size of the head of a small foetus, was felt in the right loin. As a rule, the only clinical evidence of the renal affection is a pale watery condition of the urine, until perhaps convulsions or other uræmic symptoms set in, and rapidly bring about a fatal termination.

For example, in 1871 a woman aged thirty-six was lying in the uterine ward of Guy's Hospital with cancer, when she began to complain of severe headache. After two days she screamed out violently in the night, and became unconscious; and in this state she remained until her death three days later. The autopsy showed that the cause of her symptoms was not cerebral hæmorrhage (as had been suspected), but uræmia: each kidney had its pelvis greatly dilated, its pyramids flattened, and its cortex pale, though not decidedly narrowed.

In 1869 a woman aged thirty-eight was admitted into our clinical ward shivering violently and very cold, with a dry brown tongue and other typhoid symptoms, but with her mind clear. She was said to have had prolapse of the uterus for a year, and her urine was found to contain pus. She died two days later, her temperature having been very low throughout. On *post-mortem* examination it was found that the womb had dragged down the vesical extremities of the ureters, and compressed them against the pubic arch. There was hydronephrosis on both sides, and the cortex of each kidney was greatly hypertrophied.

If the cause of the hydronephrosis affects the ureter of *one kidney* only, the opposite kidney undergoes hypertrophy, and, as the secretion of urine may thus be perfectly maintained, there is nothing to prevent the development of the hydronephrosis until a large abdominal tumour is the result.

Of the lesions that may affect one ureter and cause unilateral hydronephrosis, the most obvious is obstruction by an impacted calculus. Thus in 1877 a man aged forty-six died in Guy's Hospital of dropsy due to Bright's disease affecting an hypertrophied left kidney; in the right ureter there was impacted a mulberry concretion an inch and three quarters in circumference; the right kidney was converted into a shining loculated cyst with a smooth lining, upon which there was one little patch of renal substance about as large as a shilling still remaining. It is of course impossible that, after both ureters have been completely blocked by stones, the patient should live long enough to admit of the development of double hydronephrosis. But in 1874 a boy aged six was in the hospital for stone in the bladder, when he died of tonsillitis. Each ureter was greatly dilated, and also the pelvis of each kidney. The right ureter was blocked by a second small calculus about an inch above its orifice; the left was free, so that the distension on that side had to be attributed either to interference with the downward flow of urine resulting from the vesical calculus, or else to the passage of that calculus at a time when the right ureter was free. In 1857

there died in Guy's Hospital a woman aged fifty-six, who had a large fluctuating swelling in the left loin, and a smaller one in the right loin. Hydronephrosis was found to be present on both sides, and the pelvis of each kidney contained calculi, but it was only on the left side that impaction of a calculus in the ureter had taken place. This patient had been passing blood and pus in her urine all the while she was in the ward; but in most cases of hydronephrosis due to impaction of a calculus the urine is perfectly normal, though a history of former attacks of renal colic may perhaps be elicited, sometimes very far back. Rayer recorded a case of hydronephrosis in a man aged sixty-four, who had for a long series of years enjoyed perfect health, but who at the age of twenty-two had suffered from pain in the right kidney and along the ureter, followed by hæmaturia.

Another cause of unilateral hydronephrosis is compression of the ureter, generally near the brim of the pelvis, by a thickened peritoneal band, the result of inflammation of the serous membrane.

Sometimes, again, the ureter is thickened and narrowed by changes in its own wall, forming true stricture, the origin of which is no longer discoverable when the case comes to an autopsy. In a case of the writer's ('*Path. Trans.*,' xxiii), the ureter was found obliterated about an inch and a half below the pelvis of the kidney, and this appeared to be clearly traceable to a kick from a horse about two years previously; the injury had been followed at the time by hæmaturia. A similar instance in which hydronephrosis in a boy of twelve was directly traced to a fall, was recorded by Mr Croft ('*Trans. Clin. Soc.*,' xiv). In 1873 an autopsy in the case of a boy aged four, who died with a calculus in his bladder in Guy's Hospital, showed the ureter as thick as a lead pencil, and completely occluded by an oblique cicatrix about an inch from its origin.

Sometimes, however, no cause for the hydronephrosis can be made out, the ureter appearing perfectly free from obstruction in its whole course from the renal pelvis to the bladder. In many such cases there was probably at some former period a calculus, which underwent disintegration, or was expelled.

It is possible that some other conditions generally regarded as occasional causes of hydronephrosis are not really so, but that here also the true cause is a calculus. One of these hypothetical causes is compression of the ureter by a supernumerary renal artery. Another is obliquity of the origin of the ureter from the renal pelvis, causing a valve-like impediment. That such an appearance is not infrequently met with is certain, and the late Dr Hare recorded ('*Med. Times and Gaz.*,' 1857) a case of hydronephrosis in which the ureter on each side was coiled on itself—like a turn and a half of a cork-screw brought closely together—and adherent to the lower part of the sac. There can be no doubt that a similar condition of the ureter is the cause of the "intermitting" character of many renal tumours, as well as of the fact that after tapping the cyst the ureter sometimes becomes for a time pervious. What seems doubtful is whether cases of this kind are congenital, or whether the twisting of the tube is not a secondary result of its distension, just as one finds the duct of the gall-bladder distorted to an even greater extent and bound down by adhesions, as the result of the passage of gall-stones. Dr Fagge twice saw such a valvular condition of the upper orifice of the ureter when there was obstruction of the lower urinary passages: once in the case of an old man who died of the effects of stricture of the urethra, and in whom, although the ureter was not dilated, the pelvis

of the left kidney formed a large pouch full of dark brown foetid fluid; and again in a fatal case of lithotomy, complicated with stricture.

Dr Sainsbury has described a case of hydronephrosis from two valvular folds of mucous membrane at the origin of the ureter ('Path. Soc. Trans.,' 1886, p. 296). The writer has observed similar folds more or less markedly developed, but not sufficiently to make certain that they would have formed a mechanical obstruction during life.

Cohnheim drew attention to the occasional origin of the ureter from the side of the renal pelvis instead of from its lower end, as a possible cause of hydronephrosis. So long as the patient is in an upright position, the bladder receives only so much urine as overflows from the pelvis of the kidney. He mentions the case of a woman so affected, who for a long time passed scarcely any urine during the day, whereas she voided large quantities at night. This, however, surely proves too much, unless, indeed, the hydronephrosis was large enough to hold several hours' urine; and it must not be forgotten that in renal cirrhosis (including the consecutive form of Bright's disease) the nocturnal flow of urine often exceeds that of the day.

That hydronephrosis is sometimes congenital is well known; some cases in which the abdomen has been large from the time of birth have been prolonged for years, although they far more often terminate fatally within the first few days or weeks. But congenital hydronephrosis is traceable to some definite malformation, such as occlusion of a ureter, or more rarely of the urethra. The cases in question do not lend support to the view that obliquity or twisting of the upper end of the ureter, occurring as a malformation, can give rise to hydronephrosis.

Congenital hydronephrosis is often associated with harelip, imperforate anus, club-foot, and other defects of development. The fact that closure of the outlets of the kidneys causes during intra-uterine life an accumulation of fluid seems to show that their secreting function must already be active; and in a paper read in 1876 ('Med.-Chir. Trans.,' vol. lix, p. 98) Mr Henry Morris has argued for the view that the foetal kidneys normally pour urine into the *liquor amnii*, whence it is absorbed into the blood of the mother, to be afterwards again excreted by her urinary organs.

In an autopsy that occurred at Guy's Hospital, in 1868, Dr Moxon found the left kidney with the pyramids flattened, and the pelvis and calyces dilated. The patient was a man aged twenty-two, who suffered from a lumbar abscess, and lay constantly on his left side with his pelvis raised upon an air-pillow, so that the tendency of fluid to gravitate within the ureter must have been from the bladder to the kidney, and not in the reverse direction.

In two specimens in Guy's Hospital Museum (Nos. 1693, 1694), hydronephrosis appears to have been caused by the pressure of a misplaced renal vein, which ran across the ureter.*

Diagnosis.—Many cases are on record in which *single hydronephrosis* has been mistaken for a large *ovarian cyst*, or even for *ascites*. The most remarkable is perhaps one related by Mr Glass in the 'Philosophical Transactions' for 1747. The patient was a woman aged twenty-three at the time of her death, who had been dropsical from birth; the abdomen then

* One of the cases occurred in a woman of thirty-six, who died twelve hours after admission with eclampsia; the other was a man who died after an operation for sarcoma of the lower jaw.

measured 6 feet 1 inch in circumference, and the sac contained thirty gallons of fluid. In several other instances many pints have been taken from a hydronephrotic tumour during life, or have been found in it on *post-mortem* examination. Among the points which should distinguish such a tumour from an ovarian cyst are its having first made its appearance in the loin and not near the pelvic brim, its having no pelvic connections, the presence of the colon in front of it, and the absence of resonant intestine in the loin. It has, in fact, all the characters of a renal tumour. Fluctuation is generally well marked, and the outline of the swelling is sometimes lobulated. It may occupy a large part or the whole of one side of the abdomen, extending across beyond the umbilicus, and downwards into the iliac fossa.

In a patient under Dr Fagge's care in 1883 there was a prominence in the epigastric and in the left hypochondriac region, while in the loin the bulging was but slight; and as there was obvious pulsation, with an audible bruit, the case looked like one of abdominal aneurysm. In that instance the history given by the patient himself contained one point which, if duly attended to, would have cleared up the diagnosis. He said that on more than one occasion, after the first appearance of the swelling, it had undergone a great diminution in size; he had not, however, noticed that at those times there was any increased flow of urine, nor that the urine was altered in appearance.

This spontaneous subsidence or disappearance of the tumour, when it is observed, is by far the most important clinical character of hydronephrosis. If associated with an excessive discharge of fluid from the bladder it may be said to be pathognomonic. Even without that corroborative evidence the only cases in which a similar occurrence is likely to be met with are those in which an ovarian cyst ruptures into the uterus or into the intestine; and such events are probably always indicated by the escape of fluid through the vagina in the one case, or the entrance of air into the cyst in the other.

Two other affections may be mistaken for single hydronephrosis, namely, *hydatid* of the kidney and a large single *renal cyst*. Each of them is very rare, at least as giving rise to a palpable swelling. The former is almost certain to be set down to hydronephrosis, unless its nature is revealed by the escape of daughter-cysts through the urethra, or by the characters of the fluid removed by paracentesis. The latter could probably be only distinguished after death. Two striking instances were recorded by Mr Cæsar Hawkins ('*Med.-Chir. Trans.*,' xviii) and by Dr Hare ('*Path. Soc. Trans.*,' iv); in each of them the tumour filled the right side of the abdomen. Three or four other cases are cited by Czerny in his list of cases of nephrectomy ('*Trans. Internat. Congress*,' 1881). The writer once met with what was probably a case of the same condition, as a cystic abdominal tumour in a man between forty and fifty.

The patient was an Irish county magistrate accustomed to long journeys and much exposure, a strong, healthy man of forty-five. In June, 1890, he discovered a large lump below the liver, and more or less moveable. On examination in the following November, a round, smooth, tense, and elastic tumour was found behind the hepatic flexure of the colon, and apparently in contact with both liver and kidney. The diagnosis was a hydatid cyst, but on aspiration in February, 1891, the contents were yellowish and albuminous, and coagulated into a gelatinous mass. The urine was healthy at that time, but in the spring of 1890 he suffered from an attack of hæmaturia, and in July, 1892, the cyst had refilled, and when tapped blood-stained serum was drawn off. The patient's general health continued good when last heard of.

Neither pain nor tenderness is constantly present in hydronephrosis, though when the swelling is large it often causes a distressing sensation of distension. In some cases pricking or shooting pains are complained of,

which are perhaps due to local inflammatory changes in the peritoneum covering the sac.

The colon is sometimes tightly stretched over the tumour, in such a way as to interfere with the free passage of its contents; thus in a case recorded by Roberts the chief symptoms were at first those of intestinal obstruction, which recurred again and again during several years.

Double hydronephrosis can only be surmised when the conditions which produce it are present, along with symptoms like those of advanced Bright's disease. Two lumbar tumours are very rarely felt, except in cases of hypertrophic cystic degeneration of the kidneys (p. 665).

Prognosis.—When hydronephrosis is *bilateral*, the patient is always in danger, since the structure, as well as the functions, of the secreting tissue of both kidneys is certainly and incurably impaired.

On the other hand, the course of *unilateral* hydronephrosis is often very chronic, and it scarcely ever brings life to an end by itself. In the case recorded by Mr Glass, death was apparently due to pressure on the diaphragm and displacement of the thoracic viscera. In one observed by Mr Thompson, of Nottingham ('Path. Trans.,' xiii), it resulted from peritonitis set up by escape of the contents of the sac through an ulcerated aperture. In the writer's case of traumatic stricture of the ureter (p. 691) there had been communication with some part of the intestine, for the distended pelvis had suppurated and contained a mass of vegetable fibre, with bits of apple-core and part of a clove. In other fatal cases that have been recorded the cause of death has generally been either an independent malady (as, for example, acute tuberculosis in a case of Dr Hillier's) or else the supervention of disease in the hypertrophied kidney on the opposite side of the body. Consequently, it is not advisable to interfere actively with hydronephrosis until the patient can no longer endure the discomfort.

Treatment.—In some few instances the sac has been emptied by rubbing the abdomen. Roberts relates the case of a girl of eight, who came under his care with a soft fluctuating tumour in the left side, of about the size of a child's head. This was diligently manipulated in every direction on alternate mornings. After the third time she suddenly passed abundant urine, the tumour forthwith subsided, and did not reappear while she remained under observation. A somewhat similar result was attained in a case recorded by Dr Broadbent ('Path. Soc. Trans.,' xvi) of double congenital hydronephrosis in an infant.

But when the sac is tense, little can be hoped for from such a procedure; and there is often so much tenderness that it cannot be adopted. The only treatment then is to puncture the sac with a trocar. On the left side this may be done at a spot just anterior to the last intercostal space. But on the right side Mr Morris has shown ('Med.-Chir. Trans.,' lix) that there is danger of wounding the liver, and he advises that a point should be selected halfway between the last rib and the crest of the ilium, and from two inches to two and a half inches behind the anterior superior spine. After the operation, fluid like that which has been withdrawn from the tumour sometimes passes for a time with the urine, showing that the ureter has again become pervious. But the sac almost always rapidly fills again, and may soon regain the same size as before. Thus, in a case of double hydronephrosis, which was three times tapped by Fränkel, the patient did not micturate at all during from twelve to forty-eight hours after each tapping, the whole of the fluid secreted in the interval having doubtless

accumulated in the two sacs. It is true that unilateral hydronephrosis is commonly attended with such extreme destruction of the renal cortex that the organ can hardly be supposed still capable of forming urine; but experience seems to show that even in such cases fluid continues to be poured out into the sac by a process of transudation like that which occurs in extreme cystic degeneration (p. 665).

One of the few cases in which repeated puncture has led to permanent shrinking is that of Mr Croft already referred to ('Trans. Clin. Soc.,' xiv). In that instance, within fifty-four days of the accident which caused the disease, seventy-nine ounces of fluid had already collected. Paracentesis was performed eight times altogether, from three to four pints being removed each time. After the eighth operation, which was performed at three months' interval from the first, no further accumulation took place. In a case observed by Sir Spencer Wells ('Dubl. Quart. Journ.,' 1867) the patient, two months after a second tapping, passed two calculi *per urethram*, after which the tumour completely disappeared and did not return.

There does not appear to be much fear of setting up suppuration in the sac by paracentesis, though this result has been known to follow the attempt to cure the disease by making a fistulous opening ('Path. Soc. Trans.,' xiii, Dr Little's case). Czerny mentions ('Trans. Internat. Congress, 1881') the case of a man in whom Gustav Simon had two years previously made such an opening, and who, in 1881, was still acting as an attendant in the hospital wards at Heidelberg.

When tapping proves ineffectual, the bolder operation of removing the distended kidney has been frequently performed, and often with complete success.

Czerny, in his statistics of nephrectomy (*loc. cit.*, p. 249), gives twelve cases in which that operation has been performed for hydronephrosis or for cyst of the kidney. Seven of them ended fatally, but this high mortality may perhaps be in part attributed to the fact that in five an erroneous diagnosis of ovarian tumour had been made. According to Mr Barker a lumbar incision is in cases of this kind preferable to one in the front of the abdomen. Many, published and unpublished, cases show the importance of ascertaining that the opposite kidney retains its functional integrity. Probably this may be best done by making a preliminary opening into the sac and allowing it to drain, so that after making certain that no fluid from it any longer passes into the ureter, one can measure and test the urine which reaches the bladder from the other organ.

7. Pyuria and pyelitis.—The presence of a stone in the kidney often causes pus to appear in the urine from suppuration of the renal pelvis. As in the case of hæmaturia, it will be well to consider *pyuria* generally, and briefly to indicate the various affections that may lead to this symptom.

Urine which contains pus has a turbid, opaque appearance, and on standing throws down a dense whitish-yellow sediment which somewhat resembles the white gravel formed by amorphous earthy phosphates, but may be readily distinguished by the microscope. Moreover, it remains undissolved after the addition of an acid; and when caustic alkali is added, it forms a transparent gelatinous mass, which hangs in long strings when poured from one vessel to another.* In ammoniacal urine, pus forms a

* This chemical test for pus is in England associated with the name of Babington, though Leube attributes it to Donné.

viscid, tenacious substance, which glides out as a coherent mass when the vessel is emptied, and often causes much pain and distress in passing through the urethra.

When acids are added to purulent urine, the pus gradually falls to the bottom of the test-tube as an insoluble sediment.

It is an interesting fact that leucocytes in urine, even when it is alkaline and full of bacteria, sometimes retain their amoeboid movements on a warm stage.

Whenever there is pus in urine, there is also albumen, derived from the liquor puris. But if the quantity of pus is small, the albumen may not be discoverable by ordinary tests. It is often an important practical question to determine whether the amount of albumen observed in purulent urine is or is not greater than the pus itself accounts for; since, if it is greater, it affords evidence of the existence of Bright's disease in addition to the affection causing the pyuria. In various surgical affections of the urinary organs the propriety of operative interference depends chiefly upon this point.* If heat gives a decided precipitate after the pus has been allowed to subside, it is likely that the albumen is due to an additional cause. Tube-casts should also be carefully looked for.

Apart from calculus, pyuria may be due to a great variety of affections. The possible presence of *gonorrhœa* must never be forgotten; nor, in females, that of *leucorrhœa*, which, however, is indicated by a large number of squamous epithelial cells, as well as of leucocytes, under the microscope. Again, *cystitis* is a frequent cause, and one should remember that there may sometimes be pus in the urine from this cause without the patient complaining of much pain or having to micturate very frequently, especially if he has a stricture or an enlarged prostate.

Whatever may be the origin of cystitis, it is apt sooner or later to extend upwards as pyelitis; and suppuration of one or both kidneys is often the last of the wide-spread changes that follow ammoniacal decomposition of the urine in the bladder, from stricture, prostatic swelling, or stone. Again, pyuria may be the result of nephritis from cantharides or turpentine.

None of these causes, however, produce persistent and severe pyelitis independently of cystitis or urethritis. So that if we exclude tuberculous cases (which will be described in the next chapter) we need admit no other direct cause of pyelitis than gravel or calculus.

A case of pyuria was under Dr Fagge's observation for several years. The patient felt, in 1876, a slight pain or uneasy sensation in the left loin, for which no cause could be found. A short time afterwards he noticed some blood in his urine; but, on taking medicines which rendered it alkaline, the hæmaturia ceased. Yet from that time the urine almost constantly contained pus in small quantity, with apparently an excess of albumen; and crystals of oxalate of lime were usually to be detected, sometimes crystals of lithic acid. In 1880 he passed a small oxalate calculus, after which he was more free from pain than he had been for some years previously. All along the general health was good, and the patient was able to discharge responsible duties.

The symptoms that characterise "calculous pyelitis" are *pain* in the loin or in the abdomen, *hæmaturia* which generally recurs from time to time, and more or less constant *pyuria*.

* Leube, having added to urine 2 per cent. of pus, found that in every microscopic field, prepared with fluid that had not been allowed to settle, there were from ten to fifteen leucocytes, and that the amount of albumen precipitated by boiling occupied about one tenth of the bulk of the urine. His conclusion is that a precipitate of even one twentieth or one twenty-fifth is more than can be attributed to pus, unless at least some few pus-corpuscles are visible in each microscopic field.

Frequently rigors recur from time to time, sometimes with regular quotidian periodicity. There is often considerable pyrexia, which may assume a hectic type. Diarrhoea may be persistent and intractable; or there may be obstinate constipation from adhesion of the colon to the anterior surface of the affected kidney. When pyelitis runs on for a length of time the renal pelvis often becomes dilated into a large sac, which may be felt as an abdominal tumour. It may bulge into the loin as an elastic fluctuating mass, very painful and tender to the touch, or may push the liver upwards. If the ureter becomes from time to time blocked, this swelling may present great variations in size on different occasions, and there may be converse variations in the degree of pyuria, the urine being clear when the swelling is largest, whereas a subsidence of the tumour is accompanied by the escape of several ounces of pus into the bladder. In such cases of "pyonephrosis" the renal cortex probably always undergoes atrophy, or becomes shrunken by a process of consecutive Bright's disease. If there are calculi in both kidneys, as is often the case, pyelitis and its results are sufficient to destroy life, with symptoms of uræmia. And even when the affection is limited to one side, the opposite kidney may, after undergoing hypertrophy, become affected with Bright's disease, either as the result of lardaceous changes in it, or independently.

Nevertheless in other cases, after lasting for many months, the suppuration subsides, the kidney shrinks, and at last dries up into a putty-like mass, which troubles the patient no more.

Among the complications of pyelitis is the supervention of *perinephritic abscess*. Inflammation probably never affects the renal pelvis for a long time, nor with great severity, without spreading to the surrounding structures, which become indurated and matted together by new fibrous material; but sometimes the mucous membrane undergoes ulceration, and perforation takes place, with escape of urine and of pus into the connective tissue. When this occurs, there is usually a marked increase in the pyrexia and in the other general symptoms that have previously resulted from the pyelitis. A fluctuating swelling may appear in the loins, with extreme local tenderness; and ultimately the skin may become reddened, and the abscess, if not opened by the surgeon, may point and break of its own accord. In other cases the course taken by the pus is to enter the sheath of the psoas muscle and make its way downwards into the groin, or penetrate the hip-joint.

A point on which Trousseau laid stress is that when the psoas is affected the thigh is kept more or less rigidly flexed upon the pelvis; but this is also true of a psoas abscess due to vertebral caries or other cause than renal suppuration. Sometimes the pus may point above Poupart's ligament; or may rupture into the intestine, so that gas and fæcal matter escape into the abscess cavity, or subcutaneous emphysema appears in the back, as was twice observed by Trousseau. Lastly, a perinephritic abscess has been known to burrow through the diaphragm and the lung, and discharge itself by the bronchial tubes.

Conversely, an abscess starting from a vertebra, a lymph-gland, or some other abdominal viscus may make its way into the urinary passages.

When there is free discharge of pus in the loin the inflammation sometimes gradually subsides, and recovery ensues. But, as a rule, the prognosis of perinephritic abscess is unfavourable, the patient being at length worn out by the drain of pus, by pain and hectic fever, or by lardaceous disease.

8. *Suppurative nephritis*.—This begins in the cortex of the kidney, as minute round or irregular dots, and on section of the organ as streaks or lines, traversing the cortex to a greater or less depth, or running continuously through it and through the medulla. At an advanced period there is well-formed creamy pus; but when the disease is earlier fatal, there is often only a soft pinkish-white material, which consists of kidney-tissue infiltrated with leucocytes. Surrounding the infiltrated or suppurating tracts there is much vascular injection. Sometimes only one or two points of even commencing suppuration are discoverable, so that they are not unlikely to be overlooked. Probably the recognition of even a single point of pus in the renal cortex at an autopsy proves the existence of a septic infection sufficient to account for death.

The *causes* of suppurative nephritis vary. Sometimes it occurs as part of general pyæmia; and Moxon noticed that in cases of pyæmia resulting from perinæal section or lithotomy, abscesses in the kidneys were more apt to occur than when pyæmia was due to lesions unconnected with the urinary organs. Suppurative nephritis is often obviously traceable to an infective process, spreading upwards from the bladder along the ureters, and due to the presence of pyogenic micrococci. Dr Goodhart, in vol. xix of the 'Guy's Hospital Reports,' traced a connection between renal suppuration and the presence of erysipelas in the same ward at the same time.

As a matter of fact, the remarkable decline in the frequency of pyæmia of recent years has been accompanied by a corresponding decline in the frequency of suppurative nephritis.

In some rare cases suppuration of the kidney may possibly occur as a primary morbid process.* But in the vast majority of instances suppurative nephritis is secondary to one of the common surgical diseases of the urethra or of the bladder, or to paralysis of the bladder from some spinal lesion. The greater frequency of cystitis in men explains why pyelo-nephritis is more common in them than in women. As might be expected, most cases of this kind are also marked by the mechanical effects of obstruction to the outflow of urine from the renal pelvis, on one or both sides, which have been fully described already. The suppurative nephritis itself is not always bilateral. In some instances the whole length of the urinary tract is obviously affected with inflammation, from the bladder to the mucous membrane covering the renal pyramids; but in others the lining of the ureter and of the renal pelvis is normal. Dr Dickinson believes ('Med.-Chir. Trans.,' lvi) that the exciting cause of the nephritis is the ammoniacal state of the urine resulting from its decomposition within the urinary passages, and holds that suppurative nephritis never occurs except when the urine has undergone this change. How rapidly the disease may develop itself is well shown by a case which he narrates of an old woman admitted into the hospital for a fracture of the femur, who two days later became unable to pass her water, so that a catheter had to be used. The urine drawn off was then natural, but very soon afterwards it became offensive, and death occurred within a week of the accident, three days after the urine had changed its character. Both kidneys were found to be suppurating.

As a rule, suppuration of the kidney is secondary to pyelitis, and that again to calculus or to local infective suppuration of the bladder. When

* Dr Goodhart's paper recorded three instances, in each of which, although some degree of cystitis was found at the autopsy, it seemed doubtful whether this was sufficient to account for the renal suppuration; one was a case of enteric fever, another of mitral disease, and the third of extensive burns.

this is not the case there is no pyuria, and the recognition of the condition is much more difficult.

The *symptoms* of suppurative nephritis are obscure. Rigors are an early and frequent symptom; febrile symptoms rapidly follow, with vomiting, great prostration, feeble pulse, a mawkish smell in the breath, dry, brown tongue, and sometimes profuse sweating or diarrhœa. Convulsions are very rare, and the case ends in stupor rather than coma.

But the disease is often latent, so that the patient may die quite unexpectedly, without either febrile or cerebral symptoms.

The state of the urine throws but little light upon the *diagnosis* of suppurative nephritis. There may be a large quantity of pus present, but this is probably the result of the pyelitis and of the cystitis which are generally present at the same time. What we depend on is the recognition of the general symptoms of sepsis, and the presence of some source of pyogenic infection either in the urinary organs or elsewhere.

It might be thought that the determination of the amount of urea in the urine would throw light upon the state of the kidneys in these doubtful cases; but this expectation appears not to be verified by experience.

Dr Goodhart ('Guy's Hosp. Rep.,' xix) records two instances in which he had made quantitative analyses shortly before death, and found that the renal secretion contained thirteen or fourteen grains of urea to the ounce; a third patient passed in the twenty-four hours three pints of urine, with a total quantity of 592 grains of urea; and a fourth patient thirty ounces with 328 grains of urea. On the other hand, a man, who afterwards went out well, having had his bladder punctured *per rectum*, passed thirty ounces in the twenty-four hours with only 295 grains of urea, or less than ten grains per ounce.

The *treatment* of pyelitis, or of suppurative nephritis, depends in calculus cases chiefly on successful nephrolithotomy. If this is not possible we must promote a freer flow of urine by the administration of diluents, and of the acetate or citrate of potash. The bladder should be washed out with boracic acid lotion, and benzoate of sodium or ammonium in ten-grain doses be given, with buchu or uva ursi; or, perhaps, a recently introduced compound, named Urotropine, which has great control over vesical decomposition. As Mr Morris says, the catheter should never be employed except when the patient—during, and for some time after the passage of the instrument—is in the equable temperature of bed.

When, apart from a calculus, an abscess has formed in the kidney, the only successful treatment is to incise and drain it; and such treatment in the hands of modern surgeons often leads to satisfactory results.

Treatment of renal calculus.—The improved knowledge of the composition and chemical properties of renal and vesical calculi, which marked the first half of the nineteenth century, did not lead to improved therapeutics. On the contrary, previous attempts to dissolve calculi were almost entirely given up. In the middle of the eighteenth century the solvent treatment of calculi was investigated by the Rev. Stephen Hales and Mr David Hartley; the experiments were suggested by Cheselden, and carried on by Mr Sharp at Guy's Hospital and Mr Gardiner at St George's Hospital. Injection of soap-ley into the bladder was commonly practised in Heberden's time, and soft soap was the chief ingredient of the nostrum for which Parliament paid £5000 in 1739.

Sir Wm. Roberts reintroduced a *solvent method* which, at least for calculi of lithic acid, deserves a full trial before more radical measures are adopted ('Med.-Chir. Trans.,' 1866).

He began by making a careful series of experiments outside the human body, exposing calculi to the action of a slow stream of a solution of carbonate of potass, which proved to be more effective than the carbonate of soda. With a liquid containing from forty to sixty grains of the alkali to the pint, he found that stones lost from 15 to 20 per cent. of their weight in twenty-four hours. Even with liquids containing twenty or thirty grains to the pint the solvent action was considerable. But what is very remarkable is that above the strength of sixty grains solution ceased, in consequence of the formation of a tenacious white crust of potassium biurate on the surface of the concretion. The next step was to ascertain what doses of the vegetable salts of potass would give to the urine alkalinity equivalent to about fifty grains of carbonate in the pint; and it was found that this could be effected in adults by the administration of forty to sixty grains of the acetate or citrate, dissolved in three or four ounces of water, every three hours; in children by about half the quantity. Some patients find that the acetate agrees with them better than the citrate, in others the reverse is the case.*

It is not to be supposed that the urine passed by patients taking such doses of the potass salts can be maintained at a constant degree of alkalinity. On the contrary, it varies from hour to hour; but Roberts has found experimentally that such urine, when it is allowed to pass over a uric acid stone outside the body at blood-heat, dissolves it at the mean rate of twelve and a half grains in the twenty-four hours. Clinically, it is obvious that the best proof of the power of urine, when rendered alkaline in this manner, to act upon calculi within the body, is to be obtained in the case of vesical calculi; because their presence and their approximate size can be determined by sounding before treatment is begun, and they can, if necessary, be removed by lithotomy afterwards.

In one case of uric acid stone in the bladder, Roberts, after thirty-nine days' treatment, obtained the clearest evidence that a solvent action had been exerted. At the end of that time lithotomy was performed, and the stone was found to be eroded to a considerable extent, so that an incomplete layer of oxalate of lime was exposed, part of which was actually undermined. The proof of the efficacy of such treatment in the case of renal calculi is necessarily less complete; but there is a strong presumption in its favour.

The following case was reported by Dr Fagge:

A man came with a number of little uric acid calculi which he had been passing frequently. A vegetable salt of potass was prescribed, and a fortnight later he brought a single concretion, the only one he had passed, coated over with a white layer, which looked like phosphates, but which may have consisted of the biurate of potash. There seems to be little doubt that many other concretions must have been dissolved, for all the renal symptoms which had been troubling him disappeared, and he voided no more calculi.

The great drawback to this solvent treatment is that it is useless when a stone consists of oxalate of lime; or, in the case of mixed calculi, as soon as a complete layer of the oxalate is reached. Some have feared that making the urine alkaline may lead to a deposition of phosphates, and so augment the size of a calculus. But Roberts showed that so long as the alkalinity is due to a fixed base, there is no danger of this result; and as a matter of experience, he found that after the treatment has been continuously carried out for three months, an oxalate of lime calculus in the bladder remained entirely free from phosphatic incrustation. On the other hand, in the

* As the citrate of potass of the shops is apt to be impure, Roberts advised that it should be prepared by neutralising a solution of the bicarbonate with crystallised citric acid; the following formula yields sixty grains of the citrate to the ounce:—℞ Potass. Bicarb. ʒxiij; Acid. Citric. ʒviij gr. xxiv; Aq. ad ʒxiij.

experiments already referred to, in which uric acid calculi were exposed outside the body to a slow stream of urine rendered alkaline by fixed alkali, and in which the calculi underwent solution, it was ascertained that as soon as ammoniacal decomposition of the urine occurred, a layer of mixed phosphates was deposited, and all further solvent action ceased. It is therefore useless to attempt a solvent plan of treatment if the urine is ammoniacal. But even when putrefaction of the urine within the urinary passages has begun, the administration of benzoate or salicylate of soda may arrest it, and restore the natural acidity, so as to bring the case again within the scope of solvent remedies.*

When the presence of an oxalate calculus is suspected, the only prospect of cure apart from surgical operation lies in the possibility that it may either pass down the ureter and be voided, or else become "encysted," so as to cause no further symptoms. The possibility of the latter occurrence was clearly stated by Dr Rees in his 'Croonian Lectures' for 1856.

Operative treatment.—As in cases of intestinal obstruction and of gall-stones, so in those of renal calculi and pyelitis, abdominal surgery has, since the introduction of antiseptic precautions, become justly bolder than before, and the results obtained are scarcely less brilliant than in ovariotomy. The operations are *nephrotomy*, or incision of a hydronephrotic or suppurating kidney with subsequent drainage; *nephrolithotomy*, or removal of a renal calculus from the pelvis; and *nephrectomy*, or removal of the entire diseased kidney.

In all cases of protracted and severe pyelitis the question of surgical interference must be taken into consideration, and when decided, the operation should not be delayed; for the chances of recovery are much greater at an early period of the disease than when it is far advanced. If there is an abscess in the loin, there is no doubt of the advantage of thoroughly exploring it, and of searching for and removing any calculi that may be present; and when there is evidence of pyonephrosis it is almost always advisable to cut down upon the kidney in the loin, so as to allow of its drainage. How successful this operation may sometimes be is well shown by a case related by Rosenberger, of Würzburg ('Trans. Internat. Congress,' 1881). The patient, a medical man, had during the previous year been incised above the ilium, with discharge of several pints of offensive pus, and he was reduced to a skeleton when the lumbar operation was performed. Yet, the cavity having been washed out with solution of phenol and a drainage-tube inserted, he gradually regained his health and resumed his practice.†

Nephrectomy, or excision of the kidney, is a more formidable operation. When there is a large pyonephric sac, a lumbar incision may fail to give room for its extirpation—as was pointed out by Mr Howard Marsh ('Trans. Clin. Soc.,' 1882). Another difficulty is illustrated by two cases of Mr Barker's ('Med.-Chir. Trans.,' lxiv); in each of them the kidney was found to be surrounded by a mass of dense vascular tissue, which could not be removed. Moreover, we cannot be sure of the condition of the opposite

* Unfortunately, among adults of middle age, the frequency of oxalate of lime calculi in the kidney, compared with those of uric acid, is greater than would appear from statements based on museum specimens obtained by lithotomy, or other vesical calculi.

† If a large branched calculus is found occupying the renal pelvis, the attempt to extract it may be as dangerous as the complete removal of the kidney. A case of this kind, which occurred to Mr Marrant Baker ('Trans. Internat. Congress,' 1881), proved quickly fatal by shock and by hæmorrhage from the walls of the dilated renal pelvis.

kidney. Cases have been recorded in which fatal suppression of urine has occurred; for the organ affected with calculous pyelitis, of such severity as to justify its extirpation, was nevertheless the only functionally active kidney which the patient possessed. To obviate this risk Czerny advised making two stages of the operation, first opening a urinary fistula, and after an interval proceeding to nephrectomy. Another suggestion, made by Simon, of Heidelberg, a pioneer in renal surgery, is that in female patients, after dilating the urethra, a catheter be put into each ureter separately. Dr Teichmann, who for several years carried on investigations on this point in the *post-mortem* room at Guy's Hospital, believes that even in the male subject he can, with an instrument introduced along the urethra, nip up the mouth of each ureter in turn, and so withdraw from the bladder the secretion of each kidney separately.

Nephrolithotomy.—When no lumbar abscess or even pyelitis is present, cutting down upon a kidney with the object of removing a calculus—a procedure which was condemned by Sir Benjamin Brodie as dangerous and absurd—is now proved to be feasible. The first successful operation was by Mr Henry Morris at the Middlesex Hospital ('Clin. Trans.,' xiv) in 1880.

The patient, a girl aged nineteen, was admitted under the care of Dr Coupland. She had for several years been liable to severe paroxysmal pain in the right lumbar region, which made her life as a domestic servant unendurable, and for at least two years her urine had often contained blood. Mr Morris cut down upon the kidney, and with his forefinger almost at once detected "something rounded, about the size of the uncut end of a pencil, causing a slight irregularity of the surface of the organ at a spot just a little behind the hilus." With a bistoury he incised the kidney at this spot, and succeeded in removing a calculus, which weighed thirty-one grains, and consisted of oxalate of lime. The patient made a good recovery.

In vol. xv of the 'Clinical Society's Transactions' two similar cases are recorded, each of which was no less successful than that of Mr Morris. One, by the late Mr Marcus Beck, is that of a young man aged nineteen, who had suffered for twelve years from symptoms of stone in the left kidney, some pain in the loin increased by movement, hæmaturia, and great irritability of the bladder. Mr Beck exposed the kidney, and on thrusting a darning-needle into the organ, a stone was at once felt and extracted. It consisted of alternating layers of uric acid and of phosphates. In the other case—Mr Butlin's—the symptoms were neuralgia of the right testicle, which had continued for ten or twelve years, with some pain in the loin; but the urine never contained either pus or blood, though crystals of oxalate of lime were almost always present, and often a trace of albumen. The kidney having been exposed, a hard body was felt with the finger, and removed; it proved to be an oxalate of lime calculus. In vol. xvi of the 'Clinical Society's Transactions' there is a fourth case of successful nephrolithotomy (the stone weighing 473 grains) by Mr May, of Birmingham, and a fifth by Mr Howse. (See also the same 'Transactions' for 1884, 1885, and 1887.)

The chief difficulty is that of diagnosing with sufficient certainty that the stone is present, and that it is too large to pass down the ureter into the bladder. Mr Morris cites seven instances, in each of which an incision down to the kidney has been made without any stone being detected. See cases of this operation and of nephrectomy by Prof. Czerny, Mr Baker, Mr Lucas, Mr Barwell ('Internat. Cong.,' 1881, pp. 242—279), and the late Mr Thos. Jones, of Manchester ('Brit. Med. Journ.,' June 2nd, 1883); also the report of a discussion on Renal Surgery at Leeds in 1889 ('Brit. Med.

Journ.,' ii, 1881); and Mr Henry Morris's monograph on 'Surgical Diseases of the Kidneys,' second edition, 1900.

One of the most remarkable examples of this branch of surgery was Mr Lucas's nephrolithotomy of one kidney following nephrectomy of the other and ending in the patient's recovery; it was referred to above (p. 687).

A collection of 233 cases of nephrotomy, nephrolithotomy, and nephrectomy was published by Dr Samuel W. Gross in 1885 (fourth edition of S. D. Gross on 'Diseases of Urinary Bladder'), and a still larger one of 327 cases, in the following year, by Mons. Brodeur ('De l'Intervention chirurgicale dans les Affections du Rein,' 1886). The total mortality of these operations, including those practised from the loin and those practised by median abdominal section, has been reckoned at between 40 and 45 per cent.; but every five years witnesses an improvement in their success.

TUBERCLE, CANCER, PARASITES AND ABNORMALITIES OF THE KIDNEYS

“When death’s pale horse runs away with a person on full speed, an active physician may possibly give them a turn; but if he carries them on an even slow pace, down hill too, no care nor skill can save them.”—JOHNSON.

Fatty and lardaceous degeneration—hypertrophy and atrophy of the kidneys.

Tuberculous pyelitis—Its pathology—Symptoms, causes, and treatment.

Malignant disease of kidney—Sarcoma—Carcinoma—Symptoms—Nephrectomy.

Hydatids of the kidney—Chyluria and Filaria sanguinis—Endemic hæmaturia and Bilharzia hæmatobia.

Renal malformations—Floating and moveable kidneys.

THE present chapter completes the subject of renal diseases by dealing with degenerations, tubercle, new growths, parasitic affections, and displacements of the kidney.

Fatty degeneration of the kidney.—This is a not infrequent condition, and is usually met with in association with fatty degeneration of the liver and fatty overgrowth of the heart. The organs are smooth, pale, and greasy, and microscopically the secreting epithelium is found loaded with oil-drops. This fatty degeneration produces no clinical symptoms whatever, although it was once erroneously described as a form of Bright’s disease (*cf. supra*, p. 640). It is sometimes found in cases of diabetes and fatal anæmia, and is constantly present as the result of fatal poisoning by phosphorus.

Lardaceous degeneration, on the other hand, always produces tubal nephritis, and is marked by the symptoms above described as a special form of Bright’s disease (*supra*, pp. 649-52).

Pure renal *hypertrophy* is found as a compensatory overgrowth of one kidney when the other has been destroyed, and affecting both kidneys in cases of diabetes, of intemperance, and of polyuria generally.

Renal *atrophy*, independent of cirrhosis and cystic degeneration, is always secondary to obstruction of the ureter, by a calculus or otherwise; and it only affects one of the kidneys. Occasionally, however, we discover atrophy of a kidney as we do hydronephrosis, or the two combined, where we find no calculus or other cause, and can only suppose that whatever once obstructed the flow of urine has since disappeared (*cf. p. 691*).

TUBERCULOUS DISEASE OF THE KIDNEY.*—In cases of general tuberculosis, miliary tubercles are found after death in the kidneys, as in other organs:

* *Synonyms.*—Tuberculous pyelitis—Phthisis renalis.

but they are not known to produce any physiological disturbance, nor do they affect the course of the disease. The urine is usually free from albumen, and even when a trace is present it is probably due to fever and not to any local change; in fact there are no symptoms pointing to the kidneys during life. The tubercles are sometimes minute, grey, and translucent, but more often they are yellow and opaque, as large as those of the liver, and much larger than the tubercles of the pia mater. Both varieties are far more common in children than in adults, and are never found without tuberculous disease of the spleen, lungs, lymph-glands, or bones.

The disease now to be described is clinically very different. It consists in the gradual destruction of the kidney, generally of one side only, by the formation of cavities with caseous walls, which may fairly be called *vomicæ*, for they resemble *vomicæ* in the lungs. The same difference of opinion once prevailed about this "nephro-phthisis" as about the corresponding pulmonary disease. Many pathologists refused to recognise it as tuberculous, and called it primary "caseous inflammation;" but the best authorities in England always held to Laennec's original doctrine that yellow caseous tubercle is as much true tubercle as the grey granulations of Bayle. Since the discovery of the tubercle bacillus, the truth of this view has been established, not only for the so-called "caseous" and "fibroid" forms of phthisis, but also for the corresponding "scrofulous" inflammation of the lymph-glands, the testes, and the kidney.

Anatomy.—It has been questioned whether tuberculous disease of the kidney has its starting-point in the cortex or in the medulla of a pyramid. The following observations by Dr Fagge show that it may begin in either.

(1) In 1874 a woman aged twenty-six died in Guy's Hospital of phthisis. One kidney had in its cortex a cluster of yellow tubercles, from which a yellow streak extended down the corresponding pyramid.

(2) In 1873 a girl aged six died of tubercular peritonitis. In one kidney, near the apex of a pyramid, was a round tubercle just beginning to soften; the mucous membrane of the pelvis of the other kidney was covered with tubercles, as was also the lining membrane of the bladder.

(3) In the same year another girl, aged four and a half years, died of acute tuberculosis, the bronchial glands being caseous. In a pyramid of one kidney, not quite reaching either its free surface or its base, there was a well-marked *vomica*, with an indurated caseous border.

(4) In the same year a man aged forty died of phthisis, with caries of the spine. In one kidney there was early tuberculous disease, ulcerating so as to form a conical cavity; in the adjacent part of the cortex there were caseous nodules up to the size of swan-shot, some extending to the surface of the organ.

(5) In 1863 a boy aged fifteen, who had been admitted for vesical symptoms, died of tubercular meningitis. Both kidneys contained "softening tuberculous matter as well as distinct tubercles;" in the pelvic mucosa of the right kidney there were well-marked isolated tubercles, and also in that of the corresponding ureter and of the bladder, near its neck.

(6) In the same year a man aged twenty-two died of acute general tuberculosis and tubercular meningitis. The kidneys were stuffed with soft yellow tubercles, in some places collected in groups, and apparently about to soften into abscesses; the pelvis of the right kidney was lined with a layer of granular lymph, and this extended down the ureter into the bladder, which itself was affected with tuberculous ulceration; all round the opening of the right ureter into the bladder the mucous membrane was covered with isolated tubercles of various sizes.

(7) In 1876 a man aged thirty-four died of bronchitis and emphysema, the lungs being quite free from tuberculous lesions. In one kidney a single pyramid was eaten away at the tip, and the rest of it was changed into a gelatinous material of sulphur-yellow colour.

(8) In 1875 a young man of twenty-three died of pleurisy and of tubercular disease of the lung. In the substance of a single pyramid of one kidney there was an early patch of caseous infiltration.

(9) In 1878 a woman aged twenty-eight died of phthisis. One kidney contained a

circumscribed cheesy mass the size of a damson, and two of its pyramids were also affected with early tuberculous lesions.

(10) In 1879 a youth aged nineteen died of spinal disease with psoas abscess. One kidney showed several early tubercular masses excavating the cortex and forming vomicae with cheesy walls; in the mucous membrane of the pelvis there were also scattered grey tubercles and caseating patches.

(11) In 1880 a woman aged twenty-five died of bronchitis. In one kidney there were two typical vomicae with cheesy walls.

(12) In the same year a man died of lardaceous disease of the viscera, the result of caries of the spine. In one kidney there were two vomicae, one in the cortex, the other in a pyramid, with opaque caseating tubercles round them.

(13) Another man, also in 1880, died of phthisis. In one kidney a pyramid was eroded by a single tuberculous ulcer, and beyond this, in the cortex, there were opaque white tubercles.

These cases show that while the characters of tuberculous disease of the kidney vary within certain limits, the morbid process is always fundamentally the same, and the result to which it tends identical. They also clearly indicate how close is the relationship between this affection and tuberculous lesions, not only in other parts of the urogenital apparatus, but also in remote organs.

In a single case, inspected in 1874, an ulcer which excavated a single pyramid of one kidney had a hard calcareous wall, which seemed to indicate that the affection was arrested in its progress, and might have remained stationary had the patient lived; but all Dr Fagge's other observations went to confirm the usual opinion, that when once tuberculous disease has begun, it goes on to destroy the whole kidney. The vomicae, which correspond more or less accurately with the affected pyramids, keep increasing in size, their caseous walls spreading further and further into the renal tissue, until they lie close beneath the fibrous tissue, which may thicken and become as hard as cartilage; and at last they touch one another on all sides, or communicate by lateral openings. The mucous membrane of the pelvis is converted into a thick whitish-yellow layer. Any parts of the cortex that escape removal by ulceration are converted into a tough fibrous tissue. Almost always there is enough cicatricial tissue to provide septa by which the excavated kidney is permanently divided into a series of sacculi, more or less completely shut off from one another; and these may at length lose their caseous walls and become bounded by a smooth lining membrane. Their contents are pure pus, or yellow, caseous pulp, or a substance like putty or mortar, with abundant earthy salts and crystals of cholesterine.*

It is obvious that such material could hardly accumulate were there a way freely open for its escape. The fact is that from an early period in the course of the disease the ureter is, as a rule, blocked and impervious. Its mucous membrane undergoes the same change as that which affects the renal pelvis; its other coats are indurated, and it becomes converted into a hard cord, which may be as thick as a pencil or thicker, and has but a narrow lumen left in its centre.

Tubercular ulceration of the pelvis of the kidney is generally secondary to similar disease of the bladder or testes, or both; and it usually leads to vomicae in the substance of the kidney. It is sometimes, however, secondary to calculous pyelitis, or the two coincide.

The *bladder*, in its turn, shares in the infective process. Sometimes an excavated ulcer forms round the orifice of the ureter; sometimes the whole

* Occasionally some of them ultimately come to contain a transparent yellowish fluid.
—C. H. F.

of the cavity is found lined with caseous patches, and more or less ulcerated. In many cases, however, the vesical affection begins before that of the kidney.

In three cases examined by Dr Fagge the disease spread to the *urethra*, and reached the external orifice, where the rough greyish-yellow appearance of the mucous membrane might easily have been seen during the patient's life. In one of these cases the canal was remarkably widened, so much so that a No. 16 catheter was required to fill it. When the affection was advancing along the urethra up to the time of death, we sometimes find at an autopsy obviously recent grey tubercles on the surface of the mucous membrane.

In many cases the male *genital organs*—the prostate, the vesiculæ seminales, the vasa deferentia, and the testicles, some or all—take part in the process. Even when there is no obvious disease of the testicle, one or more hard nodules can often be felt in the epididymis if careful search is made. As a rule, the vas deferens retains its natural size when affected with tuberculous inflammation, but in one case the whole spermatic cord was indurated during life. In the prostate the result of tuberculous disease is to cause moderate enlargement, with the formation of *vomicæ*; and the same may be said of the vesiculæ seminales.

Altogether, among thirty-four cases of advanced tuberculous disease of the kidney from our hospital-reports of autopsies, hardly a single instance occurred in which the other parts of the urogenital apparatus were quite free from tubercle. Of the kidneys themselves one alone is often affected, while the other shows no trace of tubercle: among the thirty-four cases this occurred in twenty-two, while in the other twelve the disease was bilateral, but always much older and more advanced on one side than the other. The right kidney is said to be less liable to become tuberculous than the left; and of the thirty-four cases the disease had apparently begun in the left in 20, in the right in 14.

It is a remarkable fact that in women tubercular nephritis is seldom attended by tubercle of the pelvic organs. Occasionally, however, the internal genitalia, and particularly the Fallopian tubes, may become tuberculous along with a like affection of the kidneys.

Symptoms.—The indications of tubercular nephritis during life are often remarkably slight. There may be pain in the loins, occasionally paroxysmal; and in some few cases there is tenderness on pressure.

The diseased organ very rarely forms a tumour that can be felt by manipulation of the abdomen. In Mr Lucas's case ('*Path. Trans.*,' 1875, vol. xxvi, p. 129), which occurred in a little girl of seven, a circumscribed tumour was detected in the right hypochondriac and lumbar regions; after death the kidney was found to be six inches in length, and eleven in circumference. In another case at Guy's Hospital a tumour is said to have been felt, but at the *post-mortem* examination the kidney did not weigh above eighteen ounces. In a third case there was a swelling which for a time led to the suspicion that the disease was malignant; but this proved to be an abscess behind the kidney, without much enlargement of the organ itself. As a rule a tuberculous kidney is but little above the natural size.

The urine is occasionally normal, no doubt owing to the ureter of the diseased kidney being blocked; so that it is possible for tuberculous disease to go on to complete destruction of a kidney without any pus reaching the bladder.

Hæmaturia is neither constant nor profuse ; but it usually appears early. Among eighteen fatal cases with notes of the symptoms during life, which occurred at Guy's Hospital, in only ten is blood said to have been at any time observed in the urine ; and in most of these cases the bladder was likewise affected with tuberculous ulcers, so that the exact source of the hæmorrhage was after all doubtful. The most striking case was that of a man who said that a year before his death he one day passed a pint of blood by the urethra, after straining his back in lifting a heavy weight.

Pyuria is a more constant and distinctive symptom. The quantity of pus in the urine is often so considerable as to form a thick deposit. Beside pus-cells, the sediment may contain granular amorphous masses insoluble in acetic acid, and even shreds of connective tissue, the presence of which is very significant. The *bacillus* of tubercle was formerly seldom recognised in the pus-cells ; but now it is readily detected after the centrifuge has separated the pus from the rest of the urine.

In many cases the urine retains its acid reaction throughout the whole course of the disease, but sometimes—probably always when the bladder is also affected—it becomes ammoniacal and foetid. There is then severe dysuria ; but it is remarkable that occasionally pain in micturition and strangury have been conspicuous symptoms during life, and yet after death the bladder has been found apparently healthy.

Pyrexia is generally present if there are other marked symptoms, and it may assume a hectic type. It is attended with loss of appetite, often with nausea and diarrhœa, and finally with emaciation.

Course and event.—The duration of the disease from the time when the patient is first discovered to be ill is commonly from six months to two or three years. But there are many instances in which its progress is so slow, that the opposite kidney has time to become hypertrophied, and probably carries on its function with efficiency. Ultimately this healthy kidney may in its turn suffer from the effects of pressure upon its pyramids, as the result of tuberculous disease of the bladder ; or an ascending suppurative nephritis may set in and rapidly bring the case to an end ; or the hypertrophied organ may become affected with Bright's disease, as mentioned above (p. 528) ; or the drain of pus from the tuberculous kidney or from other parts of the urogenital apparatus may lead to the development of lardaceous disease, both in the opposite kidney and in the viscera generally. Thus the urine may be albuminous and may contain tube-casts, when from obstruction of the tuberculous ureter no pus is for a time being discharged.

In one case at Guy's Hospital the immediate cause of death was the extension of ulceration from the tuberculous kidney into the peritoneal cavity. Others have ended in the formation of perinephric abscesses, which have pointed in the loin, or have burrowed down in the sheath of the psoas muscle, until they made their way into the hip-joint or appeared in Scarpa's triangle. Lastly, the disease often ends in the supervention of phthisis, of tuberculous peritonitis or meningitis, or of general miliary tuberculosis.

Ætiology.—With regard to the causes of tuberculous disease of the kidney, apart from those concerned in the production of tubercle in general, we know nothing. It shares the hereditary tendency of phthisis.

It is at least twice as common in men as in women.

Of twenty-nine cases at Guy's Hospital in which the renal disease was the principal cause of death, in three death occurred between the ages of ten and twenty, in twelve between twenty-one and thirty, in eight between

thirty-one and forty, in five between forty-one and fifty, and in one between fifty-one and sixty.

Treatment.—There is little to be done medically beyond placing the patient under favourable conditions and giving cod-liver oil and steel.

At an early stage, if the diagnosis can be made before the bladder has become affected, it is certainly advisable to run the risk of nephrectomy.

Morrant Baker ('Trans. Internat. Congr.,' 1881, vol. ii, p. 262) performed this operation in the case of a girl aged seven, with the result that five months afterwards the child had greatly improved in health, could play all day long, and go out of doors for a walk. Her illness had begun with hæmaturia about twenty-two months before the kidney was excised.

In vol. xv of the 'Clinical Society's Transactions,' Dr Goodhart and Mr Golding-Bird recorded a case of nephrectomy for tuberculous disease which proved fatal about four hours after the completion of the operation. The patient had been seriously ill for about eight weeks, but had complained of pain in back for eighteen months. At the autopsy the ureter was found to be diseased in its whole length, and the mucous membrane of the bladder was thickened and opaque. In the prostate also there was some cretaceous material.

It is the liability of the disease to affect other parts of the urogenital apparatus that renders nephrectomy a doubtful expedient in most cases where the symptoms are enough advanced to render diagnosis certain. For this reason it will perhaps be found that the prospects of an operation are more favourable in women than in men. In a recent case of the writer's, Mr Jacobson removed the kidney from a girl of eighteen, and she made an excellent recovery; there were several large tubercular vomicæ in the organ when excised. One important advance in the diagnosis of the kidney affected rests upon the passage of a catheter into each ureter successively, so as to ascertain from which kidney the pus flows.

SARCOMA AND CARCINOMA OF THE KIDNEYS.—When a malignant growth gives rise to secondary nodules in distant organs, some of them are often found in the kidneys. But in such cases the renal lesion is seldom of clinical significance. Occasionally it may cause hæmaturia, or (if the primary growth is melanotic) it may give the urine a brown or black colour, as in a case under Sir William Church.

Primary malignant tumours of the kidney are decidedly rare. Statistics from various sources are cited by Ebstein (in 'Ziemssen's Handbuch') in proof of this fact, and they are borne out by the reports of *post-mortem* examinations at Guy's Hospital, where only fifteen cases occurred during a period of twenty-two years.

Anatomy.—Two distinct affections were formerly included under the name of "malignant disease," or "cancer" of the kidney. One of them is the more often met with, chiefly in infants and in children up to the age of eight or nine years, though we have had two cases in boys aged respectively eleven and seventeen. In 67 cases collected by Sir Wm. Roberts, 22 occurred in children under five years old. This is *sarcoma*, like most of the malignant growths of other parts that are met with in children. It forms a smooth rounded mass, which sometimes reaches an enormous size, weighing ten, twenty, or even thirty pounds, so that at one autopsy it was said that instead of the tumour being removed from the child's body, the body was removed from the tumour. Such a growth is often very soft and elastic, so that it may appear to fluctuate; it is therefore likely to be punctured by the surgeon, a procedure which is generally harmless, but which may be followed

by a sharp attack of peritonitis. Sometimes, besides blood, a small piece of sarcomatous tissue is brought away in the orifice of the trocar. These sarcomata of the kidney are very vascular, and interstitial hæmorrhage often takes place, causing a sudden increase in their size.* They grow, too, with great rapidity, and may destroy life within a few weeks, almost always in less than a year from the time when they are discovered. They commonly affect one kidney only, but Dr Abercrombie showed to the Pathological Society (in 1880) three cases, occurring in young children, in each of which both kidneys were invaded at the hilus by sarcoma.

A few rare and interesting cases have been recorded of renal sarcoma containing striated muscle; they probably have their origin during embryonic life before the protovertebræ are fully differentiated.

A sarcomatous tumour in the position of the kidney is not always seated in that organ. Some years ago Dr Dickinson brought before the Pathological Society (vol. xxi, p. 397) a specimen in which the growth occupied the lumbar glands, and merely pushed the kidney before it; and in other cases it affects the fibrous capsule of an otherwise healthy kidney.

The second form of primary malignant disease of the kidney is true *carcinoma*. It is seen chiefly in persons past middle age; in almost every instance that occurred at Guy's Hospital during twenty years the patient was more than forty-five years old. It is much more common in men than in women.

The tumour is not large, as a rule, but it may sometimes occupy all one side of the abdomen, or even appear to fill the whole cavity, like a great ovarian tumour in a woman. In such cases a good part of its bulk is made up of hollow spaces containing a blood-stained fluid; or there may be a large accumulation of a similar fluid in the dilated renal pelvis, which has been shut off from the ureter.

Renal carcinomata used to be called scirrhus, but they are seldom hard, and show the ordinary glandiform type (*adenoma malignum*). In very rare instances they are colloid in structure.

In 1876 Dr Fagge showed the Pathological Society (vol. xxvii, p. 204) a *carcinoma lipomatosum* (of Cornil and Ranvier) growing in the kidney. It looked like adipose tissue, but had extensively invaded the renal veins, as well as the substance of the organ, and possessed a typical alveolar structure, the alveoli being filled with large cells loaded with oil-drops.

Sometimes carcinoma of the kidney causes enlargement of the whole organ, the distinction between cortex and pyramids being still traceable in the tumour; sometimes only certain parts are affected, rounded or irregular masses of growth being separated by tracts of healthy tissue. Probably, as Waldeyer taught, the renal epithelium is always the starting-point of the growth.

Like sarcoma, carcinoma, though as a rule it affects only one kidney, has occasionally been found in both.

Ætiology.—With regard to the causes of primary malignant disease of the kidney nothing definite is known. In a few cases it has appeared to be the result of a blow or of a kick in the loin; but one may doubt whether the injury did more than bring on symptoms such as hæmaturia, by which attention was first drawn to a disease which was present before.

* For an account of the histology see Dr Paul's paper with figures in the 'Pathological Transactions,' vol. xxxvii.

In the following two cases cancer appeared to supervene on another disease of older date.

One occurred in a man of forty-five, who was said to have been troubled by passing a gelatinous substance in his water for twenty or thirty years. The right kidney was found to have its calices dilated into a number of chambers, and in the pelvis lay a large, irregularly branched calculus, "like a knotted branch of a tree." Growing from the upper part of the organ was a cancerous mass, which also extended upwards behind the liver, and penetrated through the diaphragm into the lung.

The other case was that of a man aged sixty-six, who had an attack of hæmaturia twenty years previously, and who came under treatment for a recurrence of this symptom ten months before his death. The lower part of the kidney showed the ordinary appearances of hydronephrosis, with "sacculation" from distension of the calices; but into many of the sacculi soft masses of carcinoma were projecting, and the upper part of the organ formed a solid tumour. Apart from the anatomical appearances, it is almost inconceivable that the cancer could have dated back as far as the earliest attack of hæmaturia.

The occurrence of a calculus in the kidney as a complication of malignant growths seems not to be very rare; it may probably be the primary source of irritation, as with cancer of the gall-bladder (p. 555); but sometimes a phosphatic stone may be of later development than the tumour, especially if pyelitis happens to be present.

The ordinary *duration* of cases of carcinoma of the kidney in adults is probably from six months to two years after the first appearance of symptoms.

The symptoms of a malignant growth in the kidney, whether sarcomatous or carcinomatous, are mainly three: the presence of an abdominal tumour, hæmaturia, and pain.

1. The *tumour* occupies one side of the abdomen, in the lumbar region, between the lower ribs and the iliac crest. It often bulges into the loin, and one can move it slightly forwards by pressing up the loin with one hand, while the other is placed on the front of the abdomen. It does not descend during inspiration, and the fingers can be inserted between it and the ribs, showing that it is not seated in either the liver (if on the right side) or the spleen (if on the left). It is sometimes smooth and uniform, sometimes more or less uneven or lobulated. Overlying it in front there is usually a part of the colon (the hepatic or the splenic flexure, according to the side affected), which either may be felt as a ridge, or may be traced by its tympanitic percussion-sound. The dulness obtained on percussion over the tumour is continued into that of the lumbar region behind, whereas a splenic, ovarian, or other tumour, not growing from the loins, would be bounded externally by the resonance of the descending colon. Sir Spencer Wells proposed, in doubtful cases, to inflate the gut with air, so as to render the position of the bowel more conspicuous. Dr Fagge once felt several coils of small intestine in front of a renal tumour beside the colon; they were freely moveable, and slipped away from the finger under manipulation. Sir Wm. Roberts recorded a remarkable case in which not only was the stomach made out during life to lie in front of a cancerous left kidney, but the spleen could be distinctly felt as a separate mass in the iliac fossa, lying over the lower and inner part of the tumour. Mr Holmes recorded, in vol. xxiv of the 'Pathological Transactions,' a case in which a malignant growth of the kidney pulsated, and yielded a systolic murmur, so that aneurysm was suspected.

There is often considerable distension of the superficial abdominal veins, probably due to compression of the inferior vena cava by the tumour, or by

enlarged glands; but sometimes a fungous growth penetrates the renal vein, and it may even protrude into the cava, so as to narrow its calibre. As a result the feet and legs may become œdematous; and Rindfleisch speaks of embolism of the pulmonary artery as being sometimes caused by the detachment of portions of the cancerous thrombus. In a case that was observed at Guy's Hospital in 1871 the disease made its way into one of the veins of the colon, and thence into the portal vein and its branches within the liver; ascites was the result.

Probably the same cause explains the presence of varicocele in these cases—a fact pointed out by Prof. Guyon, and frequent enough to be of some value as a help in diagnosis.

2. *Hæmaturia* is a frequent but not a constant symptom. Ebstein found it absent in twenty-eight out of fifty-two cases which he collected; but very often it is the earliest indication that anything is amiss. Sometimes it is directly brought on by a blow or a fall. It may recur again and again at regular intervals for a considerable time before any tumour can be detected, and is often set down to a renal calculus. One distinction is that the hæmaturia is not generally preceded by marked aggravation of pain. In many cases the bleeding comes from portions of the growth that protrude into the pelvis of the kidney; but occasionally its source is from a tumour within the cortex; and if such is the case, tube-casts containing blood-corpuscles may be found in the urine, as stated by Ebstein.

3. The *pain* produced by a malignant tumour of the kidney is variable in degree, and sometimes altogether absent. Its usual seat is in one lumbar or hypochondriac region, but it may radiate widely over the lower part of the chest to the front of the abdomen, or to the crista ilii, and even down the thigh. It may be either constant, dull and aching, or paroxysmal, sharp and cutting in character. Sometimes there is much tenderness to pressure. The pain is not attended with retraction of the testicle, in which respect it differs from the renal colic of calculus. If, however, clots of blood formed in the pelvis of the kidney should become impacted in the ureter, the pain may assume a different character, and become exactly like that of renal colic.

Other symptoms that may be present in cases of malignant growth in the kidney are anorexia, nausea, vomiting, and constipation or diarrhœa. The patient usually becomes wasted, anæmic, and cachectic. The temperature remains normal or subnormal, and the pulse may be slow. Death is usually by exhaustion, and is sometimes preceded for a few days by stupor or insensibility. In a case recorded by Bright in the first volume of the 'Guy's Hospital Reports,' the tumour gave way into the abdominal cavity, causing a large extravasation of blood. Some years ago a woman was admitted into the hospital for paraplegia, which had been coming on during two months, but she was said to have had hæmaturia four months before, and to have been ill for a year. At the autopsy it was found that there was a primary cancer of the left kidney, and that the growth had extended into the spinal canal. A like case has been observed by Cornil. In a patient who died in Guy's Hospital in 1870 all the symptoms were cerebral, and the immediate cause of death was the presence of secondary tumours in the brain. The writer saw a similar case in 1898.

Diagnosis.—It can be easily understood that it is often difficult to recognise malignant disease of the kidney. A man aged thirty died in Guy's Hospital many years ago of wasting and anæmia, whose case excited great interest from there being no discoverable local symptoms. At the autopsy

it was found that the right kidney was the seat of a primary growth, which had destroyed nearly its whole substance, but did not reach its pelvis.

When a tumour in the lumbar region is accompanied by cachexia and emaciation in an elderly patient, it is probably malignant, and if hæmaturia is present the diagnosis becomes nearly certain. But there are no anatomical signs to distinguish between a renal growth and one starting in the lumbar glands, the adrenals, or the vertebræ.

Treatment.—It is improbable that much success will be reached by nephrectomy in cases of sarcoma or carcinoma of the kidney. But some cases have already turned out much more favourably than could have been anticipated. In 1877 Mr Jessop, of Leeds, removed an encephaloid tumour of the kidney from a boy two and a half years old; rapid recovery took place, but about eight months afterwards the disease returned, probably in the lumbar glands, and the case ended fatally a few weeks later. In 1878 Dr Martin, of Berlin, extirpated a sarcoma of the kidney, weighing twenty-eight ounces, in the case of a woman aged fifty-three; she was up on the eighth day, went home on the seventeenth; and Czerny speaks of her as being still well two years afterwards. In 1879 Lossen, of Heidelberg, performed nephrectomy in a woman aged thirty-seven, for an "angio-sarcoma" of the right kidney, which, being moveable, was mistaken for an ovarian tumour; she recovered in six weeks, and she continued to be in good health eighteen months later. In 1881 Czerny removed a large vascular sarcoma from a man aged fifty-three, who at the time of the operation was very cachectic, and suffered greatly from vomiting; two months later he left the hospital in complete health, with the proportion of red discs in his blood twice as great as it had been previously. Against these successes, however, must be set many cases in which extirpation of malignant growths in the kidneys has either been attempted ineffectually, or has proved quickly fatal by shock or by peritonitis (see Czerny's tabular statement in the 'Trans. Internat. Congress,' 1881, vol. ii, p. 249). Dr James Israel, of Berlin, recorded some early cases of recovery after removal of a cancerous kidney, and several have been published during the last fifteen years.

HYDATID OF THE KIDNEY.—The kidney comes next to the lungs, and far behind the liver, in the order of frequency among organs infested with echinococcus cysts. In our autopsies at Guy's Hospital there are only a few recorded; in one the parasite was the size of a plum; in another, of an orange; in a third it formed a bulging elastic swelling extending from the left hypochondrium into the loin, and containing two pints of fluid.

Clinically the diagnosis rests on the recognition of an abdominal tumour, having the characters above given of tumours of the kidney, more or less tense and rounded in form, painless, and possibly yielding fluctuation or palpable *fremitus*. The diseases for which it is most likely to be mistaken are hydronephrosis, soft sarcoma of the kidney, and (in the female) cystic disease of the ovary, as in a case in which Spiegelberg performed an operation which he intended for ovariectomy. A young woman was admitted into Guy's Hospital under Dr B. G. Babington, in 1854, with an abdominal tumour which had been wrongfully supposed to be a pregnant uterus. She passed first "skins and little bladders," and afterwards blood and pus from a hydatid of the right kidney. After many months she went out in good health, but still passing the echinococci and pus. It is instructive to notice that the tumour dated from a blow. A point which may sometimes aid in

the diagnosis is the discovery of a second hydatid in the liver; this was the case, for example, in a patient who died at Guy's Hospital with a hydatid in the kidney holding two pints of fluid.

In many instances an echinococcus in the kidney probably remains for years—perhaps from childhood or middle age throughout the entire life of the host—without affecting the health. Or it may die, and dry up into a pultaceous mass, which henceforth can do no damage.

In the majority of cases it sooner or later ruptures into the pelvis of the kidney, after which the daughter-cysts and scolices pass down the ureter and are expelled with the urine. Such cases may present the symptoms of renal colic (see p. 682).

The presence of cysts or hooklets in the urine is not in itself proof of the existence of a hydatid in the kidney. For the pelvic pouch of the peritoneum is nearly, if not quite, as frequent a seat of the echinococcus (*cf.* p. 461), and in such cases it sometimes opens into the bladder from behind, as in a case in which Mr Birkett (1885) drew off hydatids from the bladder during the patient's life. In two cases of pelvic hydatids there was a tumour in the hypogastric region of the abdomen, having exactly the shape and the other characters of a distended bladder.

In cases of renal hydatid the passage of daughter-cysts down the ureter is often the first indication that anything is amiss with the patient; and when the parent cyst is small, no tumour may be discoverable in the loin. Not infrequently the rupture of the cyst is directly produced by a blow or by a fall; or the symptoms may appear to be brought on by riding or by driving, as in cases of renal calculus. Sometimes there is only a single discharge of daughter-cysts in the urine, and the patient afterwards remains perfectly well; sometimes the same thing recurs again and again, at long intervals, during a period of ten, twenty, or even thirty years; sometimes suppuration within the capsule of the cyst occurs, and blood and pus may be voided in the urine. Roberts, however, says that the ultimate prognosis is generally favourable. Of sixty-three cases which he collected, only nineteen were known to have ended fatally; and in nine of these the cause of death was some disease not directly connected with the renal affection. In some instances there has been ulceration through the diaphragm, with escape of daughter-cysts into a bronchial tube and expectoration through the air-passages; the prognosis is then very unfavourable.

The usual *treatment* of hydatid of the kidney, when a tumour can be detected, is puncture with a tubular needle, fitted to an aspirator. In two cases Roberts found that the withdrawal of only a drachm or two of fluid sufficed to destroy the life of the parasite, and caused it to pass very gradually into obsolescence, just as we found in the case of the liver (p. 565). When these measures fail, the only plan is to explore the tumour from the loin—or as some surgeons prefer, from in front—to empty the cyst, and stitch it to the edges of the wound.

CHYLURIA.—Prout first described a remarkable condition of the urine, in which it looks white and milky when passed, and soon afterwards sets more or less completely into a soft jelly, like *blanc-mange*, which takes the shape of the vessel that contains it. Sometimes it solidifies within the bladder, and the result may be severe pain, and difficulty in micturition from obstruction of the urethra. The coagulum after a little while liquefies again *in vitro*; a material like cream collects upon the surface, and there

falls a deposit which is generally of a pinkish colour, from the presence of a small quantity of blood. Prout recognised that the characters of the affection were exactly such as might be due to the admixture of chyle with the renal secretion. This view is confirmed by microscopical examination, which shows that the cause of the opacity is a finely granular material, not large fat-globules as in milk; and also by the application of chemical tests, for by ether a large quantity of fat may be extracted, and the urine also contains albumen in considerable amount. Occasionally glyose has been also present. Beside the "molecular base of chyle," granular leucocytes and sometimes a few red blood-discs are seen under the microscope.

This peculiar state of the urine is often far more marked a few hours after a full meal than when the patient has been fasting for some time.

It is to be noted that *chyle*, in a strict sense of the term, is not always present in the urine when it is more or less opalescent; if the clot which forms is translucent, it may be due to the presence of *lymph* that has passed through one or more glands on its way upwards to the thoracic duct. But if the clot is opaque, and if the state of the urine varies in relation to the patient's meals, there can be no doubt that the obstruction involves lacteals coming from the intestines.

The pathology of chyluria was long a complete mystery, but the question has step by step been cleared up, until we now seem to know nearly all about it.

Prout noticed that a large number of those affected were born, or had lived for many years, in hot climates. Next, in 1866, Wucherer, in Brazil, detected in chylous urine the embryos of a nematode worm. Six years afterwards, the late Dr T. R. Lewis, in India, discovered similar embryos in the blood; and the parent worm (*Filaria sanguinis hominis*) has since been discovered, with the complete history of its development, as already set forth at length in the chapter on Entozoa (*supra*, p. 472).

The immature ova form obstructions in the lymphatic vessels, and the result is rupture of some of the lymphatics and extravasation of their contents.

If the ova are carried by the lymph-stream to a gland, they become impacted in the smaller lymph-channels in the cortex.

If the obstruction affects the abdominal or pelvic lymphatics, the result will be more or less complete stasis of lymph, not only in the neighbourhood of the spot where the parent worm is situated, but also in the whole of one or both of the lower limbs, or in the scrotum. In the former case chronic œdema with hypertrophy is produced, and is called *elephantiasis* (*Arabum*), or Barbadoes leg; in the latter it is known as *lymph-scrotum*. Sir William Roberts recorded a remarkable case of intermittent chylous urine, together with lymphatic dilatation and lymph or chyle-vesicles scattered over the abdomen; and thus the chain of connection between these several disorders is complete.

Elephantiasis will be considered under diseases of the skin.

Lymph-scrotum consists in the formation of vesicles, which are in fact dilated lymphatics, and discharge a clear or milky fluid. The tissues of the scrotum are thickened, but feel soft and spongy. Similar vesicles may form on the inner side of the thigh. In the course of years the flow of lymph ceases, and the scrotum passes into a state of elephantiasis; or the elephantiasis may be developed in the first instance without the formation of vesicles or escape of fluid.

When *chyluria* occurs, there can be no doubt that distended lymph-vessels open upon the surface of some part of the urinary mucous membrane; but whether this takes place most often in the bladder, or in the ureter, or in the renal pelvis, has not yet been determined.

One peculiarity of the affection is that it is often intermittent, the urine from time to time losing its abnormal characters for days or weeks together. In a case recorded by Ackermann ('Deutsch. Klin.,' 1863) the patient always passed normal urine after he had been lying on his right side.

The relations now ascertained to exist between the filaria and the chyluria of hot climates leave undetermined the pathology of the disease when it occurs in persons who have always resided in Europe. Among well-authenticated instances may be mentioned: Prout's original case, Dr Beale's, Dr Dickinson's, and Dr Morgan's at Manchester. In every case there is no doubt a definite fistulous communication between the lymphatics or thoracic duct and the urinary passages, whether caused by the filaria or any other agent.*

Chyluria as an endemic disease is found in Bombay and other parts of India, in China and Japan, in Mauritius (formerly Isle de France), and in Réunion (formerly Isle de Bourbon), in the Bermudas and the West Indies, in Guiana and Brazil, and in Queensland.

The autopsy upon Dr Stephen Mackenzie's patient ('Path. Trans.,' 1882, pl. xxii, p. 394) showed a large mass of dilated lymph-sinuses and glands, extending from the bifurcation of the aorta below to the diaphragm above, and occupying the whole of the space between the kidneys. The lower part of the thoracic duct was sinuous and pouched, varying in diameter from three eighths to half an inch. About three inches above the diaphragm it became impervious, and was lost in a quantity of tough, dense material, apparently of inflammatory origin. In this case the communication between the lymph and the urine was probably in the kidneys.

When rupture of a lacteal takes place into the peritoneum, we have, as the result, Chylous ascites (p. 497); and if the same accident affects the pleura, a similar effusion of milky, more or less coagulable, fluid is poured into the chest.

It is obvious, from what has been stated with regard to the life-history of the filaria, that the *prevention* of the diseases due to this parasite is quite possible. Dr Manson suggests that wells and water-jars should be covered with a netting sufficiently fine to prevent the entrance of mosquitoes, but it must surely be a better plan to drink no water which has not been boiled or filtered. Care must also be taken to have all raw vegetables thoroughly washed with boiled water before eating them.

In the *treatment* of chyluria, when it is once established, very little can be hoped for from medicines. Bence Jones thought that by giving gallic acid to the amount of two drachms daily he was successful in restoring the urine to a normal state for periods of several months at a time. In Guiana the old women give decoctions of mangrove bark, and not without apparent success. When the loss of chyle is considerable, it sometimes causes emaciation and debility, as well as a craving appetite and urgent thirst. In such cases exercise is found to aggravate the complaint.

The duration of the disease is often very long. Roberts cites two instances, in one of which it continued for twenty-eight years, and in the other

* See a case of chylous ascites admirably worked out and recorded by Dr Whitla, of Belfast, in the 'British Medical Journal,' May 30th, 1885.

for more than fifty years. If death occurs during its course the cause is generally some intercurrent malady, such as phthisis or Bright's disease.

PARASITIC HÆMATURIA.—As far back as 1812 Chanotin recorded the prevalence of an endemic form of hæmaturia in Mauritius; and subsequent writers afterwards noticed the occurrence of a similar affection in other hot climates. But nothing was made out with regard to its nature, until in 1851 Bilharz, being engaged with Griesinger in investigating the diseases of Egypt, discovered in certain of the veins of the abdominal viscera a trematode worm, to which he assigned the name *Distoma hæmatobium*.

It was found that this parasite gave rise, in some cases, to more or less severe urinary symptoms; and Griesinger, in the 'Arch. d. Heilkunde' for 1854, suggested that it might probably be the cause of the endemic hæmaturia of warm countries. Afterwards it was shown to be generically distinct from the liver-fluke, and Cobbold proposed for it the name of *Bilharzia hæmatobia*, which is now generally adopted. In 1863 Dr John Harley detected the ova of the same entozoon in large numbers in the urine of a man who had become affected with hæmaturia at the Cape of Good Hope; and he afterwards showed ('Med.-Chir. Transactions,' vols. xlvii, lii, liv) that the complaint prevails not only in the Cape Colony, but also along the coast of Natal.

The Bilharzia is a soft, white worm, belonging to the class Trematoda (*cf. supra*, p. 475). The two sexes differ in shape. The male, half an inch in length, is flattened; but the hinder part of its body acquires a cylindrical appearance from its edges being thinned and folded inwards, so as to overlap one another and form a hollow channel. It has two suckers, a ciliated surface, a single ganglion, and an unbranched set of water-vascular canals. The female, three quarters of an inch long, is slender and filiform. The ova are about $\frac{1}{170}$ " in length, and have a sharp projecting beak-like spine, placed usually at one end, but sometimes laterally. According to Dr Zancarol, of Alexandria ('Path. Trans.,' xxxiii), ova with lateral spines are found only when the seat of the parasite is in the veins of the intestine, whereas when it occupies the veins of the urinary tract the spines are terminal; and this statement seems to correspond with previous observations. (See the drawings by Dr Cavafy, *ibid.*, p. 410.) It often happens that empty egg-shells are found in the interior of the human body, so that there can be no doubt that the ova may be hatched while in the tissues. But Dr Cobbold believed that the ova in urine never give exit to the embryos, which are often to be seen fully developed within them, and quite ready to escape. Such ova, however, when placed in water, rupture in a few minutes. The embryos are covered all over with cilia, and swim actively about.

The further steps in the life-history of the Bilharzia have not yet been ascertained; but the presumption is that the embryo finds in some fresh-water mollusc an "intermediate host," and there develops a Cercaria-form. Leuckart seems to think that the most probable way in which human beings become infected is by their swallowing encysted cercariæ in minute slugs eaten accidentally with raw vegetables. Griesinger believed that in Egypt the chief danger lay in the use of fish as food. Dr Harley inclines to think that, in Natal, bathing may give occasion for the parasite to make its way into its human host through the skin, or possibly direct into the bladder through the urethra. Males are much more liable to suffer from the Bilharzia than females. The resulting hæmaturia commonly appears during

boyhood, but never under five or six years old. It may, however, occur at any later age, for one of Dr Harley's patients was a man of seventy-six. It is rare in Europeans.

The bladder is the seat of endemic hæmaturia. At the end of micturition, the patient voids a small quantity—perhaps a teaspoonful or less—of dark blood, or filaments of mucus, which sometimes block the urethra for a few minutes; in these shreds the ova of the Bilharzia are found in large numbers. Sometimes a little pain is experienced in the loins or in the perinæum, especially after active exercise, which also increases the hæmorrhage. The health, as a rule, remains good, though more or less anæmia may ensue. During early adult life, in Natal, small calculi may be passed, in the centre of which remains of ova may be detected; and in Egypt the frequent result is the formation of a stone in the bladder, so large as to need operation.

Bilharzia, and the hæmaturia that it causes, are common in Algiers, the Nile Valley, in Natal and the Transvaal, the West Coast and Africa generally, and also in Arabia, Mauritius, and Madagascar.

Bilharz and Griesinger found the parasite present in Cairo in 117 out of 363 autopsies. The ordinary symptoms they observed were more serious than in Natal. The earliest morbid change in the bladder was the formation of swollen, hyperæmic, ecchymosed patches, varying in size up to that of a shilling, and generally coated with tough mucus or with a layer of soft greyish-yellow exudation; they were often limited to the posterior wall of the organ. In many cases there were also thick deposits of a soft granular material upon the mucous membrane, generally incrustated with urinary salts. Sometimes there were warty vegetations or fungus-like swellings. Ova and empty shells of the Bilharzia were present throughout the diseased tissues, and also in the mucus and muco-pus on the surface of the bladder. Deeper down lay the parasites themselves, in smooth-walled spaces, which communicated with the veins, and evidently were dilated venules.* The condition was one of chronic cystitis with, in the worst cases, partial necrosis or, as it used to be called, pseudo-diphtherial or pseudo-dysenteric cystitis.

How the ova effect their escape from the spaces in which the parent worms lie, and how they pass through the mucous membrane, has not been made out. Nor has the life-history of the parasite, probably including a period passed external to the human body, yet been traced. The allied liver fluke (*Distoma hepaticum*) which is frequent in sheep, and occasionally has been found in the human bile-passages (*cf.* p. 475), is passed as an ovum from the sheep, hatched in ponds, and the embryo penetrating the tissues of a fresh-water snail, develops *cercarie* by internal gemmation, which make their way out of the molluscan host in pastures, and are devoured by sheep. It is therefore probable that the Bilharzia has a somewhat similar series of transmigrations to go through.

The ciliated embryo is speedily hatched from the ova when placed in water, and swims about freely; whatever its subsequent metamorphoses, it is pretty certain that the medium of entrance into the host is water directly, or indirectly by means of cresses or of freshwater fish. Probably it enters its human host, not through the mouth or skin but through the anus, vagina, or urethra.

The diagnosis rests on the discovery, in the blood or mucus which settles

* The alimentary canal of the worms was always full of blood-corpuscles.

from the urine, of the oval eggs, like melon seeds, with a terminal or, occasionally, a lateral spike. Each is about $\frac{1}{200}$ inch long. The writer has only seen a single patient with this remarkable complaint, an Englishman who had contracted it in Natal. The ova were abundant and perfectly characteristic.

In many cases the ureters are affected as well as the bladder, and sometimes they suffer when it escapes; in exceptional instances pyelitis with renal abscess has been known to follow; or hydronephrosis may be the result, and at length complete destruction of the kidney. In most instances, however, the direct cause of a fatal issue is either pneumonia or dysentery. Beside the veins of the urinary apparatus, the only other vessels in which the Bilharzia occurs are the portal vein and its tributaries; and symptoms like those of dysentery may sometimes be produced by the Bilharzia. Some of Griesinger's cases marked by fatal "typhoid" symptoms were due perhaps to septicæmia. That the eggs may be carried to distant parts of the blood-stream is shown by a case in which a few empty shells were found in the interior of the heart; so they may act as mechanical or septic emboli.

In the *treatment* of endemic hæmaturia, Dr Harley tried daily injections into the bladder of from twenty to thirty grains of iodide of potassium dissolved in five ounces of warm water. Dr Guillemard, however, has recorded a case in which even a weaker solution than this set up acute cystitis; and the impossibility of reaching the seat of the parasites in the portal vein makes such attempts vain. Oil of male fern and oil of turpentine have been prescribed; but the conclusion at which Dr Guillemard arrives is that all treatment at present known is useless.

Although so common among the natives in the valley of the Nile and in Southern Africa, that half the male population in some districts are believed to be affected, it is said that when boys between six and fifteen contract the infection and become subject to hæmaturia, they seldom suffer, and, as a rule, recover without treatment about the period of puberty. This seems to be less true in Egypt than in the Transvaal. The prognosis when Europeans contract the disease is far from uniformly favourable, and apart from hæmaturia and cystitis, there is a considerable liability to calculus.

MALFORMATION AND FIXED MALPOSITION OF THE KIDNEYS.—One kidney is sometimes congenitally absent or atrophied. As a rule, the ureter remains intact, and the adrenal body seldom partakes in this or any other renal abnormality.* Sometimes the two kidneys have their lower ends united in the shape of a horseshoe. When the abdomen is thin and flaccid, this abnormality might easily lead to the diagnosis of an abdominal tumour, or an aneurysm of the aorta. The presence of hydronephrosis† or of pyelitis in a "horseshoe kidney" might also be misinterpreted during life.

In a specimen in the museum of Guy's Hospital both kidneys are united into a single mass, which lay within the pelvis.

Occasionally one kidney—generally the left—lies at a lower level in the abdomen than usual, over the sacro-iliac synchondrosis, or within the pelvic cavity. The misplaced organ may be mistaken for a tumour, as in a

* An exception is supplied by Preparation 1574 in the Guy's Museum, when the left kidney, ureter, and adrenals are all absent.

† As in Prep. 1578 in the same museum.

case recorded by Mr Durham in the 'Guy's Hospital Reports' for 1860; or, in the female, it may interfere with parturition.

A kidney may have two ureters, or the ureter may be in front instead of behind the renal artery and vein.*

An interesting series of malformed, misplaced, and congenitally atrophied kidneys is described and figured by Dr David Newman, of Glasgow, in the 'Clin. Trans.' for 1898, p. 118.

The persistence of a foetal condition, in what is called a lobulated kidney, does not in any way interfere with its functions.

MOVEABLE AND FLOATING KIDNEY.†—We occasionally meet with cases of the kidney becoming loose, so as to move in various directions, downwards, forwards, or inwards. It can be readily felt through the abdominal wall, and can almost as readily be pushed back for the time into its place. Pressure on the quadratus lumborum does not meet with the usual sense of resistance; and percussion may yield a resonant note.

In some cases a moveable kidney is only part of a general condition of looseness of the abdominal viscera, sometimes called Glénard's disease (*cf. supra*, p. 556).

Side.—The right kidney is much more often affected than the left. Among ninety-one cases collected by Ebstein, in sixty-five the right kidney was moveable, in fourteen the left, and in twelve cases both. In Landau's 173 cases (many of which, however, were identical with Ebstein's) 152 were right, only twelve left, and nine double.

Age and sex.—A moveable kidney is most often discovered in patients between the ages of twenty-five and forty, but several cases have been observed in children. In 122 cases collected by Landau, 43 occurred between thirty and forty, and 79 between twenty and fifty.

The same writer found in 97 cases, 87 women and only 10 men. Other collectors give the numbers as 61 to 9, as 82 to 14, as 87 to 13, or as 7 to 1.

As a rule the place in which the kidney is felt is in the iliac fossa, or somewhere between this and its natural seat. Sometimes, however, it comes in contact with the front wall of the abdomen, and is far more freely moveable. A kidney thus *floating* must have peritoneum on both sides; and there is anatomical evidence that it is provided with a mesonephron, sometimes, but not always, congenital. Possibly an imperfect degree of this condition may be a subsequent result of a *moveable* kidney making for itself a serous tunic by pushing forward the membrane that should naturally cover only its anterior surface.

In vol. xxvii of the 'Pathological Transactions' may be found a case observed by Dr Goodhart, in which the right kidney, while lying on the spine and over the psoas muscle, had become completely turned over, so that its anterior surface looked backwards; both surfaces were covered by peritoneum. As a rule, however, a moveable kidney glides about behind the serous membrane, merely dragging this with it to a slight extent.

It is very rare for a moveable kidney to be discovered after death, perhaps from the greater firmness of the adipose tissue around it. The writer once found a case in the dissecting room, but Durham found only two among

* Guy's Hospital Museum, Nos. 1589—91, and 1588.

† *Synonyms.*—Nephroptosis.—*Fr.* Reins mobiles, Reins flottants.—*Germ.* Bewegliche Niere, Dislokation der Niere, Wanderniere.

1600 autopsies at Guy's Hospital, and the late Sir Andrew Clark only the same number out of 4000 at the London Hospital.

The *causes* of the moveable and of the floating kidney are not always clear. The kidney is kept in its place against the force of gravity, chiefly by the peritoneum which covers it in front, partly by its adipose capsule, and by the pressure of the liver or spleen and the intestines. In health the tonic contraction of the muscular walls of the abdomen keeps all the organs in place; but when they are enfeebled and relaxed, a sinking of the heavier viscera is likely to occur. Sometimes the kidney is pushed downwards by an enlarged liver; in one case, at Guy's Hospital, by a cancerous liver. Cruveilhier thought that displacement of the right kidney was often an indirect result of tight lacing, through its altering the position and shape of the liver. A mobile kidney is far more frequent in women than in men, sometimes from this cause, but more often from a relaxed state of the abdominal walls in consequence of frequent child-bearing. Too energetic shampooing has, at least in one case, produced a mobile kidney. Becquet maintained that a principal cause of mobility of the kidney is congestion and swelling of the organ, recurring at the menstrual periods; and Roberts, in his article in 'Reynolds' System' (p. 645), mentions two cases in which a displaced kidney seemed to become larger and more sensitive each time that the catamenia appeared. Lancereaux confirms the statement.

Bartels and several other observers have found dilatation of the stomach to be frequently associated with a moveable kidney; and Macalister believes that this is due to mechanical traction on an occasional peritoneal fold, which at last occludes the duodenum.

The *symptoms* caused by mobility of the kidney are sometimes slight, or even absent. Walther, of Dresden, examined a large number of persons to decide this point, and detected a mobile kidney in many cases in which there were no symptoms whatever. But more frequently it causes a sense of weight and pressure in the abdomen, a feeling of dragging, as though something were loose in its cavity, or more or less intense pain, which may radiate in various directions—to the ribs, the shoulder, the epigastrium, or the external genitalia. Sometimes there is nausea or vomiting, increased by palpation. Such patients are liable to attacks of intense suffering, attended with faintness and collapse, during which the kidney becomes exceedingly tender; and after these attacks, a large amount of clear, pale urine is often passed. Active exercise, whether walking or riding, often brings on or aggravates the pain; some patients are prevented by it from standing upright, or from turning in bed, or lying on one side. There is often an apparently disproportionate degree of anxiety and of depression of spirits, leading to hypochondriasis or hysteria.

The *diagnosis* of a moveable kidney is sometimes easy, sometimes difficult and uncertain. We have in practice to distinguish it from fæcal accumulation, from an enlarged spleen, from a distended gall-bladder, from a mass of swollen glands, or from an ovarian cyst with a long pedicle. Ebstein mentions an instance in which a hydatid cyst in the mesentery was mistaken for it.

The patient should lie in bed with the knees drawn up, and the abdominal muscles must be relaxed by distracting her attention. One hand should be placed behind the lumbar region and the other gently and gradually pressed down upon the kidney. If moveable, it will pass downwards and inwards; if already dislocated, it will have followed that direction or

straight downwards. The prone position is sometimes advantageously taken by the patient instead of the supine, in order to facilitate palpation. A second exploration is often needful, and occasionally slight chloroform narcosis is a justifiable help to a certain diagnosis. The points to notice are the shape and size of the tumour, its smoothness and mobility, and the almost testicular sensation which seems to be produced by moderate pressure.

It must be borne in mind that a floating kidney is not exempt from hydronephrosis, cancer, and other local diseases—perhaps more liable to them; so that it may be a moveable renal tumour rather than a moveable kidney which we have to recognise. It should be added that the secretion of a floating kidney is in all respects normal.

The *treatment* of this affection consists in keeping the patient in bed during the attacks, and applying fomentations and poultices when there is severe pain. Leeches or hypodermic injections of morphia may be required. Afterwards an elastic abdominal belt should be applied, with a pad to maintain the kidney in its proper position. It is often, however, very difficult so to adjust an apparatus as to effect this object.

Czerny advised the injection of alcohol into the connective tissue around the organ, so as to induce local adhesions.

Hahn cut down upon the kidney by a lumbar incision, and fixed it in its proper place by sutures in 1881 (*nephrorrhaphy*). Several cases of this operation, called *néphropexie* by the French surgeons, are recorded by Ceccherelli ('*Rivista Clinica*,' April, 1884), quoted by Sir Spencer Wells. It is now frequently performed; and in 134 cases, quoted from Keen by Macalister, there were only four deaths.

In one only of the cases which have come under the writer's care has he advised an operation; in this one, at the patient's urgent request for relief, Mr Jacobson exposed the right kidney, which was brought into its proper place under chloroform, and fixed there with silver sutures. A good recovery followed, but the subsequent relief was less complete than we had hoped. In another case, in a man, the writer refused to recommend an operation, and learnt afterwards that it had already been performed, but without relief of his, no doubt, subjective symptoms. The fact is that most patients with moveable kidney are more or less neurotic—apart from hypochondriacs, who only imagine they have the complaint; so that any treatment is apt to fail.

When, however, a moveable kidney is attended with unbearable suffering it is perhaps justifiable to extirpate the organ. About a fourth of these cases have ended fatally, by peritonitis, or in consequence of the opposite kidney being diseased; but others were completely successful.*

* Besides the chapters in the systematic works of Rayer (to whom the first clear account of this curious malady is due), Trousseau, Ebstein, and Roberts, the following monographs on the subject are interesting:—Rollett's '*Pathologie und Therapie der beweglichen Niere*' (Erlangen, 1866), and Landau's '*Wanderniere der Frauen*' (Berlin, 1881), translated for the New Sydenham Society by Dr Champneys. Valuable papers on the subject have been published in England by Dr Hare ('*Med. Times and Gazette*,' 1858), Mr. Durham ('*Guy's Hosp. Rep.*,' 1860), by a committee of the Pathological Society (vol. xxvii), and by Dr Newman ('*Glasgow Medical Journal*,' August, 1883), with a useful bibliography. See also Mr Henry Morris's article in the '*Lancet*,' Nov. 30, 1901, p. 1457.

ADDISON'S DISEASE

WITH OTHER AFFECTIONS OF THE ADRENAL BODIES

“Mislike me not for my complexion.”
Merchant of Venice.

History of the discovery—The anatomical changes in the adrenal capsules—The symptoms: melanoderma, asthenia, cardiac failure, gastric disturbance—Order of the symptoms—Course, mode of death, and duration of the disease—Diagnosis—Controversial points—Origin—Relation to caries and to tubercle—Theory of the symptoms—Prognosis and treatment. Hæmorrhage, cancer, and other morbid conditions of the adrenal capsules.

ALTHOUGH many diseases have been named after their first describer, it is seldom that the merit of discovery is not divisible with earlier observers who paved the way, or with later ones who corrected or completed the work. If, however, any disease is to be associated with a single name, it is that which is the subject of the present chapter. There are no rival claims of priority, for no one had suspected either the anatomical change in the curious and unique organs which are affected, or the remarkable symptoms which denote this change during life; nor has subsequent investigation added much to Addison's original discovery, the chief conclusion reached by the inquiries of nearly fifty years being that it is not every disease of the suprarenal bodies, but one only, caseous or tuberculous inflammation, which produces the clinical results that he described.

While engaged in the investigation of anæmia, which led to the recognition of that peculiar form named by its discoverer idiopathic, and by others grave, progressive, or pernicious, Thomas Addison “stumbled,” to use his own expression, on the connection between the symptoms and the lesion which belong to what has since been known as morbus Addisoni.

This remarkable disease is rare and apparently incurable, so that its interest is at present pathological. It was reasonably hoped that the natural experiment of disease would throw light on the much disputed function of these anomalous adrenal bodies (*glandulæ suprarenales, vel atrabiliosæ, renes succenturiati*, as they were once called); but the expectation has not at present been fulfilled.

Melasma suprarenale was the name proposed by Addison in his original work on ‘The Constitutional and Local Effects of Diseases of the Suprarenal Capsules,’ which appeared in 1858, and has since been republished among Addison's works by the New Sydenham Society (1868). The name morbus Addisoni was proposed by Trousseau, and at once generally accepted.

The most important later facts on the subject will be found in the ninth volume of the 'Pathological Transactions' (1858), in Wilks's papers in the eighth and eleventh volumes of the third series of 'Guy's Hospital Reports' (1862 and 1865), and in Greenhow's Croonian Lectures on "Addison's Disease" (1875).

Wilks has drawn attention to two undoubted cases of the disease, described (but not recognised as dependent on the lesion of the adrenals) by Aran in 1846, and by Bright in 1829, the diseased organs of the latter case being still preserved in our museum, No. 1544 ('Guy's Hospital Reports,' third series, xxii, p. 266). It is only justice to add what is well known to those familiar with the recent history of medicine, that Addison's work was cut short by his death, and that the establishment of his discovery by greater accuracy in admitting cases (particularly by excluding those of cancer) was due to Sir Samuel Wilks, who in these labours, as in those by which he carried on and greatly improved the original observations of Hodgkin, has been more studious of their credit than of what rightly belongs to himself.

Anatomy.—The lesion is now proved to be a tuberculous inflammation, with the same stages as mark its progress in the lungs, the lymph-glands, the kidneys, or the testes. The earliest change yet observed is the appearance, in the medullary substance, of a firm grey mass, more or less nodulated at its growing edge, and often surrounded by clusters of miliary tubercles. At this stage, however, the disease is seldom seen in the dead-house. After death the grey material is generally found to have already spread into and destroyed the cortical substance. The capsule is then much enlarged, hard, irregular in form; and when a section is made, the cut surface shows no trace of the natural structure. The colour is sometimes a uniform grey, which acquires a pink hue when exposed to the air, but in most cases the grey substance has undergone partial caseation, forming rounded yellow nodules; and, at a later stage, the conversion is complete, so that there is only a single large yellow mass. Softening often takes place at this stage into a puriform liquid. Finally, a process of absorption begins, and the diseased organ, from being much larger than natural, shrinks into a small puckered mass, in which irregular nodules of calcareous matter are embedded.

During the early stages of this process the fibrous envelope of the organ becomes greatly thickened. Adhesions to the neighbouring parts are also formed, principally to the liver, kidneys, diaphragm, or stomach, pancreas, or vena cava.

The morbid changes in Addison's disease are probably never limited to a single suprarenal body. One, however, is generally attacked earlier than the other, and after death may be found completely destroyed, while its fellow is still in an early stage of disease.

A microscopic section at an early stage shows small round-cells, like lymph-corpuscles, lying in the meshes of a delicate, wavy, fibrillated stroma. Later on well-marked fibrous tissue, still grey and translucent, takes its place, with some elongated cells and oval nuclei interspersed between its fibres.*

In the third stage, the microscopical appearances are the same as those of testes, kidneys, or lymph-glands which have undergone caseation.

* It seems Wilks's original description in the 'Guy's Hospital Reports' for 1862 and 1865 was of specimens in this stage.

The description just given is that of "scrofulous," or "strumous," *i. e.* tuberculous inflammation, which we have met with in other organs. Histologically giant-cells occur, and the bacilli characteristic of tubercle are always to be found in the diseased adrenal.

Addison spoke of the anatomical lesion as "scrofulous," but formerly this name was applied to all caseous processes. Wilks at first doubted whether the disease was of a tubercular nature. But Virchow and Rindfleisch decided to call Addison's disease a tuberculosis of the adrenals, and there is now no doubt that they were right.

This conclusion is confirmed, as it was first suggested, by the concomitant tuberculous lesion of the bones and lungs which is often met with in this disease. In cases of general miliary tuberculosis grey tubercles may be found in the adrenals without any symptom of Addison's disease, for, as we shall see, it is not the origin but the extent of destruction which is essential. Beside, these cases are too acute for symptoms to develop. There are not wanting cases, however, in which tubercle is confined to the adrenals.

Symptoms.—The characteristic features during life which enable us to diagnose the above lesion of the adrenals are briefly as follows:—(1) gradually increasing weakness of the heart and other muscles; (2) ingravescent nausea and vomiting; (3) a peculiar discoloration of the skin.

Melasma.—The discoloration of the skin is the most striking symptom of Addison's disease. It has been variously described as yellowish or greenish brown, dusky, smoky, or as if stained by walnut-juice; the term "bronzed-skin" was at one time commonly applied, but is not often very suitable. The colour is unlike jaundice or the sallow tint of lues, of malaria, or of lead, or the yellow hue of Addison's anæmia; it resembles that of a mulatto or a Moor. As in persons who have long resided in hot countries, the discoloration is deeper on the face and neck and on the backs of the hands than on the covered parts of the body generally; but it is very marked on the genital organs and about the pubes, and also in the axillæ, the navel, the nipples, and their areolæ. In extreme cases it may be universal, so that the patient looks like a half-caste; but it is never uniformly deep over all parts of the body.

There is no sharp line of demarcation between the affected parts and those unaffected; they shade off into one another. But in most cases there are present, in addition to the general discoloration, a few small black spots resembling pigmented moles, which have more or less defined outlines.

Friction often determines the seat of pigment. Thus, in women a brown ring may be seen where the garters have pressed, or where the petticoat strings have been tied. A case is quoted of a baker's boy, whose shoulders showed dark bands corresponding with the straps by which his basket had been slung over his back. So, again, the application of a blister to any part of the skin is followed by the formation of a brown patch; and deep stains are left by eczema or any other form of dermatitis.

These stains and patches are but an exaggeration of pigmentation that occurs in healthy persons, particularly in those of dark complexion under the stimulus of pressure or irritants; and the diffused discoloration of the face, neck, and hands may be compared with what would naturally be observed in anyone exposed to the heat of a tropical sun.

Some years ago Dr Pavy had under his care a woman who suffered from the other symptoms of Addison's disease; but the only melasma present was in the form of reticulated marks on her legs; such as are seen in persons who habitually sit before the fire, and are known as *ephelis ab igne*. A short time afterwards she died, and disease of the adrenal bodies was found at the inspection.

The discoloration of the genital organs, axillæ, and nipples may be regarded as an intensification of physiological deposits of pigment; and the small black scattered spots are exactly like congenital moles. The pigmentation which accumulates where pressure or inflammation (*i. e.* hyperæmia) has existed is also physiological; and the pigment-granules are deposited in the rete mucosum just as in health. What is abnormal is its amount.

Each lip often shows a bluish-black streak along the line of contact with its fellow; and on the buccal mucous membrane irregular and ill-defined brownish stains may occasionally be seen, looking like smears of ink. Similar stains are found in the lining of the cheeks of Lascars. Another part of the mucous membrane on which stains occur in Addison's disease, is the side of the tongue; they are of a purplish or inky hue, and are situated near the free margin.

The conjunctivæ always remain pearly white, contrasting with the dark colour of the surrounding parts of the face.

The microscopical appearances of the discoloured parts of the skin bear out the close resemblance of adrenal melasma to that of the darker races of mankind. The pigment consists of yellowish-brown granules; and its chief seat is in the deeper layers of the rete mucosum, close to the papillæ. Chemically it corresponds to melanin, the black pigment of the choroid and the hair, which contains iron but is not directly related to the orange series of pigments derived from hæmoglobin, as bilirubin and uro-erythrin, nor to the yellow series which gives colour to the fat, to the corpora lutea, and to the serum. Pigment is also to be found in the cuticle, and in the papillary layer of the cutis, where it is deposited in the connective-tissue corpuscles, as it is in those pigmentary moles (Greenhow, 'Path. Trans.,' 1864, pp. 310—394).

It is a question whether the internal organs are discoloured in cases of disease of the adrenal bodies. If so, it is an exception.*

Asthenia.—Next to the discoloration of the skin, progressive muscular weakness is the most striking and important of the symptoms of Addison's disease. The patient becomes more and more languid as it advances; he takes to his bed, and his prostration increases until he becomes unable to sit up. If his head is raised, faintness and giddiness follow, and fatal syncope may occur even early in the disease.

There is often some loss of flesh; but it is not extreme except from concomitant phthisis, and a certain amount of fat may still be found after death. Anæmia, though usually present, is also moderate.†

The muscular action of the *heart* is remarkably weak, and the *pulse* very small and compressible. Breathlessness, palpitation after any muscular effort, frequent sighing or yawning, and persistent hiccough, are other sym-

* Addison's work contains drawings of a mesentery, intestine, and omentum, over all of which numerous minute black spots were scattered. They were taken, however, from a doubtful case, and the pigmentation of the peritoneum may have been due to a former attack of peritonitis. The late Dr Carrington once observed the normal pigmentation of the pia mater covering the bulb much intensified in a case of Addison's disease.

† The fact that asthenia and not anæmia is the characteristic symptom of the disease seems to be not generally understood in Germany, judging from an excellent lecture by Professor Nothnagel reported in the 'Medical Press and Circular,' Feb. 12th and 19th, 1890.

ptoms more or less constant, and the patient may complain of pain in the loins or the epigastrium.

Nausea, with retching and vomiting, is very rarely wanting, and sometimes the gastric symptoms are the most prominent as well as the earliest. The greatest danger of fatal swooning is after a severe bout of sickness.

The urine is free from albumen, and is not darker than usual.* The blood is normal or only slightly deficient in hæmoglobin, and the temperature is seldom raised except by phthisis or other complications. As a rule, it is subnormal.

Course.—The development of Addison's disease is gradual, and the order of the symptoms variable. Occasionally the skin becomes dark long before the general health begins to fail. Thus, a young lady, whose case was recorded by Addison, had become "bronzed" for a year before her death, but appeared ill during only about four months; and a patient of Dr Robert Harris, of Southport, had been noticed by his wife to be getting darker for two years, but had been troubled by other symptoms for only six months. In the majority of cases, however, the patient suffers from progressive asthenia for a considerable period before melasma becomes noticeable, and in a few unusually rapid and severe cases, death may occur before the skin perceptibly darkens.

In a series of 228 cases, collected by Greenhow, there were twenty-nine in which, when the patients died, the skin was either not bronzed at all, or only slightly; but in eighteen of these some other disease was likewise present, which probably accelerated the fatal issue. In each of the remaining eleven cases the patient's illness had been comparatively short; in one only had it lasted eight months, and in the rest less than five. One case has occurred at Guy's Hospital in which no discoloration had appeared, although symptoms had been present for twelve months before death. Probably the skin is always more or less affected when the other symptoms of Addison's disease have lasted much over a year.

Although the course of this remarkable malady is progressive, its progress is by no means uniform, and is sometimes interrupted by apparent improvement, for a time.

Death sometimes takes place very gradually. The mind may be clear to the last, or the patient may lie in a drowsy and semi-comatose state, from which, however, he may be roused to give pertinent though slow answers. In such cases the temperature falls considerably below normal. Occasionally death is preceded by muttering delirium, and one of Greenhow's patients had a convulsive fit, and lay for hours with closed jaws and rigid limbs; whenever he was touched, muscular twitchings took place. In other cases the end occurs unexpectedly, most often from syncope.

A young man under Wilks was getting better of eczema, when he was attacked with diarrhoea and vomiting; he became rapidly prostrate, and died within twenty-four hours. Adrenal disease had never been suspected, but was found to be the cause of death.

A girl, a patient of Greenhow, although weak and supposed to be sunburnt, attended school until about a week before she died. A patient under the writer's care, who was in hospital with Addison's disease, was so much relieved that he was about to go home, when he became suddenly worse and died in a few hours. Dr Wilson Smith, of Bath, has published a case of

* The hæmatoporphyrin and urobilin discovered by MacMunn in the urine appear not to be constant, and occur separately as well as together. On the urine in Addison's disease see Rosenstirn's paper ('Virch. Arch.' lxi, p. 27).

typical Addison's disease in a girl of fifteen, which ended with suppression of urine from acute early nephritis ('Guy's Hosp. Rep.,' liv, p. 229).

The *duration* of the disease appears to be very variable. At least there are great differences in the length of time between the first symptoms and the death of the patient; but it must be admitted that the period at which the suprarenal bodies are attacked cannot be determined. Wilks some years ago stated that the average duration of the cases which he had collected was eighteen months. We have seen that the symptoms may last only a week or ten days before death. The longest case was one on which Dr Fagge made a *post-mortem* examination in 1865; this had been diagnosed by Sir William Gull when the patient was first admitted into the hospital in 1860, and he then said that his skin had already been dark for two years. In this instance, therefore, the disease had probably lasted not less than seven years.

Diagnosis.—Spurious and aberrant cases.—Addison included four cases of cancer of the suprarenal capsules in his original list, and supposed that any affection completely destroying the organs would give rise to the characteristic symptoms. It was Wilks who first maintained that in all genuine instances one particular morbid change in the capsules is found. He showed that in Addison's cancerous cases the proper symptoms were not really present, and he also first defined the characters of the discoloration of the skin. In 1875 Greenhow was able to cite 183 recorded cases, in every one of which the special form of discoloration of the skin, and some at least of the constitutional symptoms, were present.

The cases which have been supposed to lead to conclusions adverse to those maintained by Wilks and Greenhow fall into two groups.

In the first group come those in which the capsules have been the seat of tuberculous disease *without the characteristic symptoms*. We have seen that in some uncomplicated cases of Addison's disease death has occurred before the time when bronzing of the skin usually develops; and that in others a rapidly fatal issue has been due to some other malady, such as phthisis or Bright's disease, by which the constitutional symptoms of the adrenal disease were masked. Apart from such cases, Greenhow could state that in every recorded instance of suprarenal disease without symptoms, the anatomical lesion of the capsules was not the one above described.

In most instances it was cancer. The adrenals are liable to be the seat of primary malignant growths, as well as secondary nodules (*infra*, p. 733). In one of our cases each of them was three times as large as the kidney, so that there was a large tumour observable during the life of the patient.

Cases of melanoderma with more or less marked symptoms of Addison's disease have, however, been recorded by excellent observers, in which, instead of the usual changes, simple *atrophy of the adrenals* has been found: by Dr Wickham Legg ('St Barth. Hosp. Rep.,' 1874), Mr Davy ('Path. Trans.,' xxxiii, p. 360), Dr Goodhart (*ibid.*, p. 340), and Dr B. Fenwick (*ibid.*, p. 354, with table of cases). See also the same 'Transactions' for 1885, and the 'Clinical Transactions' for 1886.

In an interesting paper published in the 'Lancet' for February 6th, 1886, Dr Churton, of Leeds, after relating a typical case of Addison's disease in a youth of nineteen, and five others of non-tuberculous lesions of the adrenals without symptoms, discussed the possibility of senile atrophy of these organs producing more or less modified clinical effects. But in the

patient aged seventy-two, whose case he describes, it is not impossible that the specific anatomical change of the adrenals was present; and, as he rightly states, these organs may be found of normal size and structure at an advanced age.

The second group of cases which have been supposed to justify doubts as to Addison's discovery consists of those in which bronzing of the skin has been said to exist *without the suprarenal affection*.

We must remember that other discolorations of the skin may be easily mistaken for the melasma of Addison's disease by an unpractised eye, although they are really different.

Thus, in leucoderma the skin becomes, as it were, "piebald." Some parts are much darker than natural; others are devoid of pigment, and the hairs on them are white. The white areas have very definite convex borders, while the bronzed parts shade off gradually into the healthy skin. Thus the absence of pigment in some places is far more obvious than its excess in others. A case of this kind was once sent up to Guy's Hospital from a distant county as one of adrenal disease with bronzed skin.

Addison himself included a case of leuco- and melano-dermia in his original work, but was convinced by Wilks that it was not a genuine case of suprarenal melasma, and would have excluded it if he had lived to publish a second edition of his book ('Medical Magazine,' 191, p. 650).

The pigmentation of syphilitic cachexia is very different in distribution from suprarenal melanoderma; it is irregular and unsymmetrical (*cf.* vol. i, p. 342). The discoloration, which in English patients most resembles that of Addison's disease, is that produced by arsenic, as more than one observer noticed in the recent epidemic of arsenical poisoning in South Lancashire. The cases the writer has seen were more grey and less brown—more like the tint produced by silver taken internally, and less like the tint produced by walnut-juice applied externally.

As Greenhow remarked, elderly persons of unclean habits often have the skin of the back, chest, and abdomen deeply pigmented, so that disease of the adrenals might be suspected. Beside the freedom of the face from discoloration, the roughness of the cuticle in such cases affords a distinction. The chloasma of pregnant women, the discoloration of the skin produced by malaria, jaundice, and even tinæa versicolor, have each been mistaken for the bronzed skin of suprarenal disease. It would be difficult to recognise Addison's disease in a mulatto.*

The ink-like stains of the labial and buccal mucous membrane, and the deep coloration of the genitals, are perhaps the most decisive points for diagnosis. But the other symptoms above described usually enable one who has seen several cases to recognise the disease even before the melasma has become developed.

The muscular weakness without disturbance of the bladder, the extreme feebleness of the heart's action, the gastric symptoms, and the absence of extreme anæmia, are the most characteristic.

If there is much emaciation it is usually due to concomitant phthisis or suppuration. The "pearly conjunctiva" has often led to confusion with the totally different set of symptoms and anatomical lesions also described by Addison under the name of Idiopathic anæmia, now generally called "pernicious." Other cases mistaken for Addison's disease or *vice versâ* are

* The late Dr Bevan Rake recognised Addison's disease in a Hindoo who died in Trinidad ('Lancet,' 1889).

irritant poisoning, gastric ulcer or cancer, fatty degeneration of the heart, and phthisis in a melanochoic patient.

In 1864 the writer saw a case with decided but not obtrusive pigmentation, and other symptoms of Addison's disease, in the late Dr Hughes Bennett's wards in Edinburgh. He had not seen a case, and did not believe the diagnosis. Two years later Dr Murchison brought an adrenal before the Pathological Society which had been sent him from Aberdeen, but without any history. The name and age of the patient, however, were ascertained, and proved that the specimen shown belonged to the Edinburgh patient ('Path. Trans.,' vol. xvii, p. 396).

The most extraordinary instance of perversity is perhaps that recorded in 'Virchow's Archiv' (1870). In a case of scleroderma, large patches of the skin in succession became deeply pigmented. The patient, an old woman, had Bright's disease, and died of pneumonia. Her suprarenal capsules were healthy; but her physician, instead of seeing that his diagnosis was altogether wrong, proceeded to frame a new theory of Addison's disease, ascribing it to a functional disturbance of the cerebro-spinal system.

Ætiology.—Addison's disease occurs much more often in *males* than in females. In collected cases of Greenhow's the proportion was 119 to 64.

The *age* of the patient is generally between twenty and fifty, but instances have been met with in children of five, eleven, and thirteen years of age. It is not hereditary, and does not seem to be less rare in tuberculous families than in others.

In many cases vertebral disease has preceded the disease. Wilks first drew attention to this fact, and Greenhow mentions eighteen instances. The lower dorsal or upper lumbar vertebræ are generally the seat of the mischief, with secondary abscesses in or near the psoas muscles. In some cases the vertebral disease was directly traceable to injury, in others it was clearly tuberculous caries. The most frequent complication beside this vertebral disease is phthisis.

Pathology.—The late Dr Habershon in 1863 dissected out the semilunar ganglia in a typical case, and found them surrounded by dense fibrous tissue; and similar observations have been made by many pathologists, both in England and abroad. It has been supposed that the contracting fibrous tissues which form around the capsules, just as cicatrices of vomicae in the lungs or cirrhosis of the liver or kidneys, compress and strangle the parenchyma. The semilunar ganglia have also been found enlarged, and under the microscope their cells have appeared opaque and granular (Tuckwell, 'Path. Trans.,' 1868). It has even been maintained that the symptoms of Addison's disease may be caused by affections of the semilunar ganglia independently of any primary change in the suprarenal bodies.*

But the pigmentation and wasting of sympathetic nerves and ganglia are of very doubtful significance, as Dr Hale White has shown ('Guy's Hosp. Rep.,' vol. xlvii, p. 44), while the functions they subserve probably all belong to the spinal centres from which they are derived. There is no evidence that the cardiac nerves are thus affected, and none that the ascertained functions of the splanchnic nerves are altered.

Still less does the nervous hypothesis explain the peculiar pigmentation of the skin, although this also was attributed by Jaccoud to irritation of the vaso-motor nerves.

The medulla of the adrenals, where the tuberculous growth begins, contains dark pigment, and this has been connected with Addison's melasma;

* Sir William Jenner, when President of the Pathological Society, mentioned such an instance as having come under his observation (cf. *infra*, p. 764); and another is reported by Drs Barlow and Coupland ('Path. Trans.,' 1885).

but if there were any retained pigment circulating in the blood, we should expect to find it in the urine and other excretions.*

Moreover, chronic inflammatory contraction of the post-peritoneal tissues about the solar plexus is often found without any of the symptoms above mentioned being produced during life.

The function of the adrenal bodies is at present an unsolved mystery. They are richly supplied with nerves, and the medulla is deeply pigmented, while the cortex is arranged somewhat like a tubular gland, though without a duct.

Possibly the adrenals represent a no longer active organ, and if their meaning in the economy is ever discovered it may be found to be chiefly connected with the physiology of foetal life.

What is taught by embryology and comparative anatomy is that the adrenals are composed of two distinct parts which have coalesced as cortex and medulla, both apparently mesoblastic, but the latter probably nervous and ultimately epiblastic in origin, while the former is possibly hypoblastic, and comparatively more important in the foetus than after birth.† It is a singular fact, first observed by Hewson, that in an encephalous foetus the adrenals are usually absent or imperfectly formed.‡

Of late years we have learned that even secreting glands have other functions than secretion. The dictum of Treviranus, so well illustrated by Paget, that every organ of the body is excretory in relation to the rest has now received a new and very definite meaning.

As the presence or absence of the testes in the adult male produces far-reaching effects on the antlers in stags, on the feathers in birds, and on secondary sexual characters throughout the vertebrate classes, including man himself, so we have learnt from Lancereaux and from Mering and von Minckowsky that complete removal or destruction of the pancreas allows of glucose accumulating in the blood (vol. i, p. 1014), and from the experiments of Dr Bradford that removal of part of the kidney causes increased output of urea, with other collateral effects.

So also it is certain in the case of the thyroid, a non-secreting glandular organ in structure, that the cretinoid condition described by Gull, myxœdema as it has since been styled, is due, like the congenital cretinism with which it is allied, to disease or atrophy or removal of the "ductless gland" (vol. i, p. 455).

There is therefore great probability that the adrenal "glands" have an analogous effect in health; and this probability has been made a certainty by the observations of Schäfer and Oliver, which establish the effect of the medulla of the adrenals in raising the blood-pressure in the systemic arteries, with increased cardiac action and contraction of the arterioles; while the removal of the organs produces extreme feebleness of the heart and of the voluntary muscles.§

At present, therefore, the evidence is that the seat of Addison's disease is

* See Dr Creighton's paper on the "Adrenals in the Horse," *Journ. Anat. and Phys.*, xiii, 51, and Dr MacMunn's interesting account of the spectroscopic characters of the pigments extracted from the adrenals, in the *Brit. Med. Journ.* for Feb. 4th, 1888, vol. i, p. 233; also Dr Auld's article in his 'Selected Researches,' p. 74.

† See Balfour's account in 'Elasmobranch Fishes' (1878), and Weldon's article (*Quart. Journ. Micro. Sci.*, 1882).

‡ See Lomer's paper in the 98th vol. of 'Virchow's Archiv,' and an interesting thesis by Dr K. Biesing (Bonn, 1886) with accounts of thirteen dissections, in which the sympathetic ganglia and plexus were not affected.

§ These experiments are confirmed by those of Drs Moore and Purrington (*American Journ. of Phys.*, March and June, 1900).

in the adrenals themselves—or more particularly in their medulla; and that its symptoms are due not to extrinsic nervous lesions, nor to bacteria, but to destruction of an organ the secretion of which is essential to life.

Removal of the capsules in animals is speedily followed by death; and when an infusion of the organ is injected, it also proves fatal.*

Of late years the opinion of some pathologists, to which Dr Rolleston in his *Gulstonian Lectures* inclined, has been that Addison was, after all, right in including all cases of destruction of the adrenals by cancer or other causes, and Wilks and Greenhow wrong. To this the writer cannot assent. Primary cancer only attacks one of the two organs, and therefore always leaves enough adrenal tissue to avert the characteristic combination of symptoms. Secondary cancer only destroys as much as one or two nodules can do; for the primary disease brings about death long before the whole of even one capsule is destroyed. Even a small portion of thyroid protects from myxœdema, and even a fragment of pancreas preserves from diabetes.

Moreover, as in other cases, the manner of destruction is no less important than its extent. Both adrenals must be affected, and by the gradual process of tuberculous, caseous, and calcareous ulceration and fibrous contraction which we see in phthisis or in a tuberculous lymph-gland. We do not see the symptoms of Bright's disease follow cancer of one kidney, nor those of myxœdema cancer of the thyroid, nor those of phthisis cancer of the lung. All the best marked clinical cases of Addison's disease are associated with tuberculous inflammation of the adrenals; and if any exceptions do occur it is in cases of atrophy of the capsules—also a slowly destructive process.

Prognosis and treatment.—Hitherto no proof has been given that recovery from this disease ever takes place. But many patients, after having been kept in a hospital for a long time, and perhaps after having been admitted again, depart in tolerable health and are lost sight of. In the case already mentioned, of a young man in whom Addison's disease was diagnosed five years before he died, the morbid appearances which were found in the adrenals seemed to show that they had been destroyed for a long time. A case of bronzing of the skin in a young woman whose adrenals were found diseased after death was recorded by Dr Kirby, with the history that similar symptoms had appeared four years before her death and had completely disappeared.

The treatment of Addison's disease is at present little but an attempt to alleviate symptoms. Cod-liver oil and like treatment found useful in other inaccessible tuberculous organs may be prescribed, but without much expectation of benefit. Several cases have been treated by injection or swallowing of extract of the suprarenal capsules of sheep, but the result has not been what the analogy of thyroid feeding led us to hope. In the case of a lady whom the writer saw several years ago, he prescribed this treatment, and heard afterwards of the most marvellous improvement; but some months later, on making further inquiries, he was told that the patient had lately died. (See Dr C. R. Box's report on eight cases of Addison's disease from 'St Thomas's Hospital Reports,' vol. xxvi, 1901.)

OTHER LESIONS OF THE ADRENALS.—The commonest of these is hæmorrhage, not at all infrequent as a congenital condition, and probably pro-

* Experiments were made by Brown-Séquard in 1858 and by Tizzoni in 1889; by Abelous and Langlois in 1894, and by Schäfer (who used monkeys) in 1894-5.

duced by prolonged parturition, with the consequent congestion of the internal organs. The hæmorrhage is into the soft and vascular medulla. In chronic heart disease, and also in purpura and other hæmorrhagic disease, a similar condition has been observed in adults.

The writer has more than once observed well-marked lardaceous degeneration of the cortex, and also embolism with suppuration from septic endocarditis.

Simple atrophy of the adrenals has been met with; its origin is quite uncertain, but in some cases there was no sign of surrounding "cirrhosis" of the post-peritoneal connective tissue or of a cirrhotic condition of the adrenal following tuberculous disease. We must remember that these organs attain their largest comparative size before birth, and the shrinking which is normal may, in some of these cases, be only a senile change.

Cancer is not very uncommon; most often two or three secondary deposits, occasionally a primary growth of true alveolar carcinoma in one adrenal, more often perhaps a sarcoma. The writer once saw a sarcoma occupying each capsule in an infant, without any other organ being affected. There was no melasma present.

Fatty degeneration is very common, affecting the cortical part of the capsule. In some degree it may almost be considered a normal condition, as normal perhaps as moderate fatty infiltration of the liver.

Minute adenomata may often be seen projecting from a healthy adrenal. They reproduce the structure of the cortex. Others have been discovered in the connective tissue around the viscus, and they occasionally grow to be as large as a pea. They are innocent growths, which cause, it need scarcely be said, no symptoms, and may be compared to the lienculi often found in the gastro-splenic omentum.

DISEASES OF THE PANCREAS AND SPLEEN, ETC.

“Quid te exempta juvat de spinibus una.”—HORACE.

Diseases of the Pancreas—Calculi—Abscess—Hæmorrhage—Acute inflammation—Induration—Cysts—Hydatids—Cancer.

Diseases of the Spleen—Abscess—Febrile softening—Induration—Swelling—Atrophy—Embolism—The spleen in malaria, in rickets, in leuchæmia, in Hodgkin's disease—The lardaceous spleen—New growths.

Diseases of the Lympharia.

Diseases of the Testes and Ovaries.

Diseases of the Thyroid Body—Exophthalmic Goitre.

DISEASES OF THE PANCREAS.—Like the salivary glands which open into the mouth, the important abdominal gland (*Bauch-Speicheldrüse* of the Germans), which so closely resembles them in structure and function, is little liable to ordinary inflammation, and there is not known to be any zymotic affection of the pancreas answering to that of the parotid and submaxillary glands in mumps.

Calculi have been found obstructing and dilating the duct of Wirsung, like those found in that of Stensen or Steno. In one instance there were numerous calculi, of which several are preserved in the museum of Guy's Hospital (1462-4), and others were analysed by the late Dr Golding-Bird, and found to consist of phosphates of lime and magnesia, with oxalate of lime, but without carbonate.

Mr Gould has recorded a case of a pancreatic calculus pressing upon the common bile-duct, and thus causing jaundice and enlarged gall-bladder ('Clinical Trans.,' 1899, vol. xxxii, p. 59).

When from any cause obstruction has occurred to the passage of pancreatic secretion into the duodenum, we should expect, on physiological grounds, that the digestion of fatty matters would be suspended or at least impaired; and in several cases, notably that of a young woman, a patient of Bright's, whose symptoms during life were "emaciation, diarrhœa, and the passage of fatty stools," the pancreatic and biliary ducts were found after death to be occluded by a malignant growth. But these cases are exceptional.

Wilks and Moxon describe suppuration of the pancreas in the later stages of enteric fever, a condition we may compare with the parotid buboes which are occasional sequelæ of that disease.

In some cases of abscess in the head of the pancreas, the pressure thus caused on the portal vein has led to extreme and rapidly recurrent ascites (see a case reported by Dr Musser, of Philadelphia, in the 'American Journal of Medical Sciences,' April, 1886).

Hæmorrhage.—The tissue of the pancreas is liable to extravasation of blood, though in a less degree than the medulla of the adrenal capsule—

either as the result of abdominal contusion or without apparent cause. In contrast to adrenal hæmorrhage, that of the pancreas is scarcely ever seen in infancy or childhood, and is most common in the later stages of life (*cf.* p. 732). In many cases the persons in whom it is found after death have been intemperate, but perhaps as often this is not the case. No large vessel gives way, but there is oozing and infiltration into the connective tissue of the gland.

Certain cases are due to purpura or to chronic cardiac disease, others to violence, and others are apparently spontaneous. Possibly effused blood from the pancreas pressed on the solar plexus and produced fatal effects on the heart in Sir Astley Cooper's famous case (p. 181). That slight hæmorrhage is recovered from, is plain from the rusty stains and hæmatoidin crystals which are often found in the pancreas after death from recent hæmorrhage. Pancreatic hæmorrhage is often unaccompanied by clinical symptoms; but cases are recorded of epigastric pain and collapse (see an excellent monograph by Dr Fitz, of Harvard University, 1889).

Acute pancreatitis.—The hæmorrhage just described is often accompanied with acute, sometimes suppurative, inflammation of the pancreas. Classen, Klebs, Zenker, and the late Dr Wm. Draper, of New York, were among the first to diagnose and describe this severe and dangerous disease. Dr Fitz has contributed an account to 'Allbutt's System' (vol. iv, p. 264).

The more frequent cases are hæmorrhagic, and are characterised—in addition to the sudden and severe pain, followed by collapse, above mentioned—by vomiting, occasionally with hæmatemesis, and by rise of temperature. There is much swelling, extreme tenderness and immobility of the abdomen, with thoracic respiration, pointing to peritonitis localised in the epigastric region or extending to the whole abdomen.

After death the pancreas is found softened as well as hæmorrhagic; but in some cases there is little or no bleeding, only acute interstitial suppuration, and in others actual necrosis of the pancreatic tissue.

The suppurative and gangrenous forms are sometimes secondary to hæmorrhage, but sometimes they occur independently of it, and, as a rule, are attended by a condition of the connective tissue known as fat necrosis. The origin is probably bacterial infection from the duodenum through the pancreatic duct; the *B. coli communis* has been found in these cases to be present in the pancreas.

The symptoms during life are like those of acute intestinal strangulation or of perforation of the stomach, gall-bladder, or appendix cæci. There is nothing absolutely distinctive between these events, for, indeed, the same immediate cause produces the symptoms—the shock and collapse with sudden pain, and the subsequent signs of peritonitis. The situation of the swelling may lead to a diagnosis, or the previous history may point to perforation of a gastric ulcer. Dr Pitt has reported a fatal case in the 'Clinical Transactions' for 1899, and two others, recorded though not recognised, by the late Dr Habershon. In the same volume Dr Bryant published another acute and fatal case (p. 64).

Though the disease is so severe and the symptoms so grave, prognosis is not hopeless. All cases are not equally acute, and several patients have, as Osler reports, recovered from both pancreatitis and laparotomy. Others have survived rupture of a pancreatic abscess into the stomach or bowel. Mr Mayo Robson has successfully opened such a post-peritoneal abscess of pancreatic origin by a lumbar incision.

The immediate treatment of the urgent initial symptoms is to put the patient under the influence of morphia, and give him food and water only by the rectum, with stimulants only by subcutaneous injection of brandy or strychnia. The next thing is to form a diagnosis and decide whether and how soon to call in surgical aid.

Induration.—A very different form of “inflammation” is the chronic indurative change which is occasionally seen in the pancreas, and is often called cirrhosis or sclerosis. This is usually secondary to long-standing congestion or cyanosis of the systemic veins from disease of the heart or lungs, or, as Dr White thinks, from portal congestion owing to cirrhosis of the liver; while Mr Robson connects the same condition of chronic inflammation of the pancreas with obstruction of its duct by gall-stones.

Cysts in the pancreas are occasionally met with, apart from mere cystic dilatation of the duct from obstruction by cancer or calculus. As in the kidney, the testes, and the mammary gland, there are retention-cysts in the pancreas. We have already seen how these may affect more than one organ simultaneously, most often the liver and kidneys (*supra*, pp. 566, 606), sometimes the brain as well (*ibid.*, note), and in one case the writer met with multiple microscopic and larger pancreatic cysts in a fatal case of simple cyst of the cerebellum (*Path. Trans.*, vol. xxxvi, p. 17). Their coincidence, however, was probably a mere accident. See article by Savage and Hale White (*ibid.*, vol. xxxiv, p. 1), and an article in ‘Virchow’s Archiv,’ by G. H. Rolleston and Kanthack (1892, Bd. cxxx, S. 488).

A single cyst sometimes attains a large size; in the following case at Guy’s Hospital it held six pints (Pitt and Jacobson, ‘Med.-Chir. Trans.’ 1891). An abstract is given in this article of the cases published since Senn’s papers in 1886-7.

A man aged twenty-one was kicked in the abdomen in 1886, since when he had been liable to attacks of abdominal pain. He was admitted in May, 1889, with jaundice and severe abdominal pain and vomiting. On examination a globular tumour was discovered in the epigastric region, over part of which the note on percussion was dull; but this area of dulness was variable. In June the tumour was aspirated, and half a pint of alkaline, opaque, greenish fluid, of sp. gr. 1015, and containing albumen, was drawn off. The jaundice diminished and the patient improved sufficiently to be able to go home. At the end of July the patient was readmitted, the pain, the jaundice, and the tumour having all recurred. The globular tumour now occupied the umbilical and epigastric regions, extending rather to the left of the middle line, moving with respiration, and lying behind the stomach, as shown by the varying note on percussion, and by the disappearance of the dulness when the stomach was distended artificially with gas. On August 17th the tumour was again aspirated, and eight ounces of alkaline fluid, sage-green in colour, were removed. This contained 11 grms. of albumen per litre, a large amount of tyrosin crystals, no copper-reducing substance, and no bile. It was therefore clear that the tumour was a pancreatic cyst. The cyst rapidly refilled, and Mr Jacobson performed laparotomy on August 22nd. The patient was able to get up a month later, and was discharged cured.

A very similar case in a boy of fourteen, also after an injury, was diagnosed and successfully treated by incision and drainage, by Mr Barker (*Clin. Trans.*, 1899, p. 121). The fluid was alkaline, albuminous, free from bile or urea, and possessed both amylolytic and proteolytic powers.

An almost exactly parallel third case, also traumatic, in a young man of twenty-two, where the cyst contained seven pints, is recorded by Dr S. Phillips, *Clin. Trans.*, 1901, p. 53.

An *echinococcus* cyst of the pancreas is extremely rare. The following case occurred in a boy six years old under the writer’s care:

He was admitted with jaundice, which had lasted seven months, and ascites, which had appeared six weeks before admission. The liver was felt much enlarged, and smooth and painless. The gall-bladder was also palpable, like a billiard ball in size and smooth-

ness. Paracentesis was performed, but vomiting came on, and the boy died after nearly a year's illness.

The bile-ducts and cystic ducts were much dilated, and the liver hard and smooth (cirrhosis from obstruction). The cause of obstruction was a large hydatid cyst embedded in the head of the pancreas. It measured $8\frac{1}{2}$ by $3\frac{1}{2}$ inches, and contained 250 c.c. of fluid.

Cancer.—Primary cancer of the pancreas is probably a somewhat rare disease. In this want of aptitude for malignant growths the pancreas resembles other racemose glands, with the striking exception of the mamma (due, perhaps, as Dr Creighton has suggested, to its functional activity being often interrupted and ceasing early), and differs from the testis. What is usually described as "cancer of the head of the pancreas" probably begins, as a rule, in the epithelium of the pancreatic duct or in that of the common bile-duct, and gradually involves the glandular structure. Hence it is almost always accompanied by jaundice, to which enlargement of the liver from secondary growths is subsequent. So that clinically, when there is no reason to suppose the stomach or rectum affected, malignant disease of the liver with early jaundice may be most probably ascribed to primary cancer of the head of the pancreas. Another point which has twice led the writer to a correct diagnosis is enlargement of the lymph-glands above the right clavicle.

Often the structure of the new growth is cylinder-celled or duct-cancer, but sometimes it is of the ordinary glandiform type, as in one exceptional case of the writer's in 1887, where the autopsy showed primary cancer of the splenic end of the pancreas. Like other epithelial structures, the pancreas is very rarely the seat of secondary cancer.

During the fourteen years 1884—1897, Dr Hale White found *post-mortem* records of only 31 cases of primary malignant disease of the pancreas out of a total 6708 autopsies at Guy's Hospital. As we should expect the majority of these 31 cases were carcinoma; one ascertained sarcoma occurred in a man under thirty, and two others in women under thirty-five.

The seat of the growth is, in the great majority of cases, in the head of the pancreas—probably in nine cases out of ten.

The most characteristic symptoms are pain, vomiting, and icterus. A palpable abdominal tumour and ascites are much less frequent, and a distended gall-bladder is only occasionally palpable.

Bright recorded three cases in which the evacuations contained oily matter, which separated itself and formed a thick crust, or globules like melted tallow, or a thin fatty filtrate; and in all three cancer of the head of the pancreas was found after death ('Med.-Chir. Trans.' 1833). This is just what we should expect would be the result of occlusion of the pancreatic juice from the fatty portion of the chyme; and it is remarkable that similar cases are not oftener met with.

When *lymphoma* or lympho-sarcoma affects the pancreas, as is occasionally seen, the new growth probably begins in the small lymph-glands, which are found in the pancreas as well as in the parotid. *Tubercle* very rarely affects the pancreas.

Atrophy of the pancreas is the most frequent lesion of this viscus found in cases of diabetes. It is sometimes said to be associated with pigmentation of the skin, a condition described in France as *diabète bronzé*.

Fatty degeneration of the glandular epithelium of the pancreas is not uncommon, but does not appear to be of clinical importance. It is quite distinct from the fat-necrosis which is a frequent concomitant of acute hæmorrhagic pancreatitis.

DISEASES OF THE SPLEEN are remarkable in being all secondary to, or forming parts of, other morbid processes. The viscus is very rarely the seat of acute suppuration, of chronic fibrous inflammation, of diffuse splenitis, or of any new growth, innocent or malignant.

a. An extremely rare case of abscess of the spleen was recorded by Bright in the 'Guy's Hospital Reports' for 1838; about half of the substance of the spleen was involved, and there was an opening into the colon. He met with another case, in which a sloughing abscess communicated with the stomach; but it is perhaps doubtful whether the original seat of the suppuration in this instance was the spleen itself. Dr Caton, of Liverpool, has recorded a case which ended favourably ('Brit. Med. Journ.,' 1888, i, 586).

The symptoms of splenic abscess are obscure, consisting chiefly of pain in the upper part of the abdomen, frequent vomiting, rigors, pyrexia, and emaciation. A more or less distinct fulness may also be discovered in the left hypochondrium.

During life it would be impossible to distinguish such cases from those of suppuration around the spleen, which are much more frequent. In the 'Guy's Hospital Reports' for 1874 nine or ten such instances were recorded by Dr Fagge as examples of subdiaphragmatic abscess (*supra*, pp. 483, 530-31). In some of them there was a clear history of a blow or a fall having been the starting-point of the disease; in others a chronic perforating ulcer of the stomach set up suppuration.

In 1870 Dr Fagge was consulted by a gentleman aged fifty-six on account of a rounded, tender, and rather painful swelling, occupying the left side of a very protuberant abdomen. It seemed clearly to be an enlarged spleen, and this was confirmed by his pallid appearance, and by the fact that there was a decided excess of leucocytes in the blood. However, the skin over the tumour gradually became reddened, hot, and indurated, and at the end of a month Mr Durham made a puncture with a small trocar, and drew off some pus. For some months afterwards there was a discharge of matter from the opening, but at last it closed, and the swelling disappeared. Still no doubt was entertained that the case had been one of abscess of the spleen. But ten months later a second large tumour formed in the right side of the abdomen. This also was opened, and a great quantity of very foetid pus escaped. The patient was now exceedingly prostrate and ill; but he again recovered, and in 1876 was in excellent health. Probably the real seat of both abscesses was between the layers of muscles in the walls of the abdomen.

Another case was that of a woman who was admitted into Guy's Hospital eleven days after having crossed the Channel on a very rough day. She had been very sea-sick, and had also been thrown out of her berth upon the cabin floor. A large tumour rapidly formed in the epigastrium, and in the left hypochondrium there was a second mass in the position of the spleen.

In three other cases a subdiaphragmatic abscess perforated the pleura and set up fatal empyema.

b. In all fevers the spleen is the seat of congestion, and is found after death soft and more or less swollen. In typhus, acute pyæmia, and tuberculosis, scarlatina and pneumonia, it is not obviously enlarged, but in enteric fever it is so, and can be often detected by palpation as well as percussion during life (*cf.* vol. i, pp. 116, 136).

The spleen is a favourite resort of pathogenic microbes in both men and animals. It is most swollen in enteric fever, in septic endocarditis, and in malarial fever. It is soft in these cases, and also in typhus and the other febrile infections, in which it is not enlarged.

The congested and swollen spleen of anthrax has been described in the first volume (p. 416). It gives the title of splenic fever to this disease in cattle, but is not so large when the patient is a man.

As the result of intermittent fever the spleen becomes swollen with each paroxysm, and after a time no longer returns to its natural size in the

interval, but continues gradually to increase, and so at last forms the characteristic ague-cake, large, firm, fleshy, and pigmented (vol. i, p. 392).

c. The enlarged spleen sometimes met with along with the non-alcoholic cirrhosis of the liver in children has been described above (p. 537).

The passive congestion produced by general systemic venous stasis, particularly in cases of cardiac disease, leads to hardening but not to swelling of the spleen; but that which is due to portal congestion, and particularly to cirrhosis of the liver, leads to its decided enlargement, except when this is mechanically prevented by a capsule thickened from chronic perisplenitis.

d. Emboli produce the well-known wedge-shaped blocks (*infarctus*), first deep red, then buff with a red border, then completely pale, and lastly shrivelling up into a deep scar. When the emboli, however, are septic, suppuration occurs in the spleen, with the characteristic microbes of pneumonia or the common strepto- or staphylococci. Hence a swollen and tender spleen with pyrexia and a cardiac bruit is a certain sign of ulcerative endocarditis.

Infective abscesses from general pyæmia are also met with in the spleen, but much less frequently than in the liver.

e. Miliary tubercles are extremely common in the spleen, especially in cases of general acute tuberculosis in children; and caseous tubercles also occur, but they are less frequent and seldom form vomicæ.

f. Lardaceous disease is frequent, either as a uniform infiltration, or affecting the Malpighian bodies only, and producing the well-known "sago-spleen." No local effects follow, and no symptoms result.

g. Gummata are only seldom found, and have to be carefully distinguished from the fibrous scars of old emboli; lymphatic new growths are not infrequent, and will be described in the following chapter under Hodgkin's disease (*infra*, p. 767).

The spleen in congenital syphilis has been found swollen, firm, and dense in texture, like the liver by a somewhat similar indurative fibrous change. In rickets the spleen is sometimes found below the ribs (vol. i, p. 535); but the enlargement does not appear to be always of the same character.

h. Primary malignant disease of the spleen is unknown, and even secondary nodules, whether of sarcoma or true carcinoma, are extremely rare.

i. Hydatids also are less common in this viscus than in the lung, kidney, or brain; when present they are often also found in the liver.

k. In children the spleen is large and typically healthy. As age advances it atrophies like other lymphatic organs—following the fate of the thymus, the tonsils, Peyer's patches, and the lymph-glands themselves.

Hypertrophy of the spleen is the only condition of this organ which possesses clinical as distinct from pathological interest. It is frequently associated with anæmia, and almost always with leuchæmia, when it constitutes part of the remarkable disease to be described in the next chapter.

Signs of splenic enlargement.—As was long ago shown by von Luschka, the long diameter of the spleen inclines downwards and forwards parallel with the ribs on the left side, and its normal extent corresponds very closely with the space from the ninth to the eleventh rib.

About one third of its upper part is covered by the inferior border of the lung, and thus inaccessible to percussion. Hence, as Weil and others have shown, the arch of splenic dulness forms a figure which is rounded in front and below, and flattened above; behind it has no definite limit, and merges insensibly into the renal dulness of the loin. Its length (in the long

diameter of the spleen) is about three inches; its breadth, in a direction at right angles with the length, is two or two and a half inches; the distance of the lower and anterior extremity of the organ from the edge of the costal cartilages is an inch and a half or two inches.

The patient may either be sitting upright or lying towards the right side upon the right shoulder and the right hip; but when he can stand upright, that is the best attitude to feel an enlarged spleen. The splenic dulness is often "superficial" or incomplete. Light percussion is therefore needed for its determination; and in some cases, especially when there is much subcutaneous fat or œdema, when the lung is emphysematous, or when the stomach and the colon are much distended, its extent cannot be accurately mapped out.

When the spleen becomes enlarged, its lower and anterior extremity projects more and more downwards and forwards, and soon can be felt below the costal cartilages, as an "abdominal tumour."

Guttmann met with three cases in which a spleen of normal size was displaced so as to lie far below its usual seat. In one instance, that of a man aged thirty, the organ was put back and kept in position by a bandage, whereupon the pain for which he had long been treated immediately disappeared. In another patient, a woman aged forty-eight, the dislocation of the spleen began as the result of a violent physical effort. In the third case the diagnosis was verified by extirpation of the organ; recovery after the operation took place in a fortnight.

Floating spleen.—A spleen enlarged from ague or any other cause is liable to stretch its attachments and become loose in the abdomen. Such moveable or "wandering spleens" are of clinical interest, as forming abdominal tumours. They are much more rare than floating kidneys, and usually more readily recognised. They are more common in women than in men (see a monograph by Dr F. C. Shattuck, Boston, 1878). Such "wandering spleens" have lately been successfully removed, and they are frequently found in a state of partial or diffuse fibrous degeneration.

It is remarkable that small accessory spleens (*lienculi*) are frequently met with, like the accessory adrenals above noticed (p. 733). They may enlarge after removal or partial destruction of the organ, and thus compensate for its deficiency.

DISEASES OF THE LYMPH-GLANDS.—The spleen is anatomically a collection of mesoblastic tissue, modified into characteristic (leuco-) cytogenic, "adenoid" or lymphatic "pulp," and follicles, like those of the thymus, the tonsils and Peyer's patches, and the solitary follicles of the back of the tongue, the large intestine, and other parts of the alimentary canal. Closely allied to these structures are the so-called lymph-glands, which, like the spleen, are not epithelial in origin, and are not true (secreting) glands in function. Like the other cytogenic organs they form leucocytes; like them they are largest and most active in childhood and youth; and, like thymus, tonsils, agminated follicles, and spleen, they shrink with age.

The lymph-glands serve, like the spleen, for the accumulation and destruction by phagocytic lymph-corpuscles of bacterial invaders. As the tonsil is frequently the place for the infection of scarlatina and tuberculosis, so are Peyer's patches and the spleen the place for the accumulation of the bacilli of enteric fever. So also are the lymph-glands the portals through which septic, suppurative, syphilitic, tuberculous, and cancerous infection passes

into the thoracic duct and veins, and so pervades the whole body. Not only are pyogenic cocci detained in the glandular suppuration thus caused, but the infective toxines also (as Dr Sidney Martin has shown to be the case in the spleen), beside the unknown conveyers of lues and infective tumours. The natural experiment which puts this in a demonstrable shape is the accumulation of granules of carbon and mercuric sulphide in the axillary lymph-glands of men who have tattooed their arms during life.

Accordingly we find that most diseases of the lymph-glands are secondary; and the rule is obvious:—when we find glandular swelling or abscesses to look to the source from which that particular gland draws its lymph:—at the back of a child's head, to the scalp; at the angle of the jaw, to the tonsils; in the posterior triangle, to the structures in the posterior mediastinum and behind the peritoneum; in the axilla, to the mamma and the hand; in the groin, to the genital organs or the foot, or occasionally the gluteal or anal region.

Cervical and inguinal abscesses, tuberculous, syphilitic, and cancerous glands, and the buboes of the plague, are all secondary; and so are the lesions of the mediastinal, mesenteric, bronchial, and lumbar lymph-glands which are found after death from enteric fever, bronchitis, phthisis, mediastinal, and post-peritoneal growths, as well as in diseases of the tongue, the stomach, the large intestine, the kidney, the testis, and the pancreas.

While secondary cancer is so common in lymph-glands, primary cancer never (or hardly ever) attacks them. On the other hand, while secondary sarcoma is very rare, since sarcomatous infection, like carbuncle, spreads, as a rule, by the veins and not the lymphatics—primary sarcoma of a lymph-gland is not uncommon, particularly in the mediastinum.

Lymphoma or lymphadenoma is an hypertrophied gland or a new growth reproducing lymphatic tissue, and often difficult to distinguish histologically from sarcoma with small round cells.

The multiple hypertrophy of lymph-glands with frequent implication of the spleen constitutes the condition generally named after Hodgkin, which will be described as *Anæmia Lymphatica* in a following chapter (*infra*, p. 768). The glands are sometimes found enlarged in cases of splenic leuchæmia; and still more rarely leuchæmia is seen with hypertrophied glands but no hypertrophy of the spleen—such exceptional cases are best described as *leuchæmia lymphatica*.

Status lymphaticus.—Several cases have been described under this name, by Paultauf in Vienna, and by others in Germany and America.

The lymph-glands and spleen, the thymus, the marrow, and solitary follicles of the alimentary canal, all enlarge, and the patient—usually a child—dies from some intercurrent affection or from syncope (so-called *Thymustod*). It does not appear to the writer how these cases differ from Hodgkin's disease, running a more acute course than it usually does in adults, like tuberculosis and splenic anæmia in children.

Glandular fever is the name given to another clinical group of cases, first described by E. Pfeiffer in 1889, and since by those who have seen it as an epidemic among children in Germany and in the United States. The cervical lymph-glands are chiefly attacked, so that it might be mistaken for mumps. There is no exanthem, and the glands do not suppurate. The onset is sudden, the course acute, with pyrexia and other febrile symptoms, and the result usually favourable, though convalescence is tedious. It spreads through families and towns, apparently by contagion. In this

country cases have been described by Dr Donkin, Dr Dawson Williams ('Lancet,' 1897, vol. i, p. 160), and Dr Cantlie (in the same volume).

The writer was once sent to see a medical student, previously strong and healthy, who, with fever and profuse sweating and great loss of muscular strength, had also soft tumours in the groin and axillæ, which were clearly acutely enlarged lymph-glands. The spleen was not to be felt. No local or general infection could be traced, but one feared that it might be the first stage of acute glandular tuberculosis. However, under somewhat tentative treatment with full doses of quinine the temperature sank, the appetite returned, and in about a fortnight the young man, though very weak and with shrunken limbs, was able to go home into the country, where his gradual convalescence was completed without further interruption. The writer saw him a few months ago in robust health.

Lardaceous degeneration sometimes affects the lymphatic glands, whether normal or hypertrophied. Fatty metamorphosis does not appear to occur; but fibrous degeneration is not uncommon in old age.

The diseases of the abdominal viscera which remain, the testes and the ovaries, are, by the conventional arrangement in use, assigned to the departments of surgery and obstetrics respectively. The best account of their morbid anatomy is therefore found in treatises on pathology, and of the corresponding clinical disorders in those on practical surgery and diseases of women. But the student must not forget that such divisions of our art have no counterpart in nature.

Of the diseases of the *Ovaries*, the only ones which come within the scope of a treatise on general medicine are the cystic tumours of the ovarium and the parovarium, which have been already described, so far as is necessary for their diagnosis from encysted ascites (p. 496); and the functional disturbances of these organs which are sometimes associated with epilepsy, hysteria, and hysterio-epilepsy.

In medical practice it is important to remember how frequently acute peritonitis spreads upwards from the ovaries or Fallopian tubes, how an inflamed right ovary may simulate typhlitis, and how often obstruction or strangulation by external contractions or adhesions or bands is due to old adhesions from pelvic suppuration, and therefore is to be sought in the lowest part of the abdomen.

The diseases of the *Testis* are acute inflammation from injury, "metastatic" or "sympathetic" swelling from mumps or from renal calculus, epididymitis from gonorrhœa, tuberculous (or "strumous") orchitis, syphilitic orchitis, with gummata and chronic fibroid atrophy, cystic disease, enchondroma, and alveolar, usually soft, cancer.

It is important for the physician to remember that the testes are abdominal organs which have escaped from the general peritoneal cavity, and they should be examined in doubtful cases with the same care as the liver or spleen. The discovery of epididymitis or *fungus testis* will often throw light on cases of hæmaturia and pyelitis; while the presence of sarcocele may point to syphilis in other organs, or to malignant disease of the lumbar glands. The writer remembers an obscure nervous case which Sir William Gull was asked to see: he looked at the patient, put his hand under the bedclothes, and said, "Give him mercury."

DISEASES OF THE THYROID.—The thyroid "gland," as it is still called, resembles the suprarenal capsules in having the epithelial apparatus for secretion, but no ducts to carry off the products, if any. It has no connec-

tion in structure or pathology with the thymus—a lymphatic, non-epithelial viscus, which must be classed with the tonsils and Peyer's patches, the solitary follicles of the tongue, pharynx, stomach, and intestines, and with the lymph-glands, red marrow, and spleen.

Beside the thyroid itself, in man, as in dogs, rabbits, and other mammals, there are other smaller bodies of similar, though not identical, structure, and not quite constant in number. They are called parathyroids, and may be compared to accessory adrenals and supernumerary spleens.

Atrophy.—The remarkable disease, first described by Gull as a cretinoid condition, and since named myxœdema, described in the first volume (p. 1017), is dependent on the removal or atrophy of both thyroid and parathyroids; for the secretion of these structures it is which prevents men and women from being cretinoid, and infants from being lifelong cretins. When dried and supplied as food, it cures the less grave adult form of the malady, and the congenital myxœdema of the infant cretin (vol. i, p. 1015). The constituent of the dried thyroid powder which appears to be the essential agent, is present in the colloid contents of the thyroid during life, and has been ascertained to consist of a proteid and non-proteid part, with the latter of which iodine is combined. It has provisionally been named *thyro-iodine*.

Endemic and Sporadic Goitre.—This disease, otherwise known as Bronchocele,* consists in an enlargement of the thyroid body. Sometimes this is apparently a true hypertrophy or overgrowth of the normal thyroid tissue (parenchymatous goitre); sometimes it is an increase of the fibrous tissue at the expense of the alveoli, a chronic interstitial inflammation or hypertrophic "cirrhosis;" and frequently, in addition to this or independently, one or more cysts are formed, so that there is either a single very large hollow tumour full of colloid material, or a thyroid made up of a multitude of larger or smaller dilated alveoli, like the hypertrophic cystic kidneys described in a previous chapter (p. 664).

The bronchocele most often occupies both lobes and the isthmus, but may be confined to one side. It moves with the trachea in swallowing.

The geographical distribution of bronchocele is remarkable. In Switzerland and the Tyrol it is still as common as when Juvenal wrote of its exciting no wonder in the Alps;† and it is endemic in the Pyrenees, the Andes, and the Himalayas. But in the tableland of Bavaria and Würtemberg, and in the Peak district, the same Kropf, goitre or Derbyshire neck is found. It is nowhere endemic except in the neighbourhood of mountains and usually in limestone formations. It is common in some limestone districts in Canada (Adami, 'Montreal Medical Journ.,' January, 1900).

It is probably not hereditary, and no causal connection between endemic bronchocele and race or food or drink has at present been established. It is said to occur in the domestic animals of goitrous districts.

Sporadic goitre is met with in every country, but in England is very rare in young men and not frequent in women. When it occurs, it has the same characters as the endemic disease.

It has no generally injurious effect on the health, but there is always the fear that it may by its growth press upon the trachea and cause suffocation. This is particularly likely to occur when it encircles the windpipe, or when it grows downwards into the aperture of the thorax, where the trachea is liable to be compressed against the vertebræ or the ribs.

* The word bronchus is used in its old meaning of the trachea, so that bronchocele denotes a swelling or tumour of the windpipe.

† *Quis tumidum guttur miratur in Alpibus.*—*Sat.* xiii, v. 162.

Cases have been reported of an apparently hypertrophic goitre being propagated as an infective disorder in the bones and other organs ('Payne's Pathology,' p. 306; and 'Path. Trans.,' xxxiii, p. 291). Sarcoma and also true alveolar cancer have been observed in the thyroid. Mr Horsley has traced the latter to the parathyroid.

The only effectual internal medication of hypertrophic goitre is by iodine. This was long ago empirically prescribed in the ashes of seaweed, but is now most conveniently given in doses of five to ten or fifteen drops of tincture of iodine in a solution of iodide of potassium, taken three or four times a day before food. Injection of thyroid cysts with iodine or other irritants, or treating them by setons, seems to be now abandoned as ineffectual and dangerous.

When internal treatment fails, and in the case of cystic or malignant bronchocele, the surgeon's aid is needful.

Division of the isthmus, excision of a part, or complete removal of the thyroid, though once dangerous from hæmorrhage and from pyæmia, and still formidable operations, are now frequently performed. The improved methods of thyroidectomy, introduced by Prof. Kocher, of Bern, and carried out in this country by Mr Horsley and other surgeons, have changed a hazardous and scarcely justifiable operation into one comparatively safe, and in most cases successful. Only a small portion need be left to ensure against myxœdema (vol. i, p. 1019).

EXOPTHALMIC GOITRE.*—This remarkable disease must have attracted notice long before Graves's description of its striking characters in 1835.

The priority of Graves to Basedow is shown in the late Dr Beigel's article in Reynolds' 'System of Medicine' (vol. v, p. 368). Dr Osler has drawn attention to the still earlier account of the affection which was given by Caleb Parry, of Bath, in a posthumous volume of medical essays published in 1825. This, however, was duly recorded by Beigel in the article referred to, but by apparently a slip of the pen—not a printer's mistake, for it occurs twice on the page—the name stands as Percy. The correction was made in the first edition of the present work (vol. ii, p. 87), and Parry's observations were duly acknowledged by Stokes. It was Trousseau who proposed to name the disease after Graves.

The three constant symptoms are vascular turgescence of the thyroid, protrusion of the eyeballs, and frequent and disordered action of the heart, which often beats 100 to 150 in the minute, or even 168. Muscular tremors and sweating are nearly as frequent symptoms.

The great majority of the patients are young adult women. Some of the few cases recorded in children ran an acute but benign course. It is not a common disease, and in men is decidedly rare.† Exophthalmic goitre is not hereditary, and has no special geographical distribution.

As a rule, the accelerated action of the heart is the first symptom, exophthalmos follows by degrees, and then the thyroid enlargement; but this order is not constant, and occasionally, as in one of Trousseau's cases, the leading symptoms appear almost suddenly.

The thyroid enlargement is usually gradual, and never comparable in

* *Synonyms.*—Graves's disease (1835)—Basedow's disease (1840)—Parry's disease (1786, publ. 1825)—Tachycardia strumosa—Struma exophthalmica.

† An excellent portrait of exophthalmos in a man is in the 'Guy's Reports' for 1870 (vol. xv, p. 17). Dr Cleveland found in 84 cases, 9 men and 75 women (*ibid.*, lv, p. 191).

degree to that of endemic bronchocele. There is strong pulsation, and a systolic thrill in the neck, accompanied by a loud, high-pitched, but not musical arterial murmur. The thyroid (or sometimes one half of it only) is swollen and pulsating, with a strong tactile thrill: the carotid arteries pulsate as they do in the more severe cases of sigmoid regurgitation, but the pulse, though frequent, has not the water-hammer character. A venous hum is often present.

The staring of the eyeballs is usually well marked, but in early cases may have to be looked for. The pupils are, as a rule, dilated, and the eyeballs do not converge for near vision, nor move laterally so readily as in health. The sight is unaffected and the retina and optic disc normal. There is no strabismus, the act of blinking takes place as usual, and the orbicularis can generally close the palpebral aperture; but occasionally this is impossible even during sleep, and in some recorded cases the exposed cornea has sloughed and the eye been lost. The separation of the lids, particularly when the sclerotic can be seen all round the cornea, has been dignified as Stellwag's symptom; and the fact that when the patient looks downwards the upper lid does not follow the eyeball has been appropriated to von Gräfe.

Brailey and Eyre have observed, in five cases of exophthalmic goitre occurring in girls between eighteen and twenty-four, that there is pulsation of the retinal veins, cupping of the disc, limitation of the field, and impaired vision ('Guy's Hosp. Reports,' vol. lix, p. 65).

The tachycardia is accompanied by distressing palpitation and increased rapidity of action, and frequently leads to irregularity of rhythm.

The tremors follow in well-marked cases. They affect more frequently the fingers and hands, sometimes the arms, and only occasionally the head, the lips, or the tongue. Dr Ord has seen trembling affect the leg.

Pallor and diaphoresis are as common as tremors, though less characteristic. Pigmentation of the skin is much less frequently seen; it may affect the face, or the limbs, or the trunk, and is sometimes varied by patches of leucoderma. It was present 10 times in 84 cases.

The patient usually becomes thin as well as pale, and may suffer from hæmorrhages, as Trousseau long ago remarked. Quickened respiration and dyspnoea follow the cardiac excitement, and sometimes there are severe paroxysms with cyanosis. The temperature is, as a rule, subnormal, and the urine remains healthy, though often pale and abundant.

There is much muscular weakness, but the knee-jerks are unimpaired, and there is never paralysis.

The mental condition of patients suffering from Graves's disease is sometimes hysterical, but many sufferers are patient and reasonable throughout. The staring eyes, the trembling, and the thumping of the heart against the ribs give the patients a frightened air, which is very characteristic, but they do not seem to be really less courageous than others.

Vomiting is one of the less common and most formidable complications.

The course of exophthalmic goitre is, with few exceptions, a chronic one, lasting for years, and often for life. When death cuts short a case, it is usually from some intercurrent complication, particularly dilatation of the heart. Persistent vomiting or diarrhoea are of ill-omen.

Anatomy.—After death the thyroid is found less vascular than might be expected. Its tissue is not atrophied, and Greenfield describes hyperplasia of the alveoli lined with secreting epithelium. The heart is usually found

perfectly normal, neither dilated nor hypertrophied, and if it weighs above the average for the age and sex and weight of the patient, the increase is but small. The thyroid is often normal in structure, only occasionally cystic. The inferior cervical and other ganglia have been described as swollen or atrophied or pigmented; but these changes are accidental.* The protrusion of the eyes disappears after death. The spleen is sometimes moderately enlarged, and the thymus is often found larger than usual for the patient's age. Neither dilatation of the heart nor any other lesion of the vascular or nervous system is constant.

Pathology.—The concomitants of Graves's disease are functional and organic disorders of the heart. Chorea, shaking palsy, and hysteria are the nervous disorders one would compare with it clinically, myxœdema and Addison's disease the auto-intoxications which seem to approach its pathology. In some respects it forms a contrast to myxœdema, but in others it resembles it, and the one malady has succeeded the other. It is probably not due to hypertrophy and increased secretion of the thyroid. Apart from the fact that we have no well ascertained example of an organ doing more than its proper work, the symptoms do not correspond with such an origin.

Neither the hypothesis of a nervous origin of the disorder, nor that of over activity of the thyroid is satisfactory, for the former lacks a basis of anatomy and the latter a basis of chemistry. (See Dr Greenfield's 'Bradshaw Lecture,' 1893, and Mr Edmunds' 'Erasmus Wilson Lectures,' 1901.)

The *prognosis* is grave in the sense that (except in the rare acute cases in children) there is little natural tendency to recover, and still less power of stimulating that weak tendency. Hence, most cases run a protracted course, and, even when improvement takes place, are apt to relapse. On the other hand, there is little tendency to a directly fatal result, and death is most often due to some almost accidental intercurrent illness. Many cases which are never cured improve so much that the patient may live a useful life and enjoy an average of comfort. One patient whom the writer has seen from time to time for more than twenty-five years is still living and tolerably well.

Treatment is sadly ineffectual. Digitalis, even when pushed to very large doses, does not slow down the heart, and still less does it relieve the other symptoms. Cold locally applied often gives relief. Iron, arsenic, strychnine have all been recommended, and have all been tried and failed. Bromides, opium, cannabis indica, and belladonna have each been useful in certain cases, but none can be relied on. Regulation of diet, rest and change, sea voyages, galvanism, static electricity, and faradism, baths and mineral waters—are the prescriptions to which medicine is reduced.

Feeding with thyroid extract is theoretically a kind of homœopathy; practically it seems to be useless, but harmless.

Lastly, the various attempts to cure the disease by excision of the whole or parts of the thyroid, or division of the cervical sympathetic, appear to have no proved value to set against their undoubted danger.

* Dr Hale White has conclusively shown them to be either normal or senile or inconstant ('Guy's Hosp. Rep.' for 1889, vol. xlv, p. 11).

DISEASES OF THE BLOOD

ANÆMIA

“ At this, the blood the virgin’s cheek forsook,
A livid paleness spreads o’er all her look.”

POPE.

Blood diseases—Anæmia—Its general characters—Amount of blood—number of red corpuscles—amount of hæmoglobin—Changes in the blood-discs and leucocytes—reaction—density—coagulation—Anæmic murmurs—Dyspnœa—Fatty degeneration of the heart—Other symptoms—Causes and classification—Symptomatic or secondary and idiopathic or essential anæmia.

LEUCHÆMIA—*History—Definition and characteristic symptoms of leuchæmia—The spleen—The bones and the marrow—the lymph-glands—The blood in leuchæmia—The other organs—Symptoms and course—Ætiology—Pathology—Treatment—Leuchæmia medullaris—Leuchæmia lymphatica.*

ANÆMIA SPLENICA—*Characters of the blood and spleen—the glands—the liver—Symptoms and course—Diagnosis—Treatment.*

HODGKIN’S DISEASE—*Anæmia lymphatica—History—Ætiology—Morbid anatomy and pathology—Course, prognosis, and treatment.*

IDIOPATHIC ANÆMIA—*Grave, essential, or pernicious anæmia—History—Name—Antecedents—Ætiology—Age and sex—Symptoms and diagnosis—Anatomy—Course and prognosis—Treatment.*

CHLOROSIS—*Its relation to sex and age—to menstruation—to imperfect development of the heart and vessels—The blood in chlorosis—Pathology—Symptoms—Prognosis—Treatment.*

THE ancient humoral pathology assigned most diseases, at least in their origin, to *dyscrasia* or ill-tempering of the four humours of the body—blood formed in the liver, phlegm (*pituita*) in the pituitary gland, bile in the gall, and black bile in the spleen. When “black bile” was found not to exist, and phlegm was ascertained to be restricted to mucous surfaces, “im-

purities of the blood" from a "sluggish liver" was still the easy explanation of many obscure symptoms.

Even in recent times, fevers, syphilis, pyæmia, rheumatism, and cancer have been called "blood-diseases." But for this there is no adequate reason. There is no evidence that the blood of a person who is the subject of malignant disease behaves differently to physical, microscopical, or chemical tests from that of one in health; at the utmost it is the chief channel of infection in cases of sarcoma, as the lymph is the chief channel in epithelial carcinoma. Nor are the specific diseases, pyæmia, syphilis, and the infectious fevers, specially disorders of the blood. They affect the whole body. The poisons or microphytes which are their material basis circulate no doubt with the blood, but the circulation is only the carrier and not the primitive seat of the contagion. The spirillum of relapsing fever, the bacillus of anthrax, are the essence of these diseases, and no doubt are present in the blood; but they are found in the solid organs as well, and ought to be compared to the mineral which is carried by the blood to the tissues in cases of arsenical poisoning, or to the *filariæ* which are transported by the same means to various parts of the body. Uræmia may be probably explained as a chemical "intoxication" of the blood with retained urinary constituents or their products, but here again the poisoned blood is only a secondary to a lesion of the kidneys, just as the symptoms of diphtheria are due to the toxins secreted by its bacillus; but in both cases the blood is a mere vehicle.

The only "diseases of the blood"—maladies, that is, of which we cannot trace the seat further back than to the blood—are the following:

(1) Those in which the red blood-discs are less numerous or paler than natural, or both—the condition long recognised as *Anæmia*.*

Their shape and size may also be altered, as we shall see, and in one remarkable form of disease their scarcity is accompanied by an absolute increase of the colourless corpuscles. The amount of hæmoglobin in each disc as well as in the total blood may be diminished; and perhaps the amount of albumen and other constituents of the serum may be altered—but a diminished number and paler colour of red corpuscles are, one or both, always present.

Of *plethora*, the assumed opposite condition of too much instead of too little blood, we have no certain knowledge. In anæmia there is no proof that the mass of the blood is diminished; water is rapidly absorbed and makes thin blood, but the vascular system is as full as before. Nor is it permanently over-full. If blood is injected into the veins of animals the result of the experiment is that the foreign corpuscles are rapidly destroyed. Clinically we do not find the number of corpuscles or the amount of hæmoglobin increased in any form of disease. What used to be called plethora was only local congestion; and if general plethora exist at all, it is a condition not due to there being too much blood in the vessels, but to the distal vessels being contracted and the left ventricle strong, *i. e.* to a condition of high blood-pressure in the arteries.

A condition called "hydræmic plethora" has, however, been described

* Attempts have been made to improve upon this classical word by substituting *oligæmia* or *spanæmia*. But it is a mere quibble to say that there is not complete absence of blood in anæmia. We say a person is "bloodless" in English without meaning more than that his colour contrasts with the ruddy hue of health, and the Greek privative is used like corresponding prefixes in other languages.

not only as a supposed antecedent of dropsy (*cf. supra*, p. 619) but also as a demonstrable fact in some kinds of anæmia, and particularly in chlorosis. The supposition is that there is more serum in the vessels than in health, that they are therefore over-full, and the blood diluted, *i. e.* comparatively poor in corpuscular elements. The evidence for this hypothesis is set forth in a contribution by Dr Lorrain Smith to the 'Pathological Transactions,' 1900, vol. li, p. 311.

(2) *Hæmorrhagic diseases*.—In these the blood readily escapes from the vessels, without mechanical cause being demonstrable. Their true pathology is still obscure. More or less anæmia is usually antecedent as well as consequent to the hæmorrhage; but while in the former group of blood-diseases anæmia frequently leads to secondary hæmorrhage, here hæmorrhage is the primary and anæmia the secondary symptom. This group will form the subject of the next chapter, and the present one will be devoted to anæmia.

General account of anæmia.—It is a matter of common observation that the skin and mucous membranes often become pale from deficiency or pallor of the blood. In this, as in other cases, it is difficult to fix the boundary between health and disease. Some persons are naturally pale, however favourable the conditions under which they live. Others lose their colour from time to time if they live in large towns, work at sedentary occupations, or keep late hours; but a ruddy complexion returns when they go back to the country, under the influence of sunlight, fresh air, and exercise. No doubt many are habitually pallid in workshops, or cellars, or mines, who would look very different if they could spend their lives in light, well-ventilated rooms, or in the open air. Next to want of air and light, want of good food is probably the most frequent cause of pallor. Again, there are some who owe their colourless, sallow appearance to premature sexual indulgence. Excessive smoking, and smoking at too early an age, are also causes of anæmia.

Anæmia, from whatever cause, is characterised by pallor, not only of the skin, but of all visible mucous membranes. The tint varies widely, sometimes being white like marble, sometimes dingy, and sometimes decidedly yellow, like wax. The hands and finger-nails show the change as distinctly as the face; the tongue, the fauces, the lachrymal carunculæ, are all more or less pale and often yellowish. In the dead body the amount of blood in the internal parts is also deficient; the liver and kidneys look like wax, while the heart and the great vessels contain only shreds of coagulum and a little thin pink fluid.

Estimate of amount of blood.—The evidence derived from inspections after death seems to show that the total volume of the blood is diminished in the more severe cases of anæmia, but it is difficult to obtain proof of this fact, and almost impossible to estimate the degree of deficiency. The method proposed by Quincke is to count the proportion of red discs contained in the patient's blood, then transfusing into his circulation a known quantity of healthy blood, and soon afterwards again counting the red discs in a fresh specimen taken when the new blood and the old may be supposed to have become thoroughly mixed together. A simple formula gives the total volume of blood which was present in the patient's body before the operation. In two cases of "pernicious anæmia" Quincke estimated by this method that the blood formed only 4 or 5 per cent. of the body weight instead of 7 or 8 per cent., which is supposed to be about the normal pro-

portion. The experiments, however, can seldom be carried out accurately enough to yield trustworthy results.

Number of red corpuscles.—Whatever diminution there may be of the total volume of the blood in anæmia, diminution of the red corpuscles can be estimated with comparative facility.

The “numeration of blood-corpuscles” was commenced by Vierordt, and perfected by subsequent inventors, especially by Malassez (1872), Potain, Hayem, Gowers, Thoma, and Oliver. The principle is to dilute to a definite extent a measured quantity of blood, and then to count the number of red discs contained in a certain volume of the mixed liquid.

The instrument most used in England is the *hæmocytometer* of Gowers, a modification of the *compte-globule* of Malassez and the improved apparatus of Hayem and Nachet. (1) A small pipette, holding exactly 995 cubic millimetres, is filled with the diluting solution, which is then poured into a small glass jar or mixing vessel. A good solution, which leaves the corpuscles in a state favourable for observation, consists of sulphate of soda in distilled water, of sp. gr. 1025; but normal saline solution is also used, and various other mixtures. (2) The patient's finger is pricked with a lancet, so that a drop of blood escapes without much pressure. Five cubic millimetres of blood are then taken up by a fine capillary tube, graduated for the purpose, and are blown into the diluting fluid in the vessel. If there is a difficulty in getting exactly the right quantity of blood into the tube, the best way is to take up a little more than enough, and then to let the excess escape into a soft cloth. (3) The contents of the mixing-jar are well stirred up with a glass rod. (4) A drop of the mixed liquid is placed in the centre of a cell excavated in a microscopic slide. The cell is exactly one fifth of a millimetre deep, and its floor is ruled in tenth of millimetre squares. The slide rests on a metal slip, to which two springs are attached. (5) A cover-glass is next laid over the cell, in contact with the liquid in it; the springs are brought over the edges of the cover-glass, and keep it in position with a pressure which is always uniform; the slide is placed horizontally on the stage of a microscope, and this is focussed upon the squares in the floor of the cell. (6) In a few minutes the red discs are found to have settled down upon the squares by gravitation. The average number in a square is now counted, and this, multiplied by 10,000, gives the number contained in one cubic millimetre of the blood. The average number in normal blood is believed to be 5,000,000 in males and 4,500,000 in females. It is usual, however, to state the “corpuscular richness” of blood as a decimal fraction of the normal richness, this being taken at 5,000,000 to the cubic millimetre. The decimal figure may be obtained by dividing by 5 the number of corpuscles contained in 10 squares. Thus if the number in 10 squares is 332, the “corpuscular richness” is ‘66. (See the series of results obtained by Laache, of Christiania—‘Die Anämie,’ 1883.)

The modification of the hæmocytometer devised by Prof. Thoma in 1877, and carried out by the instrument maker Zeiss, is most in use in Germany and America. With the pipette devised by Sherrington it is probably the most accurate hæmocytometer, particularly with Abbé's counting chamber, but, like other apparatus, it needs practice to use it to the best advantage.

Amount of hæmoglobin.—The deficiency of red discs in proportion to plasma affords, however, an incomplete measure of the diminution in the amount of hæmoglobin in anæmia. For practical purposes this may be estimated with tolerable accuracy by an apparatus called a *Hæmochromometer* or *hæmoglobinometer*, for which we are again indebted to Malassez (‘Arch. de Phys.,’ 1877) and Gowers (‘Clinical Transactions,’ 1879).

It consists of two glass cylinders of equal diameter, which are placed side by side upon a small wooden stand. One of them is closed, having been filled with glycerine jelly, coloured by a mixture of carmine and picro-carmine of ammonia, so that its tint is that of blood diluted with water, in the proportion of one part of blood to a hundred of water. The other cylinder is graduated in such a manner that a space equal to two cubic centimetres has 100 divisions. It is open, and a little distilled water is poured into it. Some of the blood, of which the hæmoglobin is to be estimated, is now taken up by a capillary pipette, marked for twenty cubic millimetres; this quantity is carefully measured off, and is conveyed by the pipette into the open cylinder, which is quickly shaken so as to secure the admixture of the blood with the water before coagulation has had time to occur. Distilled water is then added, drop by drop, by means of a pipette-stopper, until the tint of the

diluted blood becomes the same as that of the standard in the closed cylinder. The degree of dilution, when this point is reached, indicates the percentage proportion of hæmoglobin in the blood under examination, as compared with that of normal blood. The best way of observing the tint is to hold the apparatus up between the eye and a window, so that the light passes directly through the cylinder. The test is only applicable by good daylight.

Another method of measuring the colour, *i.e.* the richness in hæmoglobin of the blood, is von Fleischl's, which has the advantage of needing a smaller quantity of blood.

The blood, diluted to a definite degree, is put into a glass chamber, and, viewed by transmitted light, its depth of colour is compared with that of a wedge of red glass, the thicker end of which is of course more saturated than the thin. The illumination is given by lamplight reflected from a white disc.

A third method is Dr George Oliver's, and is founded on Lovibond's method of matching colours by graduated coloured glasses: these standard glasses being graduated not uniformly but according to the specific colour-curve of a solution of hæmoglobin. It may be adapted to either natural or artificial illumination, and Dr Oliver uses a camera tube for comparison of the blood and glass, without disturbance of white and coloured light of other objects.*

The amount of hæmoglobin in each red disc may of course be obtained by combining the results yielded by the hæmocytometer and by the hæmochromometer. Thus if the blood of an anæmic patient contains 60 per cent. of corpuscles, but only 30 per cent. of hæmoglobin, the average amount of hæmoglobin contained by each disc will be one half of the normal. Not unfrequently it falls as low as one third.

In such cases the deficiency of colouring matter in the blood of an anæmic patient is obvious to the naked eye when the finger or ear is pricked to allow a drop to be taken for investigation. One sees at a glance that it is pale, thin, and watery, exactly as if it had been diluted.

Changes in red corpuscles.—Alterations in the microscopical appearance of the erythrocytes are frequent in anæmia. Thus, the average diameter of a normal corpuscle being $7.5\ \mu$ ($1\ \mu = 0.001\ \text{mm.}$), the average diameter in anæmia has been found by Hayem and by Eichhorst to be reduced to $7\ \mu$, $6.5\ \mu$, or even $6\ \mu$.† Moreover, red discs are sometimes present which are far smaller than any that exist in health, their diameter being from $6\ \mu$ to $2\ \mu$; these have been termed *microcytes*. Contrasting with them there may be others which are larger than normal, having a diameter of $12\ \mu$; they have been called *megalocytes*. Quincke, Eichhorst, Bramwell, and many others have described red discs presenting curious irregular forms, being oval, elongated, curved, or drawn out into pointed processes. For these conditions the term *pœkilocytosis* has been needlessly coined. They were at first supposed to be peculiar to the special disease known as idiopathic or pernicious anæmia; but they have since been discovered in cases of secondary anæmia from phthisis, cancer of the stomach, and chronic Bright's disease. Thus pœkilocytosis is far less important than another and more uncommon condition—the presence of nucleated red corpuscles, like those

* See his 'Croonian Lectures' for 1896, 'Pulse-gauging,' 1895, and 'Blood and Blood-pressure,' 1901.

† The thousandth of a millimetre (μ) is often called a micromillimetre, but according to the usage of physical science it is a *micrometre*, and the *millionth* of a millimetre a micromillimetre.

of the embryo. Another change, that seems to have been first recognised by Drs Geo. Mackern and H. Davy, then students at Guy's Hospital, is that the hæmoglobin (or zooid) is sometimes separated from the substance of the corpuscles (or œcoid of Brücke), and forms a rounded body, which had been mistaken by previous observers for a nucleus.

Changes in leucocytes.—In the secondary forms of anæmia the white corpuscles of the blood diminish along with the red, but in graver forms they maintain or somewhat increase their numbers, so that a relative *leucocytosis* results, and in one remarkable form of anæmia with enlarged spleen, their number is so greatly increased that the term *leuchæmia* or white blood is applicable. The number of leucocytes rises during digestion and sinks during starvation. A moderate excess of white corpuscles with little or no diminution of the red is a secondary leucocytosis often met with in cases of smallpox, scarlatina, and rheumatic fever, and, above all, in pneumonia, cancer, syphilis, and septicæmia, and its absence appears to be of unfavourable prognostic import. It is often preceded by a marked diminution of leucocytes (*leucopenia*). After considerable hæmorrhage a temporary leucocytosis has been frequently observed, which is apparently the first stage in re-formation of the lost red corpuscles.

The white corpuscles of the blood are not all of the same kind, and the staining methods introduced by Ehrlich enable us to distinguish varieties of leucocytosis which are an aid in diagnosis.

By far the most numerous in health are finely granular, with a nucleus and abundant protoplasm; they stain with eosin,* exhibit lively amœboid movements, and are active phagocytes.

A large coarsely granular leucocyte which stains with acid solutions, and its granules with osmic acid, is present in very small numbers in the blood, but is abundant in the marrow of bone.

Basiphile leucocytes are rarely present in normal blood, but have been found by Grünbaum in uræmia, by Copeman in leuchæmia, and by Sherrington in cholera. Ehrlich's *mastzellen* are basiphile leucocytes with coarse granules which stain with methylene blue.

Small hyaline corpuscles with large curved nuclei, the *lymphocytes*, are present in very small numbers in health—less than a quarter of the total colourless corpuscles. They are characteristic of the lymph and lymph-glands, and become numerous in the blood in cases of anæmia and leuchæmia lymphatica (pp. 766, 769).

Finally a large hyaline corpuscle with more protoplasm than the last is abundant in red marrow, whence it is called a *myelocyte*, but is even more scarce than the lymphocyte in normal human blood. In Hodgkin's disease it is not more abundant, but is much increased in number in cases of leuchæmia.

Reaction of blood.—The popular phrase—acidity of the blood—is without basis of fact. The blood in health or disease, like the lymph, like pus, and like all secretions except the gastric juice and the urine, is alkaline. But the degree of alkalinity varies. It may be measured by the reaction to litmus paper, but the great variety of methods employed by Schäfer, Haycraft, Drouin, and A. E. Wright (of Netley), show the delicacy of the observations needful. It has been ascertained that the alkalinity of the blood depends on that of the serum; and that the alkalinity is lessened in

* This form was called neutro- (or amphi-) phile, but Hardy and Kanthack ascertained that it is oxyphile, *i. e.* staining with acid solutions.

cases of diabetes, in jaundice, and in most of the specific fevers. On the other hand, in chlorosis, and most other severe cases of anæmia, the alkalinity of the serum is increased.

Specific gravity.—After various unsatisfactory attempts by physiologists, the method devised by the late Dr Roy has, with certain improvements, solved the problem. It consists in preparing a large number of solutions of constant specific gravity and observing whether a drop of blood sinks or rises in them. Roy used mixtures of glycerine and water. Others use antiseptic solutions of salts or mixtures of chloroform or benzol. It appears from many observations by Drs Copeman, Sherrington, and Lloyd Jones ('Journ. of Phys.,' 1893 and 1897) that the specific gravity follows the amount of hæmoglobin, being lowest in the most severe cases of anæmia, and that a sudden rise of the specific gravity after injuries or operations on the abdomen may indicate a dangerous condition of shock.

Rapidity of coagulation.—This may be roughly estimated by noting the time between a drop of blood received on a clean glass slip being so far coagulated as to allow a needle drawn through it to bring out a thread of fibrin. Dr A. E. Wright has contrived a series of tubes which, when blood is drawn into them, cease to allow the contents to be blown out when coagulation is complete ('Proc. Royal Soc.,' 1893). Drs Brodie and R. Russell have devised another method by which the formation of the fibrin and immobility of the corpuscles can be observed under the microscope. The longest periods before coagulation are observed in cases of hæmophilia ('Journ. Phys.,' 1897).

Anæmic murmurs.—When the stethoscope is applied to an anæmic patient's neck just above the clavicle, there is often heard a loud and harsh continuous murmur, which becomes louder with inspiratory aspiration. This venous humming sound was termed by Bouillaud *bruit de diable*, the "diable" being a toy common in Paris in 1835, which made a similar noise. The seat of this "venous hum" (*Nonnengeräusch*, i. e. humming-top sound, in German) is in the internal jugular vein; it is usually louder on the right side than the left; and though rarely absent in chlorosis, is sometimes not audible in other, even extreme cases of anæmia, particularly in men. It is stopped at once by compressing the vein above the stethoscope. According to the theory which refers all murmurs to the formation of a "fluid vein" (cf. *supra*, p. 248), the *bruit de diable* may be accounted for, if we suppose that in anæmia the volume of the blood is diminished. The jugular veins pass through dense cervical fascia. Consequently, when in an anæmic patient the veins shrink and adjust themselves to the smaller quantity of fluid circulating through them, this part remains unaltered in size, and forms a relatively wide space, into which the attenuated stream of blood opens. The explanation is corroborated by the fact that in many healthy persons one can make a *bruit de diable* by pressure with a stethoscope in the neck, as one can make a pulsating bruit by pressure on an artery.

An "anæmic murmur" of another kind is systolic in rhythm, and is heard over the area of the pulmonary artery and aorta, usually in the former only. This murmur may be accounted for by a "fluid vein," if we suppose the trunks of the two main arteries to be dilated compared with the orifices through which the blood enters them from the heart.

Whether an anæmic murmur is ever localised at the apex is uncertain. That an apical systolic bruit may be "functional" there is no doubt, but such a functional murmur may be heard without anæmia being present.

It probably in most cases depends on temporary mitral incompetence from want of vigorous ventricular contraction (*cf.* p. 78), and in others on regurgitation through the tricuspid as a physiological safety-valve (*cf.* p. 265).

These murmurs would certainly be regarded as signs of organic disease of the heart or of the great vessels if their origin were not understood. This is especially the case with the basic systolic murmur, which often has a loud harsh quality suggestive of anything but a functional origin. Indeed, when, for instance, the patient has rheumatic fever, it is often difficult to determine whether a murmur is due to anæmia or to a valvular lesion.

An anæmic murmur is never diastolic (or præ systolic); it is basal rather than apical, and pulmonary rather than aortic in position; if apical, it is not conducted to the axilla; and it is usually accompanied by arterial and venous murmurs, and unaccompanied by symptoms of organic cardiac disease.

Dyspnœa.—Since the red discs of the blood have the function of carrying oxygen to the tissues, imperfect respiration is among the most marked effects of anæmia. In fact, the result is the same whether the oxygen is prevented getting to the blood as in diseases of the larynx or the lungs, or blood prevented reaching the air as in cardiac disease, or whether, blood and air freely meeting in the pulmonary capillaries, there is yet a want of hæmoglobin to convey sufficient oxygen to the tissues.

Hence dyspnœa is almost always present in cases of anæmia; even when the patient is at rest the breathing is accelerated without his being conscious of it; and when he makes any effort he may be seized with distressing paroxysms of suffocation and palpitation. In some cases similar attacks come on without apparent cause. Probably the nerve-cells of the respiratory centre, which are stimulated to excessive discharge by imperfectly oxygenated blood, are affected in the same manner by blood in which the total hæmoglobin is greatly diminished, although what there is of it may be saturated with oxygen. In either case the oxygen that reaches the nerve-cells is deficient. There is no excess of carbonic acid, for that is conveyed from the tissues to the lungs in solution in the liquor sanguinis. Hence we do not see cyanosis or congestion in the dyspnœa of anæmia.

Fatty degeneration.—The reduction in the amount of oxygen supplied to the tissues seems to be the cause of one of the most striking appearances found after death from extreme anæmia—fatty degeneration of the muscular substance of the heart, the intima of the larger vessels, and the secreting cells of the gastric glands, the liver, and the kidneys. At one time we supposed at Guy's Hospital that such changes were peculiar to the form of idiopathic anæmia which had been described by Addison, otherwise called pernicious anæmia. But in 1873 the characteristic appearance of the heart was observed in a woman who died of cancer of the breast; in 1874 in one after hæmatemesis from an ulcer of the stomach; in 1877 in a man who had suffered severely from hæmaturia, and also in a woman who had a bleeding malignant tumour in the neck. Moreover, Perls has experimentally produced the same fatty degeneration in animals by repeated venesection ('Virch. Arch.,' vol. lix).

The degeneration is not universally distributed, but specially affects the muscular fibres of the columns of the mitral valve and those which lie beneath the endocardium lining the septum and the ventricles generally. It gives rise to the formation of a series of parallel, opaque, yellowish or cream-coloured lines, which run across the direction of the fibres, the so-

called "tabby-cat striation" (p. 51). With the microscope the opacity and pallor are seen to be due to the presence of closely aggregated fat-granules and globules, which look black by transmitted light. The muscles of the body generally show no similar change, perhaps because for a long time before death the patient has been in bed; but in some cases the diaphragm and the intercostal muscles share in the same affection as the heart.

Muscular weakness is one of the most marked effects of anæmia. The patient may be capable of a sudden effort, but he quickly becomes fatigued, and his strength is soon exhausted. He becomes less capable of mental exertion; and his nervous centres are often in a condition known as "irritable weakness." Thus there is sometimes exaggerated sensibility to a bright light or a loud noise; and, whereas the sexual appetite is, as a rule, diminished or suspended, it occasionally happens that a morbid erethism is developed. Severe anæmia in women is almost always attended with amenorrhœa, and generally with temporary or permanent sterility; but occasionally menstruation continues, and may even be profuse.

The *pupils* are uniformly dilated, and contract somewhat sluggishly to light, and this *mydriasis* helps the pearly conjunctiva to give a characteristic aspect to the eyes.

The *pulse* is usually small, soft, and feeble, in proportion to the severity of the case; or it may be imperceptible at the wrist. But when the cause is a sudden loss of blood, the pulse is often sharp and jerking; and in chlorosis the tension is sometimes above, not below, normal.

The *temperature* of the surface of the body is often low in ordinary anæmia, but in the gravest forms of anæmia we shall see that irregular pyrexia is an important symptom.

Pathology.—The most constant character of the blood in anæmia is deficiency in hæmoglobin, and it is obvious that there are two ways in which the amount of hæmoglobin may be reduced below the normal standard:—It may escape from the vessels by hæmorrhage, or be consumed within the body more rapidly than it can be reproduced; or, on the other hand, its formation may be defective.

When anæmia results from hæmorrhage, and the patient quickly regains his colour as soon as the bleeding is arrested, all the formative processes, both chemical and histological, are, we may suppose, in a healthy state. When anæmia arises spontaneously, it may be due to a defect in production of hæmoglobin or of corpuscles; or it may be due to destruction of red discs and of the hæmoglobin which they contain.

In some instances Quinke found the amount of iron in the liver from ten to thirty times greater than normal, and that there was also an increase in the kidneys, spleen, and the pancreas (*cf. infra*, p. 781).*

The striking discovery by Virchow of the condition which he named leuchæmia led to too broad a separation between what was before known as anæmia splenica and other forms of extreme anæmia: it seemed to be forgotten that excess of white corpuscles is always accompanied by a deficiency of red; that leuchæmia is, in fact, only a species of anæmia. Addison's discovery, of equal importance with Virchow's, that anæmia of

* See Quinke's article in Volkmann's 'Sammlung klin. Vorträge,' 1876, and Dr Wm. Hunter's valuable papers in the 'Lancet,' September 22nd to October 6th, 1888; also Dr Mott's (*ibid.*, March 16th, 1889; 'Path. Trans.,' 1890; and 'Practitioner,' August, 1890).

the severest kind may occur independently of chlorosis and of any other known cause, may go on uninfluenced by treatment, and may end in death; and that this restricted group of fatal and idiopathic anæmia is marked by definite symptoms and anatomy—was not understood in Germany, where under the name of progressive pernicious anæmia a heterogeneous group of cases were united together, many of them secondary, some uterine, some malarial, and only agreeing in the fact of their fatality.

The pathological classification of cases of anæmia is not easy, since many of them are of uncertain origin and nature.

The following is practically the arrangement proposed by the writer in an article in the 'Guy's Hospital Reports' for 1882 (vol. xli, p. 254).

i. *Symptomatic anæmia*.—(1) The most obvious and direct cause of anæmia is external hæmorrhage.

In women the most frequent source of hæmorrhage is menorrhagia, and in men bleeding piles; recurrent epistaxis is a frequent cause in youth, and it also occurs with the degenerated arteries of old age.

The pallor caused by large losses of blood in a healthy subject is accompanied by muscular weakness, a rapid and irritable pulse, and many of the other signs above noted. In most cases, so soon as the bleeding is stopped, the lost corpuscles are rapidly restored with the help of a healthy appetite, assisted in some cases by wine, and by chalybeate drugs. Sometimes, however, even after traumatic hæmorrhage in a healthy subject, recovery is neither rapid nor complete. In a case which came under the writer's observation a British officer in one of the Chinese ports was waylaid and nearly murdered by assassins. He received several stabs, and lost a large quantity of blood. He ultimately recovered and returned to England, but when seen some years later he was still extremely anæmic. Similar cases of protracted or permanent anæmia not unfrequently follow a single delivery accompanied by severe flooding; and such prolonged pallor is often seen as the result of continued, though small, losses of blood by bleeding piles. An important cause of anæmia from long-continued hæmorrhage is the presence of *Sclerostomum* (*Ankylostomum*) *duodenale* (*vide supra*, p. 466).

(2) A second natural group of cases of anæmia consists of those which follow losses, not of blood, but of leucocytes and albumen, which may be considered a kind of modified hæmorrhage. Such is the anæmia of prolonged suppuration with or without lardaceous disease, the anæmia which follows chronic and profuse leucorrhœa, and long-continued diarrhœa or dysentery unaccompanied by hæmorrhage.

Closely allied to the last cases is the anæmia which accompanies too frequently recurring pregnancies, too long continued lactation, and perhaps that of Bright's disease with prolonged albuminuria. The anæmia of sexual excesses in male subjects may perhaps come under the same head.

(3) Another group is that of the cases of anæmia which depend not on hæmorrhage or excessive discharges which impoverish the blood, but on deficient nutrition; in both cases the blood as a tissue suffers, in one from the drainage of profuse expenditure, in the other from the starvation of diminished supplies. Under this head comes the pallor of inanition from whatever cause, particularly, from a clinical point of view, the starvation produced by stricture of the œsophagus, by the anorexia of gastritis and severe indigestion, and by gastric ulcer in cases which are unaccompanied by hæmorrhage.

We may perhaps include in this kind of anæmia from deficient income, that which follows enteric fever and other acute diseases, where there is neither hæmorrhage nor profuse discharge. The anæmia is accompanied by loss of flesh and is soon repaired by the healthy appetite of convalescence. The striking and rapid anæmia of acute rheumatism and of diphtheria may be included in the same group; but there is probably a destructive process going on at the same time in all cases of pyrexia.

Somewhat similar cases of anæmia are those which follow Bright's disease and valvular lesions of the heart and tuberculosis in general.

In *phthisis* the causes of anæmia are numerous: there is the wasting discharge of pus from the lungs; there is frequently hæmorrhage, and there is always impaired appetite and digestion; but here also the growth and spread of tubercle appears directly to impair the nutrition, since we see pallor and emaciation in children in whom tuberculosis of lymph-glands or serous membranes is unaccompanied by suppuration or by fever.

The pallor of *phthisis* is well known, but is scarcely more striking than the pallor of *malignant disease*, which also can be often ascribed to hæmorrhage or to loss of appetite; but it may precede any such symptoms, and is no doubt partly due to the destruction of red corpuscles with a relative increase of the white, or, to use Virchow's term, anæmia with leucocytosis.

Along with such cases of tuberculous and malignant anæmia may be placed the cachectic anæmia of *syphilis* and of *malaria*.

(4) Whether or no we admit a destruction of hæmoglobin in cases of rapidly spreading tubercle, sarcoma, or cancer, we see it unmistakably produced in the cases of *toxic* anæmia which result from the direct action on the blood-corpuscles of mercury, of phosphorus, or of lead.

In all the above cases anæmia is secondary, symptomatic, and more or less plainly referable to a sufficient cause.

ii. *Primary anæmia*.—We now come to a second broad division of cases of anæmia which own no such causes as those mentioned above.

(1) The most common and clinically well-marked kind of anæmia is that which has been known from early times as *chlorosis* or green-sickness, a form characterised by the sex and age of the patients, by the absence of emaciation, and by the striking results of appropriate treatment.

(2) To another group of primary anæmia belongs *anæmia lymphatica*, or Hodgkin's disease, *anæmia splenica*, and also the primary anæmia which is combined with a considerable excess of leucocytes and a considerable hypertrophy of the spleen—included under the "anæmia splenica" by the older writers, and often confounded with malarial cachexia—the *leuchæmia* of Virchow.

(3) Lastly remain the decidedly rare cases, first and sufficiently defined by Addison—primary but unassociated with anatomical changes in the spleen and other organs, widely distinct in their clinical aspect and prognosis from cases of chlorosis, idiopathic in the strictest sense of the word; and more closely related to leuchæmia and Hodgkin's disease than to any other form of anæmia, by their severity, their proclivity to hæmorrhage and occasional pyrexia (symptoms not present in secondary anæmia nor in chlorosis), their tendency to a fatal issue, and, lastly, in their reaction to arsenic, and their resistance to the chalybeate treatment which is so efficacious in chlorosis and most forms of secondary anæmia.

In the group of secondary forms of anæmia, there is loss of flesh; the blood is more deficient in the colour than in the number of the red discs;

and the anæmia is cured by recognising and treating its cause, and usually relieved by the exhibition of iron.

In anæmia from hæmorrhage, from starvation, from rheumatism, or acute nephritis, or phthisis, the skin is blanched, and in a xanthochroic population shows a pure dead white.

In the anæmia secondary to chronic interstitial nephritis, to cancer, syphilis, and ague, the colour is "earthy," dingy, and often the pallor is accompanied by diffuse pigmentation. In these cases also there is more destruction of hæmoglobin than of corpuscles, and the pallor is often removed by steel. A syphilitic patient recovers his natural colour under mercury, and one suffering from paludal cachexia under arsenic.

The anæmia of plumbism is often persistent for a long time after all other signs of saturnine poisoning have disappeared.

The symptomatic groups of anæmia have already been incidentally mentioned in the chapters on fever, malaria, syphilis, phthisis, cardiac disease, and disorders of the stomach and kidneys. We therefore now proceed to the primary forms as above defined, and begin with the remarkable condition described and named by Virchow.

LEUCHÆMIA.*—In 1845 Dr Hughes Bennett, of Edinburgh, recorded a case of enlargement of the spleen in which, after death, the blood was found to be full of objects which he regarded as pus-cells and attributed to "suppuration of the blood." A month later Virchow published a similar case in Berlin; but he recognised the cells in question to be identical with the colourless corpuscles of the blood, and proposed to call the affection "leukæmia," *i. e.* a condition of white blood. In 1846 Dr Fuller, at St George's Hospital, and Dr Walshe, at University College, demonstrated the same change in the blood of living patients, and the disease has ever since been universally recognised.

Subsequent research discovered, as one would expect, many previously recorded but misunderstood cases. Thus Dr Craigie, after seeing Bennett's case in 1848, remembered a similar one years previously, in which the pathologist John Reid had recognised in the blood "globules of purulent matter and of lymph," and published it in the 'Edin. Med. Journ.,' October, 1845. Donné also, in 1839, had found what he called "globules muqueuses" in the blood of a patient. Velpeau, as far back as 1827, had recorded a case in which the spleen was enlarged, and "the blood looked as if it were mixed with pus." Other cases are quoted from Bichat and Andral, with descriptions of the blood as "*saie grisâtre*," and "*sang comme saieux*." Piorry had met with a well-marked case in 1843, and had made the same mistake as Bennett, for he named the condition "*Hémite*," a word which he supposed to mean "inflammation of the blood."

In 1852 Dr Bennett admitted that Virchow's view of the condition was

* *Syn.*—Leucocythæmia.—*Fr.* Leucocythémie.—*Germ.* Leukämie—Myelocythæmia.

It matters little whether, with Virchow, we call the blood "white" or "pale," or with Bennett call it "white-celled;" neither term is literally exact. But the former is shorter, is more generally accepted by pathologists, and has the right of priority. For Virchow gave it ("Weisses Blut," 'Froriep's Notizen,' November, 1845; "Leukämie," 'Arch. f. path. Anat.,' Bd. i, S. 563, 1847) some years before Bennett proposed to amend his first name, "suppuration of the blood," by the new one of "Leucocythæmia" ('Leucocythæmia, or White-celled Blood, in relation to the Physiology and Pathology of the Lymphatic Glandular System,' Edin., 1852).

correct, but would not accept his name, and proposed the name "leucocythæmia" for the new disease.*

Definition.—A slight excess of leucocytes is not enough to justify the title of leuchæmia. Virchow long ago pointed out that they may be somewhat increased in numbers under various conditions attended with irritation of lymphatic glands, as well as during pregnancy, and in fevers (p. 769); and distinguished such minor degrees as "leucocytosis." The same condition has been since recognised in cancer and in acute pneumonia.

Huss proposed to define leucocytosis as a condition in which the proportion of white cells to red discs reaches one in twenty. Others define leucocytosis as a permanent excess over 8000 leucocytes in the cubic millimetre, and leuchæmia as a permanent excess over 10,000, *i. e.* to the standard 5,000,000 red corpuscles, as 1 to 500.

Another distinction is that in leuchæmia the white cells are mostly myeloid and have lost their power of movement on a warm stage.

As a rule, leuchæmia is accompanied by considerable hypertrophy of the spleen, so that "splenic" leuchæmia is almost a needless qualification. But there are exceptions to this rule.

Such exceptions were mentioned by Virchow as far back as 1847, and were recognised by Bennett, in his work published in 1852. In the first volume of his 'Archiv,' Virchow related an instance in which the principal morbid change was in the lymph-glands, which were enormously enlarged throughout the body. Subsequently he described two forms of leuchæmia—the one "splenic," the other "lymphatic"—distinguished by difference in the size of the leucocytes, these being comparatively large and having sometimes more than one nucleus in the splenic form, but in the lymphatic form being small, and having their scanty protoplasm in close contact with a solitary nucleus (*cf.* p. 769).

Virchow soon became aware that leuchæmia is usually absent when there is a general enlargement of the lymph-glands, a condition previously described by Hodgkin; and in the 'Krankhaften Geschwülste' he described under the heading of "lympho-sarcoma" cases of this kind, for which other German writers have used the singularly inept name of "Pseudo-leukämie." A marked example of splenic leuchæmia with lymphatic anæmia was recorded by Dr Frederick Taylor ('Path. Trans.,' vol. xxv, p. 246).

Another kind of non-splenic leuchæmia was first described by Neumann, in 1870, which Mosler and Cohnheim subsequently called Leuchæmia myelogenica or medullaris, a condition in which the red marrow of the cancellous tissue of the sternum or other bones is hypertrophied, and the yellow marrow loses its adipose character and becomes red marrow. Béhier published a case of leuchæmia with hypertrophy of the lymph-follicles (solitary and agminated glands) of the intestine. The combination of leuchæmia with hypertrophy of the lymphatic tissue of the liver or the thymus is more common.

Apart from such exceptional instances, most cases of leuchæmia are distinguished by the following characters:—(1) Enlargement of the spleen is present; (2) the increase of white corpuscles is almost entirely due to increase of the spleno-medullary large nucleated cells, which are not present in the blood in health, nor in leucocytosis; the excess of leucocytes in the

* On the history of the discovery see further Virchow ('Gesammelte Abhandlungen,' pp. 149—218) and Bennett ('Princ. and Pract. of Medicine,' pp. 857—890).

blood is great; (3) in probably all cases there is hypertrophy of red marrow also.

The spleen.—In cases of Leuchæmia the first thing usually noticed by the patient is that his abdomen is becoming larger, that there is a fulness or a lump in the left side, or that he has a dull pain there. On examination one generally finds that the spleen is already very large; larger than is found in any but an advanced stage of other forms of splenic hypertrophy. Sometimes it reaches the umbilicus, and in many cases it descends to the level of the iliac crest. As time goes on it occupies a position which could hardly have been anticipated (see the series of diagrams in Dr Fagge's article in the 'Guy's Hospital Reports' for 1869). Tethered by the vessels at its hilus, it follows a curved course, its lower end sweeping across the brim of the pelvis, and turning upwards when it has reached the right iliac fossa. Probably the fold of the peritoneum between the splenic flexure of the colon and the parietes, called *sustentaculum* or *trabecula lienis*, may, when well developed, help to direct the spleen forwards (*cf. supra*, p. 739).

Ultimately the spleen may so completely fill the abdomen below the navel that its upper end can be felt below the ribs: indeed, in women it has often been mistaken for an ovarian tumour. Its real nature may, however, be distinguished by the sharp edge which crosses the abdomen obliquely from the left lower ribs downwards, and which presents one or more notches. Its surface is almost always smooth and firm. A friction-fremitus can sometimes be felt over it, and with the stethoscope not only a rub, but occasionally a blowing systolic murmur is to be heard, like the placental *souffle*. At an advanced stage of the disease it may be separated from the parietes by a layer of ascitic fluid, through which the fingers dip before they reach it.

The spleen is often found fixed to the adjacent parts by adhesions, and its capsule may present large white opaque patches. The leuchæmic spleen is by far the largest produced by disease, and may weigh from 60 to 120 ounces up to 5, 10, or even 15 pounds. Its cut surface is generally smooth, shining, and homogeneous-looking; but sometimes it is marked with whitish lines and striæ, due to thickenings of the trabeculæ. Its consistence is firm, and its colour brownish-red. The Malpighian corpuscles (splenic follicles) are not enlarged, and are often shrunken. It not infrequently shows wedge-shaped pale nodules, resembling the blocks produced by embolism, and probably due to local thrombosis. Histologically the organ is overgrown, and its tissue is found full of leucocytes—fixed and wandering—most of them large, clear, and nucleated, some large and coarsely granular, and some lymph-leucocytes.

The blood.—In spleno-medullary leuchæmia this is paler than natural, and, when the excess of leucocytes is very great, has sometimes a greyish-red colour, like a mixture of pus and blood. Moreover, after death the appearance of coagula in the heart, or in the great vessels, is often peculiar; they are grey and opaque, instead of being yellow and translucent.

The red corpuscles are always greatly diminished in number. The amount of hæmoglobin is also diminished, but more or less nearly in proportion to the diminished number of erythrocytes, not much more as in chlorosis, nor much less in proportion as in Addison's idiopathic ("pernicious") anæmia. Nucleated red discs are observed in probably every well-marked case.

The proportion of leucocytes to red discs varies widely in different cases,

and according to the stage of the disease. From the normal ratio, which is not higher than 1 to 250 or 300, it increases till it reaches 1 to 50, 1 to 20, 1 in 10, or 1 to 5. In extreme cases the leucocytes may even be more numerous than the red, as in a case of Sørensen's, in which they were counted, and found to be as 68 to 47 of red discs.

Put in another way, in (spleno-medullary) leuchæmia the red corpuscles fall from 450,000,000 or more in the cubic millimetre to 2,000,000 or less; while the white corpuscles increase from 6000 or 8000 in the cubic millimetre to 200,000 or 250,000 or occasionally 500,000, or even more.*

The white corpuscles are not only increased in number but are also altered in character. Examined in a film, with suitable staining fluid,† the lymphocytes—small globular nucleated clear cells—are not increased or are even diminished. The normal (or normally most abundant) leucocyte (small polymorphic cell) with amœboid movement, horseshoe or tripartite nucleus, with fine granules and readily staining—oxyphil or neutrophil—is also not materially increased in number, nor the coarsely granular eosinophil cells described above. The leuchæmia depends almost entirely on the remarkable increase of the myelocytes, large globular, nucleated, granular cells, which do not occur in the healthy blood, but are characteristic of the red marrow.

These myelocytes have no amœboid movements on the warm stage—a fact many years ago observed independently by Mr Golding-Bird and by the late Dr Cavafy.

It is remarkable that this anæmia is sometimes unattended with obvious pallor of the countenance; as Wilks long ago pointed out, patients, even at an advanced stage of the disease, have often colour in their cheeks and lips, so that, seeing them in bed, one would hardly imagine them to be very ill. As a rule, however, the skin is yellowish white, like wax.

The blood-plates are said by Dr Robert Muir to be increased in number in the spleno-medullary and diminished in the lymphatic cases.

Charcot's crystals are frequently found in the blood and also in the marrow of the bones. They are of variable size, colourless, elongated, and octahedral, consisting of two very slender four-sided pyramids set base to base. Their chemical nature is uncertain; they dissolve in warm water and in alkalis, but are insoluble in alcohol and ether. They have often been found in the sputum, particularly in cases of asthma (p. 101).

The lympharia.—In perhaps one of every three or four cases there is more or less enlargement of lymph-glands, especially those of the abdomen and chest. The increase in their size is considerable; often twice as large as normal. They may be firm and fleshy, or soft and medullary. They are not found fused together, nor do they penetrate into structures adjacent—characters which distinguish them from lympho-sarcomata.

The *follicles* at the base of the tongue and the *tonsils* may be greatly swollen. There may also be diffused pharyngitis and stomatitis. The gums may be swollen or ulcerated. The *intestinal follicles*, solitary and agminated, are sometimes greatly enlarged, and a lymphoid growth is said to infiltrate the submucous tissue.

* See Dr A. E. Taylor's elaborate and valuable "Studies in Leuchæmia," published in the 'Contributions from the Wm. Pepper Laboratory,' Philadelphia, 1900, p. 148.

† Dr Louis Jenner's stain, a solution of methylene blue and eosin in absolute alcohol ('Lancet,' 1899, vol. i, p. 370), has been lately used in our wards and laboratories, and has been found particularly easy and satisfactory.

A case of Dr. Frederick Taylor's is recorded in the 'Pathological Transactions' for 1873, where the spleen weighed fifty-one ounces, and had the appearance seen in ordinary cases of leuchæmia, but there were also mediastinal and subpleural lymphomata of remarkable size. This would now be regarded as a mixed case.

The most constant and remarkable condition found after death, in addition to the enlargement of the spleen, is that in the *medulla* of the bones, first described by Neumann. The cancellous tissue acquires a yellow tint, like that seen in osteo-myelitis; and on pressure a puriform juice exudes. Moreover, the adipose tissue or yellow marrow of the shaft of long bones becomes changed into lymphatic (cytogenic or adenoid) tissue like the red marrow of the cancelli, but of the same pale unhealthy colour which is seen in the cancellous tissue itself; and in some cases the compact bone itself is transformed into a cancellous reticular tissue filled with red marrow. Under the microscope, the normal fat-cells of the yellow marrow have disappeared, leaving only free oil-drops and fatty granules. Sometimes the marrow is redder and firmer—less like pus and more like the normal contents of cancellous tissue. Giant-cells (myeloid) are found as in normal red marrow, and a few nucleated erythrocytes, while the majority of the leucocytes which fill the field have the characters of myelocytes.

Mosler met with a case in which this leuchæmic affection of the sternum was indicated during life by great tenderness on pressure; and this important symptom has frequently been since observed (see, for instance, Dr Beatty's case in the 'Brit. Med. Journ.' for April 18th, 1891).

The thymus, as above stated, is occasionally hypertrophied along with the spleen and lymph-glands, both in children and in young adults with a persistent thymus.

Of the remaining organs, the *liver* is most often diseased in splenomedullary leuchæmia. It may weigh as much as eight or ten pounds. There is not always any obvious change in the appearance of its cut surface, but in many instances there are masses of lymphoid growth scattered through the liver, especially in the neighbourhood of the vessels. Sometimes these are visible only with the aid of a lens, sometimes they are apparent as minute, greenish-white granules. With the microscope numerous leucocytes are seen between the hepatic cells within the acini, while others crowd the portal canals.

The *kidneys* are less often affected. They sometimes show scattered, greyish-white striæ, running through the cortex, and bearing a close resemblance to those which are seen in cases of ascending nephritis (p. 698). The surface is smooth and marked by ecchymoses. On microscopical examination, beside more or less abundant extravasation of red discs, numerous leucocytes are seen between the tubules. The urine is not infrequently albuminous during life, even when there are no other changes in the kidneys, indicative of any form of Bright's disease.

The *heart*, as in other forms of severe (grave or pernicious) anæmia, is often found in a state of fatty degeneration.

The *lungs* occasionally show wedge-shaped infarctus. This was the case in a patient whose spleen was excised by Mr Bryant in 1866, and who only survived the operation three hours. The patches in the back of the lungs had gangrenous centres and red borders. Such patches are produced by plugging of the pulmonary capillaries by leucocytes. The lungs in leuchæmia appear not to exhibit definite nodules of a new growth, such as are seen in Hodgkin's disease; and in most cases they are quite healthy.

Symptoms.—Next to enlargement of the spleen, the most striking sym-

ptoms are those of severe anæmia—pallor, shortness of breath, and frequent hæmorrhage, with occasional attacks of pyrexia. The lack of hæmoglobin, by diminishing the amount of oxygen which can be taken up, causes the dyspnœa, which is sometimes the chief complaint of the patient. This may be present only when muscular efforts are being made; but in extreme cases even the slightest movement is attended with great distress.* Another cause of dyspnœa is obstruction to descent of the diaphragm by the enlarged spleen.

Hæmorrhage.—Probably the over-abundant leucocytes adhere to the lining membrane of small vessels, and accumulate so as to obstruct them like minute emboli. But there is also a change in the walls of the capillaries, which renders them liable to rupture; for we shall see that liability to hæmorrhage is also a symptom of the other grave and primary kinds of anæmia.

Epistaxis is very common; it may recur every day, and is sometimes the direct cause of death. Bleeding may also take place from the gums, the stomach or bowels, the kidneys, the lungs, or the uterus. Moreover, the hæmorrhage, after a wound or other injury, is apt to be excessive; even the extraction of a tooth has, in at least one case, led to a fatal result. The amount of fibrin yielded by the blood in this disease is said to be above the normal, but, instead of coagulating in long elastic filaments, when separated by stirring, it falls in granular fragments. Purpuric spots are frequently seen upon the skin; and after death the surface of the heart may be found ecchymosed. A large quantity of blood is sometimes extravasated among the muscles or behind the peritoneum.

In the retinæ hæmorrhages are frequently observed, both during life and after death. According to Gowers they are usually small, and most abundant towards the periphery; they often form striæ, following the lines of the nerve-fibres, and after a time the blood undergoes conversion into a brownish pigment. The hæmorrhagic patches often have white or yellowish-white centres, and similar spots may be observed without any extravasations. Liebreich and other observers since have seen the retina affected with diffuse swelling, and its veins distended and tortuous—"leuchæmic retinitis." Lastly, hæmorrhage into the brain occasionally occurs, with the ordinary symptoms of apoplexy.

Pyrexia, etc.—The temperature is often raised from time to time, followed after a few days by apyrexial intervals. Sometimes this pyrexia is accompanied with shivering and sweating, and may reach 103° , or higher still; but usually it is only moderate. Patients complain of muscular weakness, headache, giddiness, noises in the ears, and palpitation. They often suffer from tonsillitis, gastric disturbance, vomiting, and diarrhœa. Functional murmurs are usually loud. The urine is very acid, and has a constant excess of urates.

Course and event.—Leuchæmia, as a rule, advances slowly towards a fatal termination. Its duration is from one to three years; in the case of a child it is much shorter. The extent to which the spleen is enlarged is of much less significance than the degree of pallor and of breathlessness; and as regards the state of the blood, it matters less whether the number of leucocytes is greatly increased than whether that of red discs is greatly diminished.

* According to an observation made by Pettenkofer and Voit, during rest the quantity of oxygen absorbed and that of carbonic acid given off were the same as in health ('Zeitschft. f. Biol.' v, 319, 1869). The figures were: O, 790 to 832; CO₂, 265 to 249.

Death often takes place unexpectedly by some complication, such as epistaxis, pleurisy with effusion, diarrhœa, syncope, or lobar pneumonia.

Occasionally hæmorrhage takes place into or on the surface of the brain, and death follows with symptoms of hemiplegia and coma. Dr Mott has published a case in which hæmorrhage into the internal ear produced deafness of that side with loss of balance.

If the disease goes on to its natural end without such complications arising, swelling of the legs, ascites, and general dropsy come on, and death ensues from œdema of the lungs.

There are sometimes well-marked remissions, but they are less common than in cases of idiopathic anæmia.

A comparatively small number of cases of splenic leuchæmia run an acute course—measured not by months but by weeks. Such cases are more common in children than in adults and are frequently mixed cases, with hypertrophy of the lymph-glands as well as the spleen. In fact, the size of the spleen is very much dependent on the duration of the disease; it is only moderately increased in these acuter cases, while the largest spleens are found in the more chronic ones. Some of these cases, first described by Fränkel in 1895, are associated with large spleen and myelo-leuchæmia; others with enlarged lymph-glands and lymphæmia (p. 766), as in three of five cases by Drs Bradford and H. B. Shaw ('Med.-Chir. Transactions,' 1898).

Ætiology.—No cause for leuchæmia has yet been discovered.

In a certain number of cases, the patient has previously suffered from *ague*. Gowers found it so in thirty among 150 cases which he collected, and at Guy's Hospital Dr Fagge believed the proportion was considerably higher. If there is more than a coincidence in these results, it is remarkable how long the interval has sometimes been, and how mild the attack of *ague*. In nine out of twenty-one cases, a period of from ten to thirty years passed before leuchæmia showed itself by any symptoms. A patient in Guy's Hospital in 1880, a Pole, had suffered from tertian *ague* in 1858, during four months in Warsaw, but first noticed an enlargement in the left hypochondrium in 1866.

The characteristic protozoa of intermittent fever have never been discovered in the blood of a leuchæmic patient, although recently Löwitt has described amœbiform parasites in the leucocytes.

Leuchæmia occurs about twice as often in males as in females. It affects persons of all ages, from infants to men above seventy, but it is most frequent in those who are between twenty and fifty, and very rare in women above that age. Cases are recorded in cattle.

Pathology.—The theory of splenic leuchæmia and its allies is still very imperfect. Many observers, starting from the fact that some of the white corpuscles of the blood are normally derived from the Malpighian bodies of the spleen, have thought to account for the phenomena of the disease by supposing that the organ, being hypertrophied, produces an excessive number of white corpuscles which pass into the circulation. But the degree of leuchæmia by no means corresponds to the hypertrophy of the spleen, and its Malpighian follicles are often found small and shrunken. Moreover we have no known parallel to morbid hypertrophy of an organ with consequent excess of function.

Others have supposed some change in the leucocytes themselves, which prevents their undergoing conversion into red discs, but it is very doubtful

whether any leucocytes do turn into red discs; more probably the latter have a separate origin.

The overgrowth of the spleen has been supposed to be due to a large number of white corpuscles being retained in its texture, the occurrence of splenic enlargement without leuchæmia to retention in the organ of all the leucocytes; and the fact that the blood is sometimes loaded with them, while the spleen remains of normal size, to their all escaping.

Ehrlich's hypothesis is that leuchæmia is but exaggerated leucocytosis, and like the latter is a protective reaction of the organism against a primary infection. Since, however, we do not know the nature of the supposed infection, nor whether it is particulate or chemical, it seems premature to adopt a view which only the gradual progress of knowledge of the process of hæmatopoiesis can disprove or confirm.

The clinical features of leuchæmia are those of severe progressive anæmia (as "pernicious" as the form so named), and depend not on the increase of myelocytes but on the diminution of erythrocytes and hæmoglobin. The remarkable enlargement of the spleen is more probably secondary to, than causative of, the condition of the blood. It is largest in the oldest cases, and absent or but slight in the most acute. If this is true, removal of the spleen will be useless, and attempts to diminish it by cold or electrical stimuli or large doses of quinine will be equally futile.

Until the nature of the infective process, if such there be, at the root of the disease, or the origin of the anæmia in other ways, has been ascertained, no rational treatment, preventive or curative, is possible; and the only plan is to endeavour to meet the varying wants of the patients.

No infective microbes have yet been discovered, and of Löwit's amoebiform parasite one can only say that, as in the case of cancer, its nature and significance are very doubtful, and some of the best pathologists regard it as an *artefactum*.

Nervous influences and toxic products of metabolism have been invoked as causes of leuchæmia, as of other diseases whose pathology is unknown.

The explanation of the disease must wait a better knowledge of the process of hæmatopoiesis and hæmatolysis than we at present possess.

Treatment.—Inhalation of oxygen has been tried with varying success. The best result the writer has seen was in the case of a powerful police constable with splenic leuchæmia who was for several months under my colleague Dr Frederick Taylor, and went out with all his symptoms relieved. He also took arsenic.

Mosler relates the case of a boy aged ten, whose spleen was considerably enlarged, and whose blood contained leucocytes in the proportion of one to twenty red discs; he took a drachm and a half of sulphate of quinine in the course of four days, then ten grains and afterwards six grains daily. He completely recovered.

Dr Goodhart, in 1876, stated to the Clinical Society that in the previous two years he had seen six cases, all in children under two years old, with moderate increase of the spleen, and with about ten times the usual proportion of leucocytes in the blood, and that they all got better under iodide of iron, or phosphorus, or cod-liver oil.

Other cases have been reported of benefit from a cold douche directed upon the left hypochondrium, and the application of a galvanic current, the positive pole being placed over the tenth rib, the negative over the enlarged spleen.

It seems to be well ascertained that steel is in these cases useless, and arsenic, though more useful, is only so for a time. When a case is already advanced, little or nothing can be done to check its progress. Quinine is

then, at least, without any appreciable result. Transfusion of blood is not more effectual than injection of salt solution, and that is only useful for a very short time. Excision of the spleen should be rejected, on account of the danger of hæmorrhage from rupture of adhesions, as well as of peritonitis; moreover, it probably would have no effect on the state of the blood.

Leuchæmia myelogenica was the name formerly given to cases like those of Neumann (p. 759), in which there are symptoms of leuchæmia with increase of leucocytes in the blood, but no enlargement of the spleen or lymph-glands. In all of these the changes above described in the bones are present; and the most characteristic form of leucocytes found in the blood are myelocytes. Hence, we may define leuchæmia as a form of severe anæmia in which abundant excess of white corpuscles are poured into the blood from the marrow. The frequent enlargement of the spleen is probably only secondary, but from a clinical point of view we may still use the term splenic or myelo-splenic leuchæmia for the ordinary leuchæmia, and myelogenica for the rarer form.

Leuchæmia lymphatica.—In a small number of cases a condition of the blood like that of ordinary spleno-medullary leuchæmia is associated with enlargement of the lymph-glands, without affection of the spleen.

But there are other cases, also rare, in which with the enlarged lymph-glands of Hodgkin's disease there is an excess of leucocytes in the blood; and it has now been ascertained by Ehrlich, Cabot, Löwitt, and many other observers that in these cases the increase of white corpuscles in the blood during life is not due to excess of medullary cells ("myelæmia"), but to an excess of the small, clear, nucleated cells found in the lymph-glands and the thymus. Instead of 7000 in the cubic millimetre they may count 200,000 or more.

To this lymphatic form of leuchæmia belong some at least of the cases of lymphoid growths in leuchæmia described in France and Germany, and by Dr T. D. Dunn in the 'American Journ. of Med. Sc.' for March, 1894.

ANÆMIA SPLENICA.*—There may be great enlargement of the spleen with anæmia, but without leuchæmia (*anæmia lienalis v. splenica*). One case occurred to Mr Spencer Wells, the spleen weighing 6 lbs., another to Mr Squire, the spleen weighing 13 lbs. Wilks described the disease in the second edition of his 'Lectures on Path. Anat.,' p. 476 (1875).

Several cases have been recorded, chiefly in France, among children and young adults, as primary splenic hypertrophy. It is also known as pseudo-leuchæmia splenica—an absurd name.

It was again described by Banti in 1882. The latest accounts are by Bruhl, by Dr Frederick Taylor ('Guy's Hosp. Reports,' lii, p. 173), and by Dr Samuel West in 'Allbutt's System' (vol. v, p. 539).

A well-marked case was published by the writer in 1870 as a spleen from a case of fatal anæmia, and another in 1875 of enlarged liver and spleen from overgrowth of adenoid (lymphatic) tissue without leuchæmia ('Path. Trans.,' xxi, p. 390, and xxvi, p. 199). Splenic anæmia is a rare form of disease even among children and in young adults.

The anæmia is usually marked before the spleen can be felt below the ribs. In the muscular weakness and absence of emaciation it resembles the idiopathic anæmia of Addison, as also in the epistaxis and retinal hæmor-

* *Synonyms*.—In France splénomégalie primitive; in Germany Pseudo-leucæmia splenica. Also splenic cachexia.

rhages which mark its progress. There is often pain from perisplenitis, but not oftener, perhaps, than in cases of splenic leuchæmia. The microscopical appearances of the blood are those of profound anæmia:—diminution of the red corpuscles down to 2,000,000 in a cubic centimetre, or as low as 1,370,000 (case of Dr F. Taylor), and corresponding diminution of hæmoglobin—with no increase of leucocytes. The progress is downwards—as in other forms of primary anæmia, with hæmorrhage and pyrexia—and the last stage is marked by dropsy and feeble pulse, with sometimes albuminuria, but without emaciation; petechiæ and larger hæmorrhages are found in the mucous and serous membranes or the solid viscera. After death the spleen is found enlarged to double its ordinary size, and cases are on record of its weighing from two to five pounds. To the naked eye it resembles the spleen of leuchæmia, but there is more fibrous tissue and less of adenoid tissue (Malpighian bodies and “splenic pulp”).

The liver is often enlarged and sometimes cirrhotic. The lymph-glands and marrow are, in the uncomplicated cases, healthy.

The heart is sometimes fatty or dilated.

If we distinguish by the special name of leuchæmia those cases of the anæmia splenica of older authors in which there is excess of leucocytes as well as defect of red corpuscles in the blood, we must, as we have seen, admit the anatomical divisions of leuchæmia splenica, leuchæmia myelogenica (always, or almost always, combined), and leuchæmia lymphatica, with their combinations. In the same way, when there is no excess of leucocytes, we have a corresponding series which may be called anæmia splenica, anæmia lymphatica, and anæmia myelogenica. The last scarcely occurs as an independent malady, but is sometimes present as a complication of anæmia lymphatica (*infra*, p. 769); the second will presently be described as Hodgkin's disease.

The pathology and origin of splenic anæmia are quite unknown. Attempts to find a living organism—schizomycetic or amœbiform—have hitherto failed. It is most frequent in children and youths.

The prognosis is hopeless, for the course is progressive and pernicious, and no treatment has yet been found of avail.

ANÆMIA LYMPHATICA.*—Another grave form of anæmia is characterised by enlargement of the lymph-glands, though the spleen is often affected also. These cases present at the bedside a common group of symptoms, those of primary and severe anæmia; associated with a more or less general and sometimes extreme enlargement of the lymph-glands, a moderate increase in the size of the spleen, with the presence of scattered nodules in it, and diminution of erythrocytes and of hæmoglobin without increase of leucocytes.

A name to denote these cases which has met with general acceptance is that of Hodgkin's disease, proposed in 1865 by Dr Wilks, to whom we are mainly indebted for the recognition of the malady in this country. He thus replaced the title “Anæmia lymphatica,” which he had given it in earlier papers in the ‘Guy's Hosp. Reports’ for 1856 and 1862. He found, in fact, that his own observations had been anticipated by a former lecturer on pathology at his own school, Dr Thomas Hodgkin. In a communication made by that accomplished physician to the Royal Medical and Chirurgical Society

* *Syn.*—Hodgkin's disease—Adénie—Pseudoleukämie—Lymphadenosis or Lymphomatosis.

in 1832 there are recorded several instances in which the spleen and lymphatic glands were jointly affected; of which at least two, and probably four, are examples of the disease now to be described. In Germany, Billroth, Wunderlich, Virchow, and many others have recognised the condition as *pseudoleukæmie* or *Hodgkin'sche Krankheit*. In France a case was described by Velpeau in 1839, and one by Bonfils in 1856, but the first complete account of the disease was given by Trousseau in one of his graphic lectures under the name *Adénie*.*

The glands.—In many cases an overgrowth of lympharia in some particular region has been present for two or three years before any sign of extension of the disease to other parts. Dr Gowers mentions the case of a boy whose axillary glands were excised by Mr Chr. Heath six years after they first became enlarged, and in whom, four years later, the cervical glands on the same side had alone become affected. Sometimes the morbid change has seemed to spread by continuity, as from the cervical glands to the thoracic, or from the inguinal to the lumbar; but in other instances it has apparently developed symmetrically on opposite sides of the body, as in both sets of axillary glands, or it has sprung up simultaneously in distant parts.

The affected glands are neither painful nor tender, and even when they have reached the size of pigeons' eggs, remain freely movable, and unattached to the skin. But after a time they sometimes become fused together by an extension of tumour-growth through their capsules, from one gland to another. They may reach an enormous size; there may be several packets in different regions, each the size of a child's head, and weighing after death as much as ten pounds. They are generally firm and elastic to the touch, but they may be very soft.

When a general failure of the health precedes the development of external glandular swellings, one cannot help suspecting that the affection has already begun in some of the deeper glands; and this suspicion is confirmed by a case, recorded by Wilks, of a man who died in Guy's Hospital in 1856 in an extremely weak and anæmic state, with an enlarged spleen; for the autopsy showed that the mediastinal and the lumbar glands were much enlarged, although the superficial glands were unaffected. In such cases the diagnosis may sometimes be cleared up by recognising signs of pressure; for example, spasmodic cough and dyspnoea, distension of the veins, œdema of an arm or leg, or pain in the course of the lumbar or sacral nerves. Sometimes the first sign of the cause of the anæmia is the discovery of effusion in one pleura.

Gowers some years ago made an autopsy in a case of Sir William Jenner's, in which, there being general glandular enlargement, a mass of growth extended from the abdominal glands, and involved the solar plexus and nerves going to the adrenal bodies; here there was a discoloration of the skin having the distribution of Addison's disease, notwithstanding that the adrenals themselves were healthy. Osler has since described a similar case.

* The term *Adenia* has not met with acceptance, and has a fatal resemblance to *adenocèle* and *adenoma*. Lymphatic anæmia is distinctive and can easily be replaced by a better when we know more of the nature of the thing signified. The practice of affixing discoverers' names is very inconvenient, and often historically unjust. In this very case though Hodgkin's character and service to pathology are well worthy of remembrance, his share in the merit is scarcely equal to that of Sir Samuel Wilks. Of all names, *pseudoleukæmie* is the worst. Nature is never false, and there is no more reason to call this group of cases false than to call those named *leuchæmia* by Virchow—false Hodgkin's disease.

Enlarged glands lying outside the great visceral cavities sometimes interfere with adjacent structures. In the neck they may compress the trachea or the œsophagus, or hamper the movements of the lower jaw, and in the armpit they may press on the axillary vessels and nerves, so as to cause pain and swelling of the arm. Several such cases have been recorded by oculists in which the lymphatic growths affected the orbits.

The blood.—Pallor is, sooner or later, a conspicuous symptom in Hodgkin's disease. The cheeks and the lips are bloodless and waxy-looking; the more so as there is generally subcutaneous œdema, even when albuminuria is not present. The tint is white, not yellow nor chlorotic. The patient often loses flesh as the disease goes on.

The state of the blood is that of grave anæmia. When drawn, it is strikingly pale, and has been compared to diluted claret; it coagulates slowly and imperfectly. The red discs in a case that occurred at Guy's Hospital in 1877 were estimated at 76 per cent. of the normal number, and a much greater reduction has been recorded, down to 25 per cent. (C. R. Murray). Small and irregular-shaped corpuscles are occasionally met with—microcytes and poëilocytes.

The number of leucocytes is, in most cases, normal; and if it is slightly increased, the condition is leucocytosis, not leuchæmia. The leucocytes are small, clear cells, with a single nucleus and scanty protoplasm—lymph-corpuscles. The presence of eosinophil leucocytes is certainly not diagnostic of anæmia lymphatica.

It must be remembered that many of the cases of lymphatic anæmia occur in children, who very readily develop leucocytosis. A moderate increase of white corpuscles, in the normal proportion of their several kinds, is present in scarlatina, pneumonia, syphilis, diphtheria, variola, rheumatism, and cancer, while this leucocytosis is slight or absent in enterica, influenza, and tuberculosis.

There is always enlargement of the lymph-glands, but only moderate enlargement, if any, of the spleen; and there appears to be no doubt that in most of these cases there is no trace of the change in the bones which always accompanies splenic leuchæmia.

Clinically both leuchæmia and lymphatic anæmia are varieties of ingravescens anæmia; but in most cases the observation of the blood after appropriate staining distinguishes them.

Nevertheless, mixed cases are not uncommon, and more than one case is recorded in which the leuchæmia was due to lymphocytes, not myelocytes; and yet, after death, the spleen and marrow were affected and the lymph-glands were not.

Hodgkin's disease has been observed wherever careful observations have been made, though it is nowhere a common disease. According to Macfadyen's experience, it does not occur in domestic animals.

The usual symptoms of grave anæmia accompany Hodgkin's disease. There is *dyspnœa*, which on exertion may become extremely distressing, and the respirations are accelerated, being often from 24 to 36 in the minute. *Hæmorrhage* and particularly epistaxis may appear, though less frequently than in splenic leuchæmia. The *temperature* of the body often rises to 100° or higher; in one instance it reached 103·2°. According to Gowers, the pyrexia is sometimes continuous, with slight diurnal fluctuations; sometimes it lasts only for a few days at a time, the febrile periods being separated from one another by intervals of normal temperature; sometimes

it is marked by morning remissions, the daily range amounting to 3° or even more. A persistent high temperature appears to be a sign which, more than any other, indicates a speedy and fatal termination.

In 1860 a man died in Guy's Hospital nine days after his admission; he lay with his eyes closed, and was scarcely sensible; his skin was hot, and he had occasional rigors; his spleen could just be felt; there was a mass of enlarged glands in the left side of the neck, and the autopsy showed that it was a case of Hodgkin's disease.

In a case under Professor Bäumler's care, reported from Freiburg-in-B. by Dr Brauneck, there was a temperature of 40.5° C. (105° Fahr.). 'Ueber einen Fall von mutipler Lymphombildung' (Hodgkin'sche Krankheit), 1886.

The *course* taken by cases of Hodgkin's disease is very variable. Sometimes the patient is ill only a very short period before death, and the lymphatic glands may then undergo very rapid enlargement.

In 1867 Dr Fagge recorded the case of a man aged thirty in the 'Guy's Hospital Reports' for 1881. A month previously he had a slight cough and hæmoptysis, which probably were due to tubercular disease of the lungs, since this was found to be present at the autopsy. Three weeks before his death he was suddenly seized with a dull heavy pain at his chest, and six days later, on March 16th, he became covered with purpuric spots. On the 21st hæmaturia set in. He also expectorated a quantity of blood, which seemed to come from the mouth. He was admitted on March 23rd. The spleen was then much increased in size, its edge being felt about half an inch below the ribs; but no enlargement of lymphatic glands was discovered. During the next few days his temperature ranged from 99.4° to 99.9° . The diagnosis was "purpura hæmorrhagica." On the 28th he had epistaxis. On the morning of the 30th, at about 7 a.m., he noticed, for the first time, that the glands of his neck were enlarged and tender. It was then found that many other glands of the body were likewise swollen, although not so tender. Though he was perspiring freely, his temperature was 103° . Extreme dyspnoea set in two days later, on April 1st, and he died, suffocated by œdema of the larynx, at noon on that day.

On *post-mortem* examination the cervical and the axillary glands were seen to be enlarged, so that some of them measured an inch in their long diameter; they were soft, of a pinkish cream-colour, and spotted with ecchymoses. The tonsils presented a similar appearance, and were half an inch thick. The thymus formed a large pear-shaped mass. The spleen weighed twenty ounces; it was pale and soft. The kidneys were very pale, and spotted all over with patches which looked as though they were suppurating. Leucocytes were visible in large numbers in the liver between the hepatic cells, and as many as twenty-five were counted in a single short capillary vessel in the substance of the heart. Nevertheless the blood was examined a day or two before the patient's death, without discovering any excess of white corpuscles.

As a rule, however, the progress of the disease is slow. Gowers gives a table showing the duration of fifty fatal cases, the length of which could be approximately fixed; thirty-three of them ended within two years. The most common mode of death is gradual exhaustion; but suffocation sometimes occurs from pressure of the enlarged glands upon the trachea, and sometimes starvation from pressure upon the œsophagus. Epistaxis has occasionally been directly fatal. Coma, delirium, and convulsions, without discoverable cerebral lesions, were observed in some cases by the late Dr Southey. Pneumonia, œdema of the lungs, and pleurisy are not infrequent complications, and may be the immediate cause of death. Diphtheria of the fauces has been reported frequently, but these cases were probably sloughing of lymphoma of the tonsils, not true diphtheria.

The difference in consistency noticed above (p. 768) has been connected with the course of the disease, the harder glands being more frequent in the chronic, the softer in the more acute cases; but the rule is not without exceptions. The acuter cases are more common in children.

Anatomy.—The morbid anatomy of Hodgkin's disease varies in different

cases. The affected glands usually appear whitish yellow, waxy, smooth, and firm, both on surface and on section; but sometimes they are opaque, white, soft, medullary, perhaps spotted with hæmorrhages, and occasionally of a uniform deep reddish-grey tint. They very rarely become caseous,* and only cohere, if at all, in the later stages of their growth.

It used to be said that anatomically the glands in Hodgkin's disease are merely hypertrophied. But careful examination by modern histological methods shows that, though they consist of nothing but adenoid tissue, its disposition is more diffuse and uniform, and particularly that the distinction between the medulla and the cortex is absent, or at least less distinct than it is in a normal lymph-gland.

The earliest glands to appear enlarged are the cervical, and next the axillary and inguinal. Subsequently the great collections of lympharia in the mesentery, and those in the mediastinum and behind the peritoneum enlarge. Many which are minute or invisible during health are discovered by this disease. Thus the writer saw one removed from the surface of the *latissimus dorsi* by the late Prof. Chassaignac.

The *spleen* is only moderately enlarged, its weight in the cases noted by Dr Fagge having varied from eight to twenty-eight ounces. On section there are, as a rule, found scattered through its substance several firm, whitish-yellow masses, of round or irregular shape, from the size of peas to that of hazel-nuts. Wilks used to compare them to pieces of suet in a pudding, or to the almonds in "hardbake." Sometimes, however, the spleen in Hodgkin's disease is uniformly red and homogeneous.

In a case recorded by the writer ('*Path. Trans.*' for 1870, p. 390), in a girl of seventeen, there was sloughing angina and colitis.

The *liver*, which may be greatly increased in size, sometimes contains distinct nodules, but more often it merely shows tracts of cytogenic tissue running along the portal canals, or minute nodules scattered between the lobules and distinctly recognised only with the microscope.

In a case under the writer's care in 1874 the liver weighed 88 and the spleen 83 oz., without leuchæmia or enlargement of lymph-glands (*anæmia hepatica*) ('*Path. Trans.*,' xxvi, p. 199).

The *kidneys* are also sometimes affected, either with a diffuse interstitial growth, or with more or less sharply defined tumours. There may also be recent tubal nephritis.

The solitary *follicles* of the intestine, and also Peyer's patches are sometimes greatly swollen and medullary-looking. In some cases the "lenticular" lymph-follicles of the stomach are also enlarged (Virchow, '*Krankh. Geschw.*,' p. 509);† and it is worthy of notice that the *tonsils* and the follicles at the root of the tongue may be affected in the same way, because their enlargement can be seen during life (see cases by Moxon, '*Path. Trans.*,' xx, p. 369; and Legg, '*St Barth. Hosp. Rep.*,' vol. xi).

Another organ which is accessible to clinical investigation is the *testicle*. In a patient under Dr Taylor, each epididymis was enlarged so as to be two or three times the size of the testicle; and a similar condition was recorded in one of Hodgkin's original cases.

In the former case the anterior mediastinum contained a flattened tumour, one inch thick, with the left innominate vein running through its centre. In this instance there were also in each parietal pleura large flat

* On this point see Mosler's paper ('*Virch. Arch.*,' lvi, p. 14, and *infra*, p. 772.

† In two cases at Guy's Hospital lardaceous degeneration was found in the enlarged glands, but surgical operations had been performed, which led to suppuration.

nodulated bands of lymphoid growth of a red colour, running parallel with the ribs. In other cases the pericardium and base of the heart have been invaded, or the disease has spread into the lungs from their roots.

Not infrequently the *thymus* has been greatly enlarged and infiltrated with a soft white growth (Guy's Museum, No. 3128).

Our present knowledge of the histology of the "adenoid tissue of His," and of its (leuco-) cytogenic function, throws clear light upon the close pathological relation of the spleen and lymph-glands with the thymus, the "solitary" follicles of the intestine, the "closed" or "lenticular" follicles of the tongue and of the stomach, with Peyer's patches and the tonsils, and with the diffused cytogenic tissue of the liver.

The origin and nature of this disease are, like others in this chapter, at present unknown; but it forms a pathological link between Virchow's disease (with anæmia, leuchæmia and enlarged spleen), splenic anæmia without leuchæmia or lymph-glands, and idiopathic anæmia without either leuchæmia or lymphatic overgrowth.

Diagnosis.—The recognition of Hodgkin's disease is generally easy at an advanced stage; but at the commencement, when the only symptom is a mass of glands in the neck, in an armpit, or in one of the groins, they may prove to be "simple lymphoma," or tuberculous, or syphilitic, or secondary to some deep-seated malignant tumour. As an instance of secondary sarcoma of lymph-glands, the nature of which was unrecognised during life, may be cited the case of a girl aged ten, whose body was examined in 1880, she having died immediately after an operation for the excision of a mass of glands in the left axilla and above the left clavicle. It turned out that there was a primary tumour in the left broad ligament, and that the lumbar glands were also sarcomatous, as well as one of the mediastinal glands, from which the new growth was extending into the right auricle of the heart.

Such new growths, if "innocent," are called lymphoma or lymphadenoma, *i. e.* a local tumour without anæmia and its concomitant general symptoms. If malignant, they are called lympho-sarcoma or, better, sarcoma of a lymph-gland (cf. vol. i, p. 87). In the latter case they are of harder feel and more painful. The proof of "malignancy" is reproduction in the lungs or other viscera; the distinction between lymphoma (or lymphadenoma) and lympho-sarcoma (or sarcoma beginning in a lymph-gland) is the histological distinction between lymphatic and sarcomatous growths.

It is worthy of remark that a mass of hypertrophied mesenteric glands has been mistaken for a fibroid tumour of the uterus.

The distinction between Hodgkin's disease and tubercle is almost as difficult as between it and sarcoma. A tuberculous gland is as a rule tender, adherent, with the skin reddened and œdematous, and sooner or later it suppurates. As a rule it affects one side of the neck, or one groin or one axilla. Moreover tuberculous glands are secondary to tubercle in the organs from which they derive their lymph, while lymphadenoma is idiopathic. We must remember that an enlarged gland in the case of anæmia lymphatica may become infected by the bacillus of tubercle and caseate. That in the strict sense is a complication.

Treatment.—In doubtful cases where only a single gland or an accessible clump of glands is found, it is probably well to have the tumour excised as early as possible. Verneuil recorded one striking case in which an immense mass was removed with perfect success, and the patient was still in good health seven years afterwards.

Of internal medicines, *arsenic* and *phosphorus* have been most recommended.

The reputation of arsenic began with a case of Billroth's, in which the disease had existed for ten months, the patient being a woman of forty; the cervical, the axillary, and the inguinal glands were greatly enlarged; within a fortnight after the commencement of the treatment they were already reduced in size, and after two months she was discharged with only a single gland of the size of a nut on each side of the neck. Equal success has been since obtained in several cases, although they are the minority.

Injection of liquor arsenicalis into the enlarged glands by a subcutaneous syringe has also been practised at Vienna. The writer tried it two or three times, but it produced pain and inflammation without apparent benefit.

Cacodylate of sodium is a new method of administering large doses of arsenic without producing digestive disturbance, and has been practised also by subcutaneous injection. If its therapeutical powers are as strong as its toxic powers are weak, it will be a valuable mode of treatment.

Phosphorus was first given by Verneuil; Gowers in one case saw its administration followed by diminution in the size of the glands, and improvement of the leucocytosis, but the patient died from intercurrent renal disease. It must be borne in mind that the glands have sometimes become much smaller shortly before death, independently of treatment. On the whole the evidence of phosphorus being useful is very small, and it is not unlikely to do harm.

Iron has often been given for the anæmia, but without any benefit; iodide of potassium is equally useless, unless by a happy error in diagnosis the disease is really syphilis; and cod-liver oil is as powerless in these cases as it is efficacious in those of tuberculous disease of the lympharia.

Bone-marrow has been highly recommended, and much used, but the good results are few, and the failures numerous.

The prognosis is always very grave, but the only promising mode of treatment at present seems to be the administration of large and long continued doses of arsenic.

IDIOPATHIC OR PERNICIOUS ANÆMIA.*—In the present chapter we have surveyed various clinical forms of anæmia, and have found that they may be classified in several ways. The most important is that which follows their *ætiology*, and distinguishes the want of blood which is the result of direct hæmorrhage, or in other ways clearly secondary and symptomatic, from that which is associated with special anatomical lesions of the spleen, marrow, and other lymphatic organs. None of these varieties are idiopathic, *i. e.* primary, essential, and unexplained by antecedents or circumstances. But there remain certain rare cases which come under none of the preceding heads, and are in the proper sense of the term *idiopathic*. This, therefore, appears to be their most appropriate designation until we know their true cause; and it happens to be also the historically appropriate name, since it was proposed by the physician who first recognised and described this remarkable form of disease.

Of the *pathology* of anæmia we are too ignorant for it to furnish a satisfactory ground of distinction. But we may more or less probably

* *Synonyms*.—Primary, essential, or idiopathic anæmia—Addison's anæmia—Pernicious anæmia.—*Fr.* Anémie grave—Anémie essentielle.—*Germ.* Progressive perniciöse Anämie (Biermer)—Anæmatis (Pepper).

divide cases on this basis into those which depend on want of power to form blood (*i. e.* to form hæmoglobin) and those which depend on an accelerated process of destruction of red blood-corpuscles. The former would be marked by milder symptoms and pale urine. In the latter, the symptoms would be severe, the urine would be dark, and iron would be found accumulated in the liver and other viscera. The former cases would be benefited by good food, good air, and preparations of steel; the latter would not.

Classification, based on the histology of the blood, distinguishes cases of slight diminution of the number and colour of red corpuscles without other change (mostly symptomatic), cases of moderate diminution in number with great deficiency of hæmoglobin (chlorosis), cases of great diminution of red corpuscles in number with increase of leucocytes (leuchæmia and lymphæmia), and, lastly, cases of extreme and progressive diminution of the discs in number, with microcytosis and pœcilocytosis.

Again, we might divide slight and recoverable cases of anæmia from those which show a more obstinate or ingravescent character, which, however they begin, after a progressive course show a pernicious or malignant character, and end in death. This was the point of view of Biermer, Gusselow, Immermann, Quincke, and other physicians, who, writing for the most part in South Germany or Switzerland, and sometimes from a gynæcological standpoint, have included cases of chlorosis, of menorrhagia, malaria, gastric ulcer, starvation, and other kinds of secondary anæmia along with Addison's idiopathic cases to form a heterogeneous assemblage which only agree in a fatal issue.

Addison's idiopathic cases are not only essential, and grave or "pernicious," they are also marked by two symptoms—pyrexia and hæmorrhage—which they share with Anæmia lymphatica (Hodgkin's disease), Anæmia splenica, and Leuchæmia. Different as they are in many respects, they form a natural group, and equally deserve the epithet of "pernicious," "progressive," or "grave." Moreover, they all agree in being intractable to iron and benefited by arsenic.

The history of the recognition of idiopathic anæmia is as follows:—In the year 1855, the late Dr Thomas Addison, in his work on 'Diseases of the Supra-renal Capsules,' states that the discovery of that disease had been made by him while seeking in vain to find a cause for a remarkable form of fatal anæmia, cases of which had for a long time occasionally come under his observation. To this affection, he added, he had been accustomed in his clinical lectures to apply the term "idiopathic," by way of distinction from chlorosis and other anæmic states as could be traced to "the usual causes or concomitants." Ever since, this form of anæmia has been recognised by all who have been his colleagues or successors at Guy's Hospital; and allusions to it have been made in successive volumes of our 'Reports' and elsewhere, particularly by Wilks in the 'Reports' for 1857, and in his 'Lectures on Pathological Anatomy' (1859). Dr Frederick Taylor published in the volume of our 'Reports' for 1878 no fewer than twenty-three cases which had been recorded year by year since 1853.

When, however, Professor Biermer, of Zürich, described the same conditions in 1868 under the name of "progressive pernicious anæmia," it was thought at first that a new disease was brought to light.

Before Addison's work appeared, isolated cases had been published by Coombe in 1823, by Piorry and Marshall Hall, and Dr Barclay in the 'Medical Times' for 1851. Lebert, who was then at Zürich, described what

he called "essentielle Anämie" in 1858, without knowledge of Addison's observations, and his cases were not all of the same kind. Cases recorded in New England by Channing, as early as 1842, have been discovered by Drs Pepper and Musser ('Philadelphia Med. News,' October, 1882).

The following is Addison's original description of this remarkable disorder, published in 1855:

"For a long period I had from time to time met with a very remarkable form of general anæmia occurring without any discoverable cause whatever, —cases in which there had been no previous loss of blood, no exhausting diarrhœa, no chlorosis, no purpura, no renal, splenic, miasmatic, glandular, strumous, or malignant disease.

"Accordingly, in speaking of this form in clinical lectures, I, perhaps with little propriety, applied to it the term 'idiopathic,' to distinguish it from cases in which there existed more or less evidence of some of the usual causes or concomitants of the anæmic state. The disease presented in every instance the same general character, pursued a similar course, and, with scarcely a single exception, was followed, after a variable period, by the same fatal result.

"It occurs in both sexes, generally but not exclusively beyond the middle period of life; and, so far as I at present know, chiefly in persons of a somewhat large and bulky frame, and with a strongly marked tendency to the formation of fat.

"It makes its approach in so slow and insidious a manner that the patient can hardly fix a date to his earliest feeling of that languor which is shortly to become so extreme. The countenance gets pale, the whites of the eyes become pearly, the general frame flabby rather than wasted, the pulse perhaps large, but remarkably soft and compressible, and occasionally with a slight jerk, especially under the slightest excitement. There is an increasing indisposition to exertion, with an uncomfortable feeling of faintness or breathlessness on attempting it; the heart is readily made to palpitate; the whole surface of the body presents a blanched, smooth, and waxy appearance; the lips, gums, and tongue seem bloodless; the flabbiness of the solids increases, the appetite fails, extreme languor and faintness supervene, breathlessness and palpitation being produced by the most trifling exertion or emotion; some slight œdema is probably perceived about the ankles. The debility becomes extreme, the patient can no longer rise from his bed, the mind occasionally wanders, he falls into a prostrate and half-torpid state, and at length expires. Nevertheless, to the very last, and after a sickness of perhaps several months' duration, the bulkiness of the general frame and the obesity often present a most striking contrast to the failure and exhaustion observable in every other respect.

"With perhaps a single exception, the disease, in my own experience, resisted all remedial efforts, and sooner or later terminated fatally.

"On examining the bodies of such patients after death, I have failed to discover any organic lesion that could properly or reasonably be assigned as an adequate cause of such serious consequences; nevertheless from the disease having uniformly occurred in fat people, I was naturally led to entertain a suspicion that some form of fatty degeneration might have a share, at least, in its production, and I may observe that in the case last examined, the heart had undergone such a change, and that a portion of the semilunar ganglion and solar plexus, on being subjected to microscopic examination, was pronounced by Mr Quekett to have passed into a corresponding condi-

tion" (pp. 211, 213, in Addison's collected works, republished by the New Sydenham Society in 1868).

Wilks stated in the 'Guy's Hospital Reports' for 1855 (3rd series, vol. i, p. 363) that "in that class of cases which has specially gained the attention of Dr Addison, and which he has designated idiopathic anæmia," no excess of white corpuscles is found in the blood.*

A full discussion of the question of priority will be found in the writer's article in the 'Guy's Reports' for 1882 (vol. xli, p. 236).

Antecedents.—Among the fifteen cases (all in women) described by Biermer in 1871, as also in those previously recorded by Lebert and afterwards by Quincke and by Gusserow, there were many of severe anæmia in women which were not idiopathic, but secondary to pregnancy or parturition or menorrhagia, or were combined with amenorrhœa as extreme forms of chlorosis. Thus Lebert describes his cases as "Puerperal chlorosis," and Gusserow his as "hochgradigste Anämie Schwangerer."

In another group of cases of grave or even fatal anæmia, the origin of the disease was in protracted diarrhœa, or gastric ulceration, or (as in some of Immermann's and Quincke's cases from Basle and other parts of Switzerland) privation and poverty.

Idiopathic and fatal anæmia is always rare, but may be seen in its typical form in France, Germany, and America, as well as in Great Britain,—in fact, wherever there is a large population and competent observers. It was at one time supposed to be particularly common among peasant women in Switzerland, but this probably depended on compilers including uterine and malarial cases.

In some instances the marrow of the bones has been found after death changed in the way described above (p. 762). These cases fall under anæmia myelogenica (pp. 626, 632); but in many cases of fatal idiopathic anæmia the bones have been carefully examined, and no such change has been found.

Lastly, obscure cases of severe and incurable anæmia have been found after death to be due to the presence of *Sclerostomum duodenale* (p. 294), or, as believed, to that of *Bothriocephalus latus* (p. 285).

Instead of correcting their mistaken diagnosis, some writers have published these and similar cases as "progressive pernicious anæmia" due to medullary degeneration, or to gastric disease, or to parasitic worms, and

* Again, in his 'Pathological Anatomy' (1859), Wilks wrote, "We occasionally meet with cases of fatal anæmia where no disease is found in the body, etc.;" and again, "These cases of simple anæmia which have been called idiopathic." Moreover, in the 'Guy's Hosp. Reports' for 1857 (3rd series, vol. iii, pp. 205—211) and 1859 (p. 108), he put several cases on record, referring them all to the simple or idiopathic form of anæmia described by Addison.

Habershon published a case as "Idiopathic Anæmia" in the 'Lancet' for 1863 (vol. i, p. 518). Trousseau referred to Addison's observations in the first edition of his 'Clinique médicale,' 1865 (tome iii, p. 533). So also Dr Frederick Taylor, in the 'Guy's Hosp. Reports,' 3rd series, vol. xxiii; Dr Bramwell, in a paper published in the 'Edin. Med. Journ.' for November, 1877; Dr Stephen Mackenzie, in a lecture published in the 'Lancet' in 1878, and his three lectures in the 'Brit. Med. Journ.,' Jan., 1891; Dr Coupland, in his 'Gulstonian Lectures' (1881), and the present writer, in a paper published in 'Virchow's Archiv' six years before (Bd. lxxv, 1875). Not only Addison's colleagues and pupils at Guy's Hospital, but also Dr Bristowe, Dr Quain, Dr Sutton, and Dr Howard, in Canada, were perfectly aware of the remarkable form of anæmia described by Addison.

Lépine, in France ('Revue mensuelle,' January, 1877); Gardner and Osler, in Montreal ('Canada Med. and Surg. Journ.,' March, 1877); Osler (Pepper's 'System of Medicine'); and Pepper and Musser, in Philadelphia ('American Journ. of Med. Sc.,' October, 1875, and April, 1877), confirm the same judgment. It is surprising that in Professor Immermann's article on the disease in 'Ziemssen's Handbuch' its recognition was still ascribed to Biermer, and Addison's name was not even mentioned.

have even hinted that like causes would be found in all instances if carefully looked for.

But Addison's discovery was that patients may die from want of blood, which is not preceded by hæmorrhage or exhausting discharges, which does not originate in malaria or starvation, and is not explained by organic or parasitic disease discovered after death. The anæmia is not accompanied by excess of leucocytes in the blood, nor by anatomical changes in the spleen, the lymph-glands, or the marrow, nor is it associated with the symptoms of chlorosis. It is not uniformly "pernicious," it is not continuously "progressive;" but it is "grave," and, apparently for the present, "primary," "essential," or "idiopathic."

No doubt these cases have their true cause; no disease is idiopathic in the sense of being spontaneous. But their causes are not those which are known to produce anæmia; they are at present as unknown as those of idiopathic epilepsy or psoriasis or osteo-arthritis. No doubt some rare cases of apparently secondary anæmia or of chlorosis may go on from bad to worse, uninfluenced by treatment, and at last develop symptoms of the deepest and gravest anæmia, just as plumbism or cerebral tumours may produce typical epileptic fits, or as frequently repeated rheumatism or gout or gonorrhœal synovitis may produce at last, more or less completely, the characteristic lesions of osteo-arthritis. But the important clinical fact remains that patients may die of extreme anæmia without any known cause; while in the symptoms during life, in the distribution as to age and sex, and in the appearance after death, these cases resemble one another and form a natural group.*

Since the third edition of this book, Dr William Hunter has published a most laborious and exact analysis of cases of Addison's anæmia, and argues in favour of its origin from chemical products of decomposition in the alimentary tract, in the mouth, the stomach, and the intestines. The scientific value of the observations on which this theory is founded is considerable, and they go far towards proving what has been long suspected—that the anæmia described by Addison is due to destruction, not to lack of production, of red blood discs (*cf.* p. 781). But it seems hard to believe that so remarkable and fatal a condition should depend on so common, and apparently impotent, a cause as suppuration of the alveoli of the teeth, stomatitis, or gastro-intestinal sepsis. Dr Hunter's treatise (published in 1901) is the most complete account of the malady yet published; and whether or no he has discovered its true cause, and thus deprived it of the title idiopathic, he fully accepts its distinction from other forms of anæmia, both clinically and pathologically.

Age and sex.—Among the points in which idiopathic anæmia differs widely from chlorosis is its range with respect to age and sex. Of twenty-eight cases that occurred at Guy's Hospital from 1859 to the end of 1879, sixteen were in males, twelve in females. Of eight more, supplied by a former medical registrar, Dr E. W. Goodall, which ended fatally in the next ten years (1880—1889), five were in males and three in females.

* Dr Bramwell has recorded two cases of foreign sailors, in each of whom "pernicious" anæmia appeared to arise out of an attack of yellow fever. Dr Stephen Mackenzie, in his valuable lecture in the 'Lancet' (1878), cites three instances in which it followed a severe mental shock: one patient had accidentally poisoned her father instead of giving him his medicine, another had seen a child run over in the street, and the third had been attacked by a sheep in a field, immediately before the anæmia set in. Wilks has quoted similar cases ('Brit. Med. Journ.,' November 28th, 1884). So also Lépine, Coupland, and Musser.

Putting the 36 cases from 1859 to the end of 1889 together, we have 21 men and 15 women. Of these 36 patients, 2 only were under twenty, both being boys; 4 occurred between twenty-one and thirty; 10 between thirty-one and forty; 10 between forty-one and fifty; 9 between fifty-one and sixty; and 1 at sixty-eight.

In the table published by the present writer in the 'Guy's Hospital Reports' for 1882 (vol. xli, pp. 293—303), of 102 collected cases of Addison's anæmia, all adequately examined during life, and all verified by autopsy after death, there were 6 between seven and fifteen years of age, 4 between fifteen and twenty, 29 between twenty-one and thirty, 26 between thirty-one and forty, 21 between forty-one and fifty, 13 between fifty-one and sixty, and 4 between sixty and sixty-nine. Thirty-six cases, since collected from American sources by Dr Musser, agree very closely as to the ages of the patients.

In the tabular statements of Heinrich Müller, Eichhorst, and some other observers, there is marked excess in number of female patients over male. Of 44 recorded at Zürich only 9 were men. But this depends on including secondary anæmia, due to parturition, pregnancy, or lactation, cases of chlorosis, and cases which recovered or which were unverified by inspection after death. If from Eichhorst's list of only 30 men to 65 women we exclude all but primary cases, the numbers are reduced to 12 to 11, and this is much nearer the truth.

Among 107 cases collected by the writer there were 48 in men and 59 in women. In Dr Coupland's 110 cases (some identical with the last) the figures were 56 to 54, and in Dr Musser's 39 they were 24 to 15. So that there is either no sexual disposition to the disease, or the disposing factors in each sex are counterbalanced.

Symptoms.—When a patient comes first under observation, his usual story is that for some weeks or months he has been gradually getting paler, weaker, and more breathless.

The anæmia still gradually increases, and the complexion, instead of being white, becomes of a clear lemon-yellow tint, so that the disease has been mistaken for jaundice. If, however, the conjunctivæ sometimes show a similar colour, this depends on the presence of unusually yellow adipose tissue beneath the mucous membrane. Dr Bristowe's case of fatal jaundice ('Path. Trans.,' vol. ix, p. 432) was no doubt, as he suspected, one of Addison's idiopathic anæmia, and the universal but not deep colour may have been the lemon-yellow tint of this disease, for the colour of the eyes is not mentioned.*

As a rule there is no wasting; the face and limbs are plump, and there is no deficiency of subcutaneous fat; in only a few exceptional cases has emaciation been recorded. Dryness of the mouth and of the throat is often complained of; the breath is sometimes foetid; nausea and vomiting are frequently present, especially in the morning, and there is occasionally pain after food. The bowels have been constipated in some cases, relaxed in others; in 31 cases recorded by Dr Hale White in the 47th volume of the 'Guy's Hospital Reports' (p. 162), more than half the patients suffered from vomiting, and more than a third from diarrhœa.

* In a case of a clergyman who died at the age of seventy from extreme anæmia, recorded by Dr Douglas, of Newbury, jaundice was actually present as a complication, but as there was no autopsy, there may have been carcinoma present ('Lancet,' July, 1882, p. 140).

The pulse is frequent and jerking. Pulmonary, arterial, and jugular murmurs are common.

There is always marked dyspnoea on exertion, and often palpitation. One patient said that every muscular effort caused pain at the back of the head, and this occipital headache is, perhaps, more common than that of the vertex.

Many patients have epistaxis again and again; in women sanguineous vaginal discharges frequently occur; in some persons the gums bleed or purpuric spots appear on the legs, but neither of these symptoms is common.

With the ophthalmoscope *retinal hæmorrhages* can often be detected. Biermer seems to have been the first to notice them. They appear either as linear striæ, or as rounded spots or patches, which may have whitish or yellowish centres, sometimes consisting (according to Manz) of accumulations of leucocytes. The optic discs are said to be swollen in some cases, their vessels to be tortuous, and the retinae generally to have a peculiar smoky appearance. A boy aged ten, who came under Dr Mackenzie's care, had well-marked optic neuritis. As a rule there is no defect of vision; but one of Immermann's patients went to the hospital because one eye had suddenly become blind. A patient of the writer's recovered after hæmorrhage had deprived him of nearly half the field of vision of one eye.

The *urine*, as stated by the writer in 1882, in the most typical cases of idiopathic anæmia, is of a deep clear colour, one of the characters which point to destruction rather than deficient formation of hæmoglobin. This point has since been taken up by Dr Hunter ('Pract.,' 1880) and Dr Mott ('Lancet,' March, 1889, and 'Path. Trans.,' vol. xl, p. 127); see also Mr Hopkins' observations ('Guy's Hosp. Rep.,' vol. 1, p. 354).

There is no albumen present, and urea is deficient. The ankles may become œdematous, but there is no considerable anasarca nor ascites, nor have the pericardium and the pleura contained more than a few ounces of serum after death.

Another characteristic symptom of Addison's anæmia is irregular *pyrexia*. It was first noticed by Immermann, and it has been present in almost every case the writer has seen. Its course is uncertain; the temperature sometimes rises to 104°, or even higher, and after a few days of high temperature there is perhaps a more or less prolonged interval of *apyrexia*. As a rule there are no subjective symptoms of fever. In some cases pyrexia appears early, but usually not until an advanced stage. Before death the thermometer often falls to 97°, or even to 95°.

The red discs of the *blood* are very often found altered in form (*pæcilocytosis* of Quincke and Eichhorst), but such changes are not peculiar to this form of anæmia, as was once supposed; and in some cases no abnormal microscopic appearances can be detected in the blood-discs.

The number of erythrocytes is seldom more than 1,100,000 per cubic millimetre, whereas, even in extreme chlorosis, 1,300,000 is the lowest number the writer has seen. It may reach 750,000, 428,000, or (as in a case of Lépine's) 378,750. In another under Worm Müller, of Christiania, reported by Laache, it fell to 360,000.

Some blood-discs are pale and large (instead of 7 or 7.5, 8 or 9, or even 14 μ in diameter), others are small and deeply coloured (*microcytes*),* and these seem to be nearly constantly present; they are not, however, as Eichhorst thought, pathognomonic. Nucleated red discs are seldom seen. Drs

* First noticed by Dr Leared in 1858 ('Path. Trans.,' vol. ix, p. 439).

Davy and Mackern observed that hæmoglobin readily leaves the disc ('Lancet,' 1877, ii, p. 642), and Dr Copeman that it easily crystallises ('St Thomas's Hosp. Rep.,' vol. xvi).

The white corpuscles are not increased in numbers, even relatively, to the red, so that they must share in the atrophy of the histological elements of the blood. They maintain the power of amoeboid movement on the warm stage.

Spinal symptoms.—Several cases have now been published in this country and abroad of degeneration of the lateral and posterior columns of the cord, associated with extreme anæmia, of the type now generally called "pernicious." The observations began with one of Lichtheim in 1887, and a case, with figures of the cord and of the blood, by Dr F. E. Batten, appeared in the 'Clinical Transactions' for 1891 (p. 56); see also Dr R. Russell's account in 'Brain,' 1900.

Diagnosis.—This depends upon our definition of the disease and on the rigour with which we apply the terms of our definition. Moreover, it cannot be certain until verified by an autopsy. But when the following characters are present, we may with considerable confidence make the diagnosis:—(1) Absence of organic disease and of any sufficient cause of anæmia, particularly of menorrhagia, the constant drain of bleeding hæmorrhoids, and of such parasites as the *Anchylostomum*; (2) severe and ingravescient anæmia, with considerable diminution in number (to one half and under) of the red blood-discs, together with the presence of dark, small corpuscles; (3) absence of emaciation; (4) occasional pyrexia; (5) hæmorrhages, and especially retinal hæmorrhage.

The diagnosis of primary pernicious anæmia has been made in cases which have afterwards proved to be secondary to internal cancer, to ulcer of the stomach, phthisis, mitral disease, or morbus Brightii.

The clinical likeness to Addison's disease of the adrenals, remarked by Bristowe, Broadbent, Pepper, and other writers, is less close than has been supposed. In the latter disease the patient is pale, but not excessively anæmic, the blood is normal or nearly so, and there is decided emaciation. The bronzed colour of the skin is in strong contrast with the clear yellowish pallor of Addison's anæmia; retinal hæmorrhage and pyrexia are absent, and there is frequent complication of phthisis or caries of the spine.

The distinctions from chlorosis are as clear. Apart from the age and sex of the patient, the absence of hæmorrhage and pyrexia and the good effect of martial preparations distinguish all but very exceptional cases of chlorosis.

In 1877 a man aged thirty-eight was in the clinical ward of Guy's Hospital for seventeen days with what was believed to be either acute yellow atrophy of the liver or cirrhosis. He became delirious and violent, and had to be placed in a separate room; afterwards he was insensible, but his alarming symptoms ultimately subsided and he was discharged. However, three months later he returned to the hospital, and it was then obvious that his disease was Addison's anæmia. He sank and died, and the liver was found to be healthy. Other instances of the same kind, and scarcely less striking, have occurred.

In the year 1866 a man aged sixty-seven lay for a long time in the clinical ward of Guy's Hospital with what was believed to be the idiopathic anæmia of Addison; but at the autopsy it was found that the lungs were full of miliary tubercles, and that the liver, the spleen, and the kidneys also contained them in smaller numbers.

In 1879 a woman died aged fifty-nine, whose skin had been of a lemon-yellow colour, and whose blood had been found to contain red discs of irregular form, so that the diagnosis was supposed to be beyond question. However, the heart weighed twenty ounces, the minute arteries in the pia mater were greatly thickened, and the kidneys, although large, were hard and glistening, and showed an excess of fibrous tissue under the microscope. It was a case of chronic Bright's disease.

When there is a doubt as to the possible presence of the *ankylostomum* in the case of anæmia, it is important to examine the fæces of the patient with a microscope, since the ova of the parasite can be recognised in them without difficulty.

The nearest allies of idiopathic anæmia are the other forms of grave anæmia, particularly that in which the marrow of the bones is alone affected (*leuchæmia myelogenica*). Indeed, the diagnosis must in some cases depend upon the result of inspection after death.

Morbid anatomy.—The only changes found *post mortem* are the result, not the causes, of the profound anæmia. The blood is pale, thin, and fluid, "like washings of muscle." Hæmorrhages are found in the retina and in various internal organs. The fat is of a deep yellow tint, and the muscles of a remarkably dark hue. There is more or less effusion of dark-coloured serum in the great cavities. The heart is in a state of fatty degeneration (p. 51), occasionally the diaphragm and other muscles, and sometimes the liver and kidneys also. The gastric glands have been found atrophied by Dr Austin Flint, Ponfick, Dr B. Fenwick ('Lancet,' 1877), and other pathologists. But this condition is not constant; it occurs without marked anæmia, and it would, if an efficient cause of disease, lead, one would suppose, to wasting rather than pallor. The spleen is not enlarged. Degeneration of the semilunar ganglia was described by Quekett in one of Addison's original cases, and has since been observed by Eichhorst, Brigidi, and others; but it is more frequently absent, and is probably of no significance.

A very important fact is that excess of iron has been detected in the liver, derived, no doubt, directly from broken-up hæmoglobin.

This fact, first observed by Quinke in 1876, and confirmed by Dr William Hunter ('Lancet,' Sept. and Oct., 1888) and Dr Stevenson ('Guy's Hosp. Reports,' xlvii, 166), of great accumulation of iron in the liver, and to a less extent in the other viscera, has been frequently observed in cases when steel has not been given as a drug. Another source of fallacy in the earlier cases, was that the blood was not washed out from the liver: but when this has been done, the amount of iron is still far greater than in a healthy liver. (F. G. Hopkins, 'Guy's Hosp. Rep.,' vol. 1, p. 350, and plate vi.)

If ferrocyanide of potassium is allowed to soak into a cut surface of the liver, the addition of hydrochloric acid causes no effect in an ordinary liver; but in cases of Addison's anæmia it causes precipitation of ferrocyanide of iron, and stains the tissue blue.

Dr Hunter has succeeded in imitating the destruction of hæmoglobin and the deposition of iron in the liver by the administration to animals of toluylendiamine.

Course and prognosis.—The course of the disease is not uniformly "progressive." On the contrary, there are often decided pauses, and even apparent improvement. The duration usual is from three months to a year. In 15 cases it is given as from five to eight weeks. In two cases which occurred at Guy's Hospital the disease is said to have run on for three years, and one patient stated that for seven and a half years he had been getting paler and weaker. In ten it lasted between eighteen months and two years.

According to Immermann the fatal termination is sometimes sudden from syncope; but usually it approaches very slowly, there being delirium, apathy, or complete insensibility for two or three days before death (*coma*

vigil), while sometimes a cadaveric odour has been exhaled from the body.

It must be remembered that relapses are common in this as in other forms of anæmia.

As to the possibility of recovery from the disease, there is good reason to believe that several cases have permanently recovered under, and we may hope as the result of, treatment. Addison himself met with one such case. Other writers have recorded a few, and the writer collected twenty authentic cases in the vol. xli, 'Guy's Hosp. Reports' (p. 304).

A patient was sent to the writer in March, 1880, by Dr Thurston, of Ashford. He was then fifty-three years old, and was well nourished, but for six months had been growing pale and weak. The temperature was normal, the urine dark, and the fundus oculi pale without retinal hæmorrhage. The case was regarded as either idiopathic anæmia or internal cancer. In October he was paler than ever, with more dyspnoea and œdema of the ankles, and soon after an attack of pyrexia came on. In February, 1881, he came to town again, and a considerable hæmorrhage in one retina was discovered. He took arsenic from October, 1880, till February, 1882, when he had recovered his colour and strength and felt perfectly well. He had also become free from an eczema which had long troubled him. In 1882 he was reported well, and in 1885 came to London again on account of a return of eczema, and was in good health. In 1891 Dr Uthoff, of Brighton, under whose care he then was for another complaint, informed me that there has been no return of anæmic symptoms.

Dr Hale White has since recorded a case of apparently complete recovery from Addison's anæmia in a man aged forty-two, under treatment by arsenic. Three years later he was in good health, and his red corpuscles were fully up to the average in number and colour ('Guy's Hosp. Reports,' vol. xlvii, p. 168).

Treatment.—In Addison's anæmia, as in leuchæmia and Hodgkin's disease, preparations of iron have proved to be altogether ineffectual. Too often no medicine can be found which will check its downward progress. *Arsenic*, however, has sometimes been successful in cases which appeared to be of this nature. Dr Bramwell first recorded two such instances ('Edin. Med. Journ.,' November, 1877): one is that of a man aged thirty-eight, admitted to hospital for extreme anæmia, with retinal hæmorrhages; he had been ill seven months, and became much worse under the administration of the tincture of iron, which was continued for a fortnight, but he completely recovered in the course of four months, while taking the liquor arsenicalis in doses gradually increased from two to twelve minims. Dr Finny had two successful cases ('Brit. Med. Journ.,' January 3rd, 1880).

In the patient above mentioned recovery took place under full doses of arsenic, and it was so also in Dr White's case.

Phosphorus has sometimes been thought to be useful. Dr Broadbent published a case of apparent recovery under its use in a lad of sixteen ('Practitioner,' Jan., 1875). The writer had under his care several years ago a patient who regained his appetite for a short time under the exhibition of phosphorus in doses of one twentieth of a grain, and became able to get downstairs; but he afterwards relapsed and died. In other cases it has completely failed, and perhaps has sometimes done harm.

Dr Gibson has published a case of recovery, after arsenic had failed, under the administration of β -naphthol.

Feeding with extract of red marrow has been advocated by Prof. Fraser, of Edinburgh, and appears to be worthy of further trial, although, unfortunately, it has in several cases failed in the hands of others.

A once trusted remedy, *transfusion of blood*, is probably of no use in

cases of anæmia. That the red discs retain their structure after having been transferred from one person to another seems to be probable from the fact that no change in the urine is observed after the operation; whereas, when lamb's blood is used, except in very small quantities, an invariable result seems to be the escape, by the kidneys, of hæmoglobin which has been set free by the disintegration of the foreign blood-corpuscles. But transfused blood-discs are no longer oxygen-bearers.

Transfusion was repeatedly tried on animals (1665) by Lower and Sir Christopher Wren, and afterwards on human beings (1667) by Denis and Emmerez, at Paris, where the operation had been described nine years previously. The Dutch anatomist, Regner de Graaf, devised apparatus for the purpose. The operation fell into disrepute from the many fatal results which followed, but it was revived by Dr Blundell 150 years later ('Med.-Chir. Trans.,' vol. ix; and 'Guy's Hosp. Rep.,' 1837, vol. ii, p. 256), and was extensively practised between 1870 and 1890.*

It is now ascertained that intravenous infusion of milk or of saline solution is equally useful for the time, and equally fails to prolong life afterwards.† A safer way of obtaining the same result is to inject hot water into the rectum or sheep's serum into the axilla (see a valuable article on this part of the subject by Dr Beddard, in the 'Guy's Hosp. Reports' for 1900, vol. lv, p. 29).

CHLOROSIS.‡—From the time of Hippocrates this name has been applied to a form of anæmia occurring in young women, which is marked by a sallow, olivaceous tint, by amenorrhœa and by constipation. There is no emaciation, no pyrexia, and no secondary hæmorrhage, nor is there any anatomical change as in leuchæmia or splenic or lymphatic anæmia. The peculiar complexion of this Green-sickness, as compared with other kinds of anæmia, is not observed in every case, but when present is characteristic of chlorosis. The greenish olive hue is best seen in those girls who are naturally of a dark complexion; indeed, χλωρό-, as descriptive of the pallor of fear or of death, is more applicable to modern Greeks and Spaniards than to the xanthochroic inhabitants of Northern Europe.

Origin.—At one time chlorosis was supposed to be always caused by disappointment in love; and it certainly often follows a sudden shock, or violent emotion, as in modern instances cited by Trousseau and by Dr Allbutt.

* Its history will be found detailed by Mr C. E. Jennings ('Transfusion of Blood and Saline Fluids,' 3rd edition, 1888). See also the papers by Dr Aveling ('Trans. Obstet. Soc.,' 1864) and Dr Braxton Hicks ('Guy's Hosp. Rep.,' 1869), and the Report by Professor Schäfer to the Obstetrical Society (vol. xxi, 1880). See also Dr Wm. Hunter's three lectures on 'Transfusion' before the College of Surgeons (August, 1889).

Transfusion in extreme cases of anæmia proved unsuccessful in the hands of Gusserow, Quinke, Bradbury, and Bramwell. It was practised in a case published by the writer in the 'Guy's Hospital Reports' for 1882-3 (p. 231) with only temporary benefit. But Quinke had five successful cases, including one in which arterial transfusion was practised, beside one of temporary benefit. In all but one of these cases the patients were women. In twenty cases, collected by Dr Coupland in his Galstonian Lectures already quoted (1881), there were fourteen deaths, the remaining six being those just mentioned by Quinke.

† Compare the experience in the case of Cholera, vol. i, p. 290.

‡ *Synonyms.*—Morbus virgineus, Icterus albus, Leucophlegmasia virginum.—*Fr.* Pâles couleurs, Maladie de vierge.—*Germ.* Bleichsucht.

The Greek word used by classical writers is not χλώρωσις, but χλωρότης (from χλωρός, green, pale), of which the vernacular term "green-sickness" is the translation.

It bears a close relation to the development of the menstrual function, for the age at which it shows itself is almost always between the fourteenth year and the twenty-fourth. The catamenia may have begun with more or less regularity, and there may be an interval of amenorrhœa before chlorosis appears. Those who become the subjects of the disease have often been pale from childhood, and Dr Ashwell, in the first volume of the 'Guy's Hospital Reports' (1836), declared that chlorosis, when it afterwards assumes an aggravated form, has probably always existed in some degree. In many instances menstruation has never appeared, and puberty can hardly be said to occur; the axillæ and the mons veneris continue devoid of hair, and the uterus may retain throughout adult life the form characteristic of childhood. In some cases of chlorosis the catamenia are premature; and Niemeyer thought that whenever the menses appeared early while the breasts were still undeveloped, obstinate chlorosis followed. One may see, however, marked chlorosis in a previously healthy and fresh-coloured girl: and, as Immermann says, these cases are easiest to cure. On the other hand, a girl in whom the blood-forming organs have never produced enough hæmoglobin to give a healthy tint to the cheeks will be likely to suffer more than others from the establishment of the menstrual function.

Though chlorotic anæmia is "a malady most incident to maids," we may regard the occasional development of an analogous condition in pale and delicate boys, when the glandular activity of puberty begins, "as a kind of masculine green-sickness." But anæmia in males never takes the full characters of chlorosis.

Virchow believed that in many, if not in all, of those who become chlorotic at puberty there is, besides a natural deficiency of blood, a congenital hypoplasia or imperfect development of the heart or of the blood-vessels. He found that the aorta is much smaller, and its walls much thinner than in healthy persons of the same age. Thus he speaks of instances occurring in the bodies of well-developed women, in which the aorta would hardly admit the little finger, whereas it ought to be large enough to receive the thumb; and he cites an observation, made by Rokitsky, of an abdominal aorta that was no bigger than an iliac artery should be or even a carotid. But, on referring to the description of Virchow's cases, published in 1872 ('Ueber die Chlorose und Anomalien im Gefässapparate'), we find that in several of them there was stenosis of either the mitral or the aortic orifice. Now, the aorta in its whole length has been repeatedly found extremely narrow in those who were the subjects of obstructive disease of valves on the left side of the heart. But—with a few exceptions, perhaps in the case of the mitral orifice—such affections are not congenital, but due to endocarditis occurring in childhood. Thus it seems that the hypoplasia of the aorta, instead of being itself a primary defect, is but a secondary result of the valvular lesion. Virchow speaks of some chlorotic patients being dwarfed in stature, but this may have been due to acquired disease of the heart in childhood, which often checks the natural growth of the body. After all, the cases which give an opportunity for anatomical examination after death are exceedingly few, and we cannot believe that the multitude of robust, vigorous young women who have been cured of chlorosis, would show anything but well-developed arteries.

Though the specific gravity of chlorotic blood is below that of health, the liquor sanguinis is abundant and of high specific gravity; which appears not to be true of ordinary anæmia met with in the male sex, and at earlier

or later periods in women. Dr Lloyd Jones also believes that this condition is most often met with in large families, and that chlorosis is only an exaggeration of what is a physiological preparation for the period of gestation and parturition. (Report to the British Med. Assoc., 1897.)

Symptoms.—These are generally identical with those of other forms of anæmia (p. 609). The pulse is regular, and often of increased rather than diminished resistance. Beside breathlessness, feeble muscular power, and a rapid, irritable pulse, vascular murmurs are seldom absent, and are more marked and constant than in any other form of anæmia. A systolic bruit in the pulmonary area (probably dependent on dilatation of the trunk of the artery of the lungs) is very frequent, usually accompanied by a systolic murmur in the carotid and other arteries. The most constant and characteristic bruit is a continuous *humming sound** in the internal jugular vein (p. 613). This is sometimes better heard on the left, more often on the right side; it is increased in loudness during deep inspiration, and has a slight respiratory rhythm even when the patient is breathing quietly; its locality, continuity, and loud harsh quality are very distinctive. The sound is almost exactly imitated by holding a large univalve shell to the ear.† Though rarely absent in cases of chlorosis, it may be heard in other forms of anæmia, and not infrequently in healthy persons if the head be turned away and the stethoscope pressed firmly down.

A chlorotic patient seldom grows thin, and may become fatter than before. There is no dropsy beyond slight œdema of the ankles. It does not appear that retinal hæmorrhage has ever been observed, and certainly no other local bleeding occurs; nor is there menorrhagia, but amenorrhœa. Leucorrhœa is frequently present.

One symptom of anæmia in general is almost constant in chlorosis—a torpid state of the colon. The *constipation* of chlorotic girls often lasts for many days and sometimes for a fortnight; even if the irritation of scybala occasionally produces what is called diarrhœa, the fæcal accumulation is still unaffected. This symptom, like the amenorrhœa, has been supposed to be the cause of the anæmia by poisoning the blood with retained fæcal products; but the first of the triad is certainly pallor, and the constipation may be referred to the same muscular weakness as affects the skeletal muscles. Obstinate constipation has so far wider a range of sex and age than chlorosis or any other form of anæmia, that it is impossible to accept the late Sir A. Clark's theory that chlorosis is caused by auto-infection.

The blood in chlorosis is pale in proportion to the severity of the case. The discoloration depends chiefly upon deficiency of hæmoglobin in each corpuscle, and the number of the red blood-discs is but little diminished; nor are their size and shape altered, as in the more severe forms of anæmia already described. This marked *achromatosis*, slight *oligocytosis*, and absence of *pæcilocytosis* and nucleation can be readily observed by help of the hæmocytometer and hæmoglobinometer, and by appropriate staining of a blood-film.*

The result of the laborious and extensive blood counts recently made in the Massachusetts General Hospital was an average of 4,050,000 erythro-

* *Fr.* Bruit de diable.—*Germ.* Nonnengeräusch. The late Prof. Boileau told the writer that he had adopted the name from a toy which (in 1864) had long ago gone out of use; but the *Nonne* in German means neither more nor less than a common humming-top.

† "Tu quate, sonus abit; tu levias tange labella
Auribus attentis, veteres reminiscitur ædes,

Oceanusque suus quo murmurare murmurat illa."—LANDOR.

cytes per cubic centimetre with an average of only 41 per cent. hæmoglobin, in contrast with an average of only 1,200,000 red discs in “pernicious anæmia.”

The white corpuscles are not increased in number according to Cabot, but there is sometimes moderate lymphocytosis (*cf. supra*, pp. 752, 766). Coagulation is rapid, whereas in “pernicious” anæmia it is slow.

Diagnosis.—To distinguish chlorosis from other varieties of anæmia, we depend first on the age and sex of the patient, the peculiar tint, the hæmic murmurs, the absence of loss of flesh, and the presence of constipation and amenorrhœa.

Bright’s disease, mitral disease, lead-poisoning, menorrhagia, all produce secondary anæmia, which, when it affects young women, may be mistaken for chlorosis. But these are easily recognised: in practice we have most often to make sure that the case is not one of gastric ulcer, phthisis, or idiopathic anæmia of Addison.

It has often happened that a chlorotic girl has been unexpectedly attacked by acute and fatal peritonitis; and an autopsy has discovered the cause of both anæmia and death in a perforating ulcer of the stomach.

The early stages of pulmonary disease are frequently overlooked in cases of anæmia, and the only way to avoid the mistake is to take the evening temperature once or twice every week, and to examine the sub-clavicular spaces from time to time. In 1861 a girl aged eighteen, who was admitted into Guy’s Hospital for chlorosis, gradually sank and died; a large caseous mass, which produced no marked symptoms, was found in the cerebellum, and there were a few scattered tubercles in the lungs.

Gastric ulcer and phthisis usually cause wasting as well as anæmia; but Addison’s idiopathic form of anæmia occurs, like chlorosis, in stout subjects. The distinction from chlorosis is that in the latter the diminution of colour of each blood-disc under the microscope is much greater than their diminution of number. Thus in a recent case under the writer’s care, the hæmoglobinometer showed the diminution of colour to be about 25 per cent., while the number of red corpuscles was about 3,500,000 per cubic millimetre, which is not uncommon in women. Another distinction is the absence of pyrexia, and a third the absence of hæmorrhage, especially retinal hæmorrhage. The good effects of iron would confirm the diagnosis. As a matter of fact we do not find that “pernicious” essential anæmia begins at the age or with the symptoms of chlorosis.

Prognosis.—Chlorosis is never directly fatal, but phthisis, hysteria, chorea, gastric ulcer, and exophthalmic goitre are particularly apt to occur in its course; and its presence aggravates the danger of enteric fever or pneumonia. It does not disappear without treatment.

Some women have to take steel year after year in order to keep free from anæmia. Indeed, those who have been chlorotic in early womanhood are very liable to recurrence in later life, especially if they become exhausted by child-bearing or by lactation. Hence some writers admit of a climacteric chlorosis.

Treatment.—Chlorosis resembles secondary anæmia in being amenable to treatment by iron. Splenic and lymphatic anæmia, leuchæmia, and grave idiopathic (“pernicious”) anæmia are seldom benefited, even for a time, by ferruginous medicines. But in most cases of chlorosis sufficient doses of

* A rapid method of forming a rough estimate of the corpuscular richness of the blood is by Hedin’s hæmatocrit, a centrifuge which collects the corpuscles into a graduated glass tube.

steel act in a "specific" (*i. e.* constant and unexplained) manner. The famous Griffith's mixture (*Mist. Ferri Comp.*) is deservedly trusted in England, and the scarcely less famous Blaud's pills in Germany.* *Mist. Ferri Co.* and *Pil. Aloes et Myrrhæ* go well together. Another excellent combination is sulphate of iron, sulphate of magnesia, and aromatic sulphuric acid. The alkaline preparations are generally most serviceable, but now and then the *Tinctura Ferri Perchloridi* succeeds better.

The rules of treatment are to exhibit laxatives along with the preparations of steel, to increase the latter until a decided effect is produced, and if it disagrees, to diminish the dose or change the preparation, but not to abandon the drug.

In some cases in which there is much gastric disorder, it may be advisable to give only bland preparations, such as the ammonio-citrate or the potassio-tartrate or the tasteless solution of dialysed iron. But it often happens that the tincture of the perchloride, or the sulphate, can be borne by patients who have a pale flabby tongue, nausea, vomiting, and even pain after food; and such treatment is generally attended with signal success. Large doses seem to be much more serviceable than small. Probably their advantage lies mainly in the fact that only a small part of what is swallowed is absorbed into the blood; the greater portion is converted into a sulphide in the small intestine, and passes away in the blackened fæces.

The effect of iron on the blood often appears marvellous, especially when the obvious improvement in the patient's appearance and in her breath and strength is confirmed by watching the daily increase of the number and colour of the corpuscles by means of the hæmocytometer and hæmoglobinometer.

The fact that iron is so important a constituent of hæmoglobin suggests that it acts directly as a food; but to make this view tenable one must assume that there has been a deficiency of iron in the dietary of those who are thus benefited; and there is seldom any evidence of this. One must therefore suppose that it stimulates the process by which hæmoglobin is developed and red corpuscles formed.

Much attention was attracted by the publication in Bunge's brilliant lectures on physiological and pathological chemistry (which were translated by Wooldridge in 1890), of a theory on the action of iron in chlorosis (pp. 93—105). He believed that only iron in organic combination as in food can be absorbed, and that therefore all the martial compounds given pass through the body unchanged. The objection that they cure chlorosis he met by assuming that hydrogen sulphide and alkaline sulphides are formed in the intestine, as the first step in the morbid condition, that they combine with the iron of the food, and render it as useless as if it had come from a druggist's shop; but that if inorganic salts of iron are given, they combine with the sulphides, and so protect the organic iron of the food. This ingenious theory has been confuted (1) by curing chlorosis with iron injected under the skin; (2) by experience showing that manganese or hydrochloric acid do not cure the disease as they should do on Bunge's hypothesis.

* *R. Ferri Sulph. gr. iiss, Pot. Carb. gr. iss, Sacch. gr. j, Tragac. gr. ij—iv. Misce, ft. pil.* Signetur: Two, three, and at last four or five to be taken thrice daily.

HÆMORRHAGIC DISEASES

“The mariner, his blood inflamed
With acrid salts, his very heart athirst
To gaze at nature in her green array;
Upon the ship’s tall side he stands, possessed
With visions prompted by intense desire;
Fair fields appear below, such as he left
Far distant, such as he would die to find.”

COWPER.

SCURVY—*History—Early Symptoms: purpura, spongy gums, hæmorrhagic inflammations—Night-blindness—Course and prognosis—Ætiology—Prophylaxis—Pathology—Diagnosis—Treatment—Infantile scurvy.*

PURPURA—*Symptomatic and primary purpura—Ætiology—Characters and events—Morbid anatomy—Pathology—Diagnosis—Treatment.*

HÆMOPHILIA—*History—Inheritance usually in males through females—Course and symptoms—Pathology—Diagnosis—Prognosis—Treatment.*

THIS group of maladies, like the last, are anæmic as well as hæmorrhagic, but here the anæmia is the consequence, not the cause, of the bleeding.

They resemble primary anæmia in being “diseases of the blood” in the proper sense of the term, *i. e.* they depend, so far as we know, on a primary disorder of the blood, which leads to brittleness and rupture of the capillaries, and perhaps also to want of power to coagulate.

SCURVY.*—*History.*—There is no certain account of this remarkable disease in the classical authors; but in the Hippocratic writings a condition is described, marked by foul breath, gums receding from the teeth, bleeding from the nose, and ulcers of the legs, which has been probably referred to scurvy.

At the time of the Crusades scurvy made its appearance in unmistakable form as a malady of the camp. At the siege of Damietta, in 1218, an epi-

* *Synonyms.*—Scorbutus.—*Fr.* Le scorbut.—*Germ.* Scharbock or Schormund.—*Dutch* Scheurbuick. The name is said by Immermann to occur for the first time in the Botanicon of Euricius Cordus (1534) in the German form “Scharbock,” derived from a Danish word, *Skörbeck*, signifying “disease of the mouth.” *Scorbutus* is only a Latinised form of the same. According to Professor Skeat, however, *Scheurbuyck* in old Dutch means not “sore mouth,” but torn or ruptured belly. He gives the vernacular word “scurvy” an independent origin, from scurf (scab, *schorff*, scrape). Dolæus writes, “Danicè *Scharbuck* indicat oris erosionem et ventris torsionem: inde et Foresto placet *gingibrachium* appellare, cum et brachia interdum afficiat: ab aliis et dicitur *gingipedium*, quod gingivas et pedes infectet” (*‘Encycl. Med. Dogm.,’* cap. xii, § 1). Afterwards (§ 3) he describes “*dolor intestinorum totiusque abdominis, lancinans, tensivus et quasi raptorius, unde fortasse nomen ab Hollandicis accepit, die Scürrbüyk.*”

demic of scurvy appeared, and in 1250 the army of Louis IX of France suffered from it before Cairo.

Sea-scurvy became well known towards the end of the fifteenth, and in the sixteenth centuries, among the crews who then first dared long sea voyages to the East Indies, round the Cape of Good Hope, and afterwards to the West Indies and America.

Vasco de Gama had 100 men out of his crew of 160 sick with scurvy after he had doubled the Cape. The disease frequently appears in the narratives of the voyages of Dampier, Anson, and Cook.

It was found that, beside the "sea-scurvy," there was a precisely similar "land-scurvy," which arose from time to time among the inhabitants of besieged towns, in the inmates of prisons and asylums, and among the poorer classes generally, when exposed to privation or famine. Unfortunately the subject was thrown into complete confusion by the publication of a work, 'De Scorbuto,' by Severinus Eugalenus (1658), in which the symptoms and effects of almost every disease were jumbled together. He seems to have had many followers; and one of the results of their teaching still remains in the popular use of the word "scurvy" as a name for eczematous and other eruptions, and in the traditional practice, in country districts of England, of calling obstinate chronic sores on the leg "scorbutic ulcers." Indeed, the voluminous medical literature of the seventeenth century shows so complete an ignorance of the real characters of the disease, that Hirsch doubts whether it can ever have been really common on land. During that period the term *scorbutic* seems to have been used as much as "rheumatic" is at the present time in Germany, "arthritic" in France, and "gouty" in England, to impose an arbitrary pathology on obscure symptoms. Against this use Sydenham, the first physician of modern times who depended on observation rather than authority, and on facts rather than words, strongly protested. He asserted that true scurvy, of which in the fifty-ninth chapter of his 'Processus Integri' he gives an admirably terse and accurate description, is comparatively rare in England.* Three years after Sydenham's death was published and dedicated to William III., in 1691, the 'Encyclopædia Medica Dogmatica' of John Dolæus; and in the twelfth chapter of the third book he gives an account of scurvy which presents the exact opposite of Sydenham's masterly sketch, as one sentence shows: "Prout Beatus Bontekoe non absque ratione omnes fere morbos Scorbuti nomine comprehendit."

In 1753 Dr James Lind, Physician to His Majesty's Royal Hospital at Haslar, gave a full and exact account of scurvy as seen in sailors, and this has been followed by all later writers.

The fullest historical account of this disease, now happily of little more than antiquarian interest, will be found in the late Dr George Budd's article in Tweedie's 'Library of Practical Medicine.' In the fifth volume of 'Reynolds' System of Medicine,' Dr Buzzard gives a valuable description from his own observation of scurvy as it appeared among the allied troops in the Crimea, when there were 23,000 cases in the French army alone. He reviews many other instances of the outbreak of the disease in Scotland, in Ireland, among prisoners of war, in gaols, in Arctic exploration, and among our soldiers in the Punjaub, in Burmah, and in the Cape Colony;

* "Hic enim obiter (sed et libere tamen) dicam, quod, licet nullus dubitem quin scorbutus in his plagis borealibus revera inveniat, tamen eum morbum non tam frequentem quam fert vulgi opinio occurrere, persuasum mihi habeo" ('Observ. Med.,' lib. vi, cap. v, § 9).

and he shows how neither climate nor crowding, squalor nor starvation, bad water nor salt meat, can any or all of them produce scurvy.

Onset and early symptoms.—The more definite symptoms of the disease are often preceded by a general failure of health and strength. The face becomes pale and sallow, with a livid discoloration of the lips and cheeks. So characteristic is the patient's appearance, that the cause may often be known at a glance; and when several are attacked at the same time, each is struck with the altered aspect of the others. The skin is dry and scurfy, and its hair-follicles are prominent and rough to the touch, as in the condition known as "goose-skin," and in *pityriasis tabescentium*. The muscles waste and become soft and flabby. The spirits are depressed and gloomy, the mind apathetic, while lassitude, fatigue, and shortness of breath are felt after exertion. The patient is very sensitive to cold; and pains, described as "rheumatic" (*i. e.* myalgic), are experienced in different parts of the body, especially in the loins and in the calves of the legs. These pains are worse after exertion, and are relieved by rest and sleep.

Hæmorrhage.—After a week or two an eruption appears on the skin, first over the legs, afterwards on the arms and trunk; rarely upon the head or face. It consists of reddish or purple spots, chiefly of small size, and most of them with a hair-follicle in the centre. In severe cases there may also be bullæ, containing a sanguineous fluid which presently dries up into crusts, while their bases often ulcerate. More common are larger subcutaneous extravasations of blood (*vibices*), with edges ill-defined and fading off into varied tints; they sometimes break down, and form large ulcers, with a spongy floor, exuding a thin, bloody, putrid fluid. Some of the nails may be detached by effused blood, and cast off by ulceration.

A still more characteristic symptom is the formation—sometimes the rapid formation—of ill-defined brawny indurations in the connective tissue, especially of the hams, but also behind the ankles, along the back of the thighs, over the recti abdominis, or in the armpits. The skin over them may be free from discoloration, but they consist of extravasated clotted blood, mixed perhaps with gelatinous exudation.

Blood is often poured out into the substance of the muscles, forming more or less obvious swellings, and rendering their contraction painful and difficult; or it may detach the periosteum from the bones over a more or less extensive area, most frequently along the front of the tibia, where an enlargement results which has sometimes been mistaken for a syphilitic node. A similar change may also affect a rib, or the scapula, or one of the jawbones, and may sometimes lead to superficial necrosis and exfoliation. In yet other instances extravasation takes place along the epiphysial lines of growing bones, and causes their separation; or, if there is a recent fracture which has undergone repair, the callus may soften down, and the broken ends again become loose. Or effusion of blood may take place into the joints, especially the knees and ankles.

The characters of the blood in scurvy are those of secondary anæmia. In severe cases the diminution of blood discs and of hæmoglobin may be very great, with moderate leucocytosis; but there are no characteristic or peculiar features, and no specific microbes have been discovered in the blood or the tissues.

The most characteristic symptom, which is scarcely ever absent, is the state of the gums. Their edges become bluish-red, spongy, and detached from the teeth. They are tender, and bleed at the slightest touch, and may

be so swollen as to rise above the teeth or even protrude between the lips. This change is most marked opposite the incisors, but it may also spread towards the molars. When there is a gap among the teeth, the corresponding part of the gum remains healthy; and in infants before dentition has begun, as well as in toothless old people, the gums are unaffected. Fauvel, at the Salpêtrière, had in 1847 a case in an old woman in whom a single remaining tooth was surrounded by a mass of swollen gum; the tooth was extracted, and the gum soon became level and firmer. When severe, a scorbutic state of the gums renders mastication of solid food impossible. There is a horrible foetor of the breath, the teeth become loose and may fall out,* while a grey false membrane forms on the affected parts, and the gums may slough, so as to expose the alveolar processes. The fact that the mucous membrane of the rest of the mouth remains unaffected, proves that the peculiar spongy state of the gums is caused by the presence of the teeth.

The tongue is swollen and marked by the teeth at its edges. Lasègue and Legroux speak of having frequently seen ecchymoses on the palate during the epidemic which accompanied the siege of Paris in 1871.

Hæmorrhage from mucous membranes is of frequent occurrence in scurvy. Epistaxis is the most common, but bleeding may also take place from the stomach and intestines. In many epidemics dysenteric symptoms are present, but this appears to be due to a coincidence of the two diseases. Hæmoptysis seems to be rare, except when gangrenous pneumonia sets in as a complication. More frequently the pericardium, or one of the pleural cavities, or both together, are attacked with inflammation, attended with abundant blood-stained exudation.

Although the face does not often present purpuric spots, the skin round one or both of the eyes is sometimes puffed out into purple swellings, while the conjunctiva covering the eyeball assumes a brilliant red colour and projects above the level of the cornea. Dr Buzzard saw many cases during the Crimean War of this condition, which was attended with neither pain nor discharge; they were usually severe cases, and often ended fatally. Sometimes hæmorrhage takes place in the globe of the eye, especially into the anterior chamber, and may lead to iritis, or to hæmorrhagic choroiditis, or to panophthalmitis with sloughing of the cornea.

Night-blindness.—The most curious effect of scurvy is what is known as nyctalopia or “night-blindness.”† The patient can see well during the day, and at night he can distinguish objects near a candle and even read with a good light. But when he has not the assistance of artificial light, he becomes so blind, even though the moon may be shining, that he has to be led about. The pupils may be dilated in such cases, but there are no ophthalmoscopic changes. A sudden attack of this night-blindness is sometimes the earliest indication of the disease. In vol. ii of the ‘Ophthalmic Hospital Reports’ (1859) were published articles by Dr Bryson and others,

* “Upon examination we found their teeth loose, and that many of them had every other symptom of an inveterate sea-scurvy.”—Cook, ‘Second Voyage,’ book i, chap. iv.

† Amblyopia nocturna. Hemeralopia or Hemeropia would better express the modern meaning of the word, for *νυκταλωπίασις* or *νυκτάλωψ* (an irregular compound of *νύξ* and *ᾠψ*) is used by Aristotle and Galen to signify *day-blindness*, “a defect of sight incident to children with black eyes,” only able to see by night (*v. Littré sub voce*). Nyctalopia is the term, however, used by Heberden in a remarkable case he reports of night-blindness in a man who had suffered from ague and saturnine palsy, and who probably died of chronic Bright’s disease (‘Commentaries: Oculorum morbi’). “Night-blindness” is, at all events, an unambiguous name.

founded on observations made during the Crimean War and on the fleet in different parts of the world. Mr Donald Green, oculist to the Seamen's Hospital, believed that this curious symptom is due to fatigue of the retina, and is purely functional; and this seems to be now generally accepted. It is present whenever a condition of anæmia and exhaustion is coincident with bright light during the day, and may occur from the glare of the sea or reflection from the snow.

The writer had later a case of nyctalopia under observation in a tall slender young man who had suffered from dysentery and land-scurvy in the highlands of California, due apparently, to hardships and especially to a monotonous diet of tough beef with lack of vegetables. He described the blindness at night as coming on suddenly and lasting until his diet was improved on returning to England. It was, he said, well known on the western ranches.

The order in which the various symptoms of scurvy develop themselves is very different in different cases. Sometimes the gingival affection is the first to appear, and it may remain the sole manifestation of the disease; but sometimes it follows the purpura by a considerable interval. There may have been no sign of illness until a trifling blow causes extensive extravasation of blood, or an ordinary purge is unexpectedly followed by profuse intestinal hæmorrhage, or an old chronic ulcer of the leg becomes spongy and its discharge sanious, *i. e.* thin and stained with blood.

Course and event.—The course of scurvy is slow and protracted. There is no fever, but the appetite is bad, and there is often much thirst, with a longing for vegetables and fruit. The pains in the limbs become so severe as to interfere with sleep; anæmia and emaciation advance rapidly, and subcutaneous œdema is present in many cases. The urine is sometimes albuminous, even when the kidneys are afterwards found to be healthy. The pulse becomes extremely small and weak, and the heart's impulse may be imperceptible. The muscular weakness is frequently so great that the patient faints if he attempts to sit up in bed. At the "Dreadnought" hospital ship it used to be a rule for all patients admitted with scurvy to be hoisted up the ship's side in a recumbent position. Indeed, syncope is sometimes the cause of premature death, which otherwise does not ensue, even in bad cases, until after the lapse of several weeks. It is rare for hæmorrhages of mucous membranes to destroy life. Death is more often due to gradual exhaustion and prostration, and the mind is usually clear to the last. In other cases it is the result of some complication, such as dysentery or acute pneumonia (which may pass into gangrene) or ulcerative endocarditis; or, again, it follows extravasation of blood into the cerebral membranes, or the hæmorrhagic form of pleurisy or pericarditis already mentioned.

In the great majority of cases—at least when the patient comes under medical care—recovery takes place. The improvement produced by proper treatment is often immediate and striking; but nevertheless many weeks or even months pass before the health is completely restored. The purpuric spots undergo changes of colour like those which are seen in bruises, and gradually disappear; the smaller ones round the hair-follicles merely turn brown. The brawny indurations slowly subside, but they not unfrequently leave behind them thickening and fibrous bands which may cause permanent contractions of parts of the limbs, especially at the knee or the ankle, with atrophy of the corresponding muscles.

The late Dr Carrington's experience was, that at present these protracted

cases are not seen at the Seamen's Hospital at Greenwich, and that during six years no permanent injury had followed scurvy. In the worst cases it was asserted that the rations had been short and the water bad.

Cause and prevention.—Our knowledge of the ætiology of scurvy is now so far perfect that we can predict its occurrence, and are able to prevent and to cure it.

So satisfactory a result of scientific medicine should lead us to follow carefully the path which led to it. It has been that of patient and thorough investigation, without speculation, or idle, vague, and perfunctory "explanations." It was first necessary to define what was meant by scorbutus, which in the mouth of Eraglenus and Dolæus meant anything or nothing, as scrofula did until our own day. This was done by Sydenham. The next step was to give up the "philosophical" point of view expressed by the great Dr Arbuthnot that "the scurvy is a distemper of the inhabitants of low countries, and amongst those who inhabit marshy, fat, low, moist soils near stagnating water, fresh or salt; invading, chiefly in the winter, such as are sedentary or live upon salted or smoked flesh and fish, or quantities of unfermented farinaceous vegetables, and drink bad water." It was necessary to ascertain not whether the commonplaces of ætiology there assigned *might be*, but which of them, if any, *was* the true cause of the disease: not the exciting, predisposing, or co-operant, but the efficient cause, without which the effect is not. One by one the other supposed causes have been disproved by experience, and by the middle of last century scurvy was proved, though not universally believed, to be caused by something wrong in diet. The inquiry has since been pushed further, and of all the constituents of healthy diet, the one of which the lack causes scurvy is now proved to be a due supply of *fresh vegetable food*. The question whether scurvy ever arises when a proper quantity of such food has been taken may be answered in the negative. Immermann, indeed, cites in 'Ziemssen's Handbuch' a few instances in which he says that the disease prevailed notwithstanding that there was an abundant supply of vegetables. But on looking up the first of them, an epidemic which occurred in the barracks of Rastadt, in the winter of 1851-2, Dr Fagge found that Opitz, who recorded it nine years later in the 'Prager Vierteljahrschrift,' says expressly that the poor lived almost entirely on soup and beef and dumplings, because vegetables were very scarce and dear. Even had they been cheap, it would have proved little; for it is not enough to assert that rations have been duly served out; we want to know that they have been eaten.

Potatoes have a high antiscorbutic value; the disease was exceedingly prevalent in Ireland after the failure of the potato crop in 1846, and at Millbank Penitentiary in 1832 an outbreak occurred which was directly traced to the introduction, a few months previously, of a new dietary from which potatoes were omitted. Peas and beans are incapable of preventing scurvy, and the same is true of wheat, rice, and other cereals, but water-cress and all green vegetables are useful. The modern methods of preserving vegetables in a succulent condition leave them with their antiscorbutic properties almost intact; but complete desiccation seems to destroy their usefulness. Apples tend to ward off the disease; but cider is said to have no such power, though the acid wines of France are believed to possess it.

In the absence of ordinary fruit and vegetables, many antiscorbutic plants have been discovered. Thus, Captain Cook, with wise forethought,

provided *sauer-kraut* for his seamen, and other antiscorbutic remedies are enumerated in the account of his second voyage. Scurvy-grass has often been discovered and eaten, and in the Crimea our soldiers found by experience that dandelion leaves were an excellent antiscorbutic.

It is, however, undoubtedly true that scurvy does not always show itself in those who fail to get a proper share of vegetable food, and that not only among Zulus and Esquimaux, but when the patients have been accustomed to a mixed diet. There must, therefore, be some accessory causes to produce scurvy; or some conditions must be capable of counteracting its chief cause. Certain supposed causes of scurvy have only an indirect effect; thus it is more common in cold than in hot climates, and in winter than in summer, only because a low temperature is unfavourable to the growth of plants. Nor is the complaint produced by want of sunlight, by living in cold, dark, damp dwellings (such as cellars or the casemates of a fort), by muscular fatigue, or by excessive drinking.

The absence of fresh vegetables from the dietary can, however, be made good in more than one way. Thus *fresh meat*, if eaten in large quantities, is an efficient antiscorbutic, especially when raw or but slightly cooked. Indeed, one of the earlier views with regard to the ætiology of scurvy was that it was a direct effect of the salt pork which formerly was a large part of the diet of sailors; but it has often occurred when no salted provisions of any kind had been taken.

The antiscorbutic value of *milk* was discussed in a valuable essay by the late Dr Parkes in the 'Med.-Chir. Review' for 1848. The conclusion seems to be that drinking a pint or a pint and a half of milk every day does not supply the omission of vegetables in preventing scurvy; but children and others who live mainly on milk undoubtedly remain free from the disease. On the other hand, infants suckled by scorbutic mothers have often been attacked; and it is very likely that the milk of cows fed almost entirely upon hay may not possess the same properties as when they have had plenty of grass.

The most important of all antiscorbutic agents, however, in the absence of green vegetables, are the juices of certain *fruits*, especially the orange, lime, and lemon. Their value was recognised as early as the sixteenth century by Albertus Salomon.* In 'The Surgeon's Mate,' by John Woodall, published in 1617, we read (p. 184), "The Lemmons, Limes, Tamarinds, Oranges, and other choyce of good helpes in the Indies which you shall find there, do far exceed any that can be carried thither from England. The use of the juice of Lemons is a precious Medicine and well tried."

But it is only since 1795 that *lime-juice* has been regularly furnished to ships in the Royal Navy, with the result that scurvy, which used to cause the most fearful mortality among our sailors, is now scarcely ever seen. In 1780 the number of cases of scurvy received into Haslar Hospital was 1457, in 1806 one, and in 1807 one.† This remarkable effect of wise precautions was due chiefly to Dr Lind.

Were it not for the systematic use of lime-juice, every long voyage, when vegetables are no longer to be had, would probably be an experiment demonstrating the real cause of the disease. The usual plan is to serve out an ounce daily to each man.

* Alberti Salomonis 'Scorbuti historia,' Vitæbergæ, 1574, vol. ii, p. 784.

† Sir Thomas Watson assigns the merit of the Admiralty order in 1795 to Dr Blair and Sir Gilbert Blain. It was issued shortly after the terrible effects of scurvy had rendered Lord Howe's fleet almost useless after the victory of the 1st of June.

In the spring of 1876 an outbreak of scurvy took place among the men of the sledging parties sent out from the ships "Discovery" and "Alert" engaged in the Arctic Expedition. These men had no supply of lime-juice with them, and received only very small quantities of potatoes.* In spite of assertions to the contrary, the Admiralty Committee appointed to inquire into the matter reported that the disease was really due to the absence of lime-juice. In future it would, under similar circumstances, be well to concentrate the juice so as to render it more portable; and glycerine might be added to prevent freezing.

No case of scurvy from the Royal Navy has been admitted into the Seamen's Hospital at Greenwich since 1879. Most of the patients now treated there are Norwegian sailors. A table of cases from 1864 to 1896 is given by Dr Johnson Smith, in his article on "Scurvy," in the 5th vol. of 'Allbutt's System.' The figures from 1880 onwards to 1883 are 46, 36, 28, and 15; from 1884 to 1888 they varied between 6 and 12; from 1889 to 1896 they were only 2, 3, 4, 0, 3, 1, 0, and 3 in the successive years.

Dr Neale in 1896 described in the 'Practitioner' his experience of scurvy in the arctic regions, and came to the conclusion that it is caused by want of ventilation and want of proper food, that it begins with anæmia, and that its chief antidote is fresh blood. This view was also taken by Mr F. G. Jackson, who quotes Nansen's experience to the same effect, and states that the Samoyeds of Waigatz Island, Siberia, are free from scurvy while they eat fresh meat; but if they migrate southwards, and live on salted, and often tainted fish, scurvy soon appears. In the 'Proceedings of the Royal Society' for March, 1900, Dr Vaughan Harley maintained that scurvy is not caused by lack of fresh vegetables, but by the ptomaines of tainted meat; that lime-juice does not prevent it. To support this view Dr Harley instituted experiments on monkeys, and believed that he had produced artificial scurvy by feeding them with tainted meat. The examination of the blood was most accurate and complete, but it does not appear to the writer that the experiment proves more than that tainted meat is unwholesome for monkeys as well as for man. One cannot suppose that the Samoyeds or the Esquimaux never eat tainted meat; and we see every summer in England the effects of tainted meat and game, of stale fish and ill-preserved tinned food; they are quite different from the symptoms of scurvy. The new evidence brought forward is only that fresh meat is antiscorbutic, which was previously admitted. But it was not fresh meat which banished scurvy from the English navy a century ago. Salt beef and pork, often no doubt imperfectly salted, were the food of our sailors for fifty years later, yet scurvy did not appear after the order of the Admiralty for the use of lime-juice. It still appears to the writer that scurvy is caused by the want of some element of food supplied by fruit and vegetables and fresh meat or blood. The poison of decomposed fish or meat or game, causes severe diarrhœa and perhaps collapse with vomiting, but not purpura or the other characteristic features of scurvy.

Theory of scurvy.—Why the absence of vegetables from the food should cause scurvy, and how lime-juice is able to take their place and to prevent the development of the disease, are questions upon which there has been much speculation. In the last century dilute sulphuric acid and vinegar were often used, in the vain hope that they too might prove to be anti-

* They had been given extra rations of lime-juice for some time before starting, "in order to saturate their systems."

scorbutics; and crystallised tartaric and citric acids have since been tried, but have also failed.

Sir Alfred Garrod, in 1848, put forth the theory that the essential cause of the disease was the absence of a due supply of potassium in the food. He showed that there was a great deficiency of the salts of this alkali in dietaries which were known to be liable to give rise to scurvy, and that it was present in abundance in all the substances which possessed antiscorbutic properties. It is curious that whereas his views have never been widely accepted in this country, they are, with a slight modification, upheld in Germany. This modification, which was advocated by Dr Buzzard and the late Dr Ralfe, consists in regarding as valueless those potass salts that pass out of the body unchanged, and in attaching importance to those alone which undergo conversion into carbonates and may be supposed to enter into the composition of red blood-discs or of muscle. It has, in fact, been demonstrated that nitrate of potass is incapable of preventing scurvy; but it has not yet been shown that the pure citrate or tartrate of potass possess antiscorbutic value at all comparable with that of lime-juice or fresh vegetables. Muscular fibres and red corpuscles contain more potassium salts than the serum or other tissues of the body, and this fact brings them into some relation with the antiscorbutic vegetables and fruit; but that seems to be as far as we are warranted in going.

The immediate cause of the purpura and of the other hæmorrhagic symptoms of scurvy is, no doubt, a lesion of the walls of the smaller *blood-vessels*. But hitherto no visible change in them has been detected with the microscope. In 1871 Lasègue and Legroux examined the capillaries in seven fatal cases which had occurred during the epidemic in Paris at the time of the siege; they could discover nothing but some scattered fat-granules.

After death from scurvy the blood within the body has sometimes been found coagulated; sometimes it has been liquid. It has not seldom been pale and watery; but this is only equivalent to saying that anæmia is a symptom of scurvy; and for the same reason the red discs are deficient in number. Such oligocythæmia necessarily involves a deficiency of potass. salts and of iron, which has, in fact, been shown to occur by several chemists. Laboulbène (1861) made out a slight degree of leucocytosis; but this again may probably have been merely relative, depending upon the scarcity of red discs. Mr Busk's old observation as to the increased coagulability of the blood in scurvy, though long disputed, appears to be correct.

The late Dr Ralfe investigated the state of the *urine* in scurvy, and believed that the uric acid is increased but that the acidity of the urine is diminished, and that there is a great reduction in the amount of alkaline phosphates. He supposed that the primary change in the blood is a diminution in its alkalinity.

Dr A. E. Wright, in the 'Army Medical Reports' for 1895, recorded accurate and direct examination of the blood-plasma in cases of scurvy at Netley, and believed the disease to be caused by diminished alkalinity of the blood from eating salt meat and cereals without fruit and vegetables.

Dr Geo. Lamb, however, repeating Dr Wright's observations on cases of scurvy in the Bombay jails, found no such condition as prevailed at Netley. The normal alkalinity of serum being neutralised by sulphuric acid, 30 to 35 times diluted, that of these scorbutic patients was the same as in other natives free from scurvy, $\frac{N}{30}$ ('Lancet,' Jan. 4, 1902). He also

disproved a less plausible theory of scurvy which ascribed it to the presence of the *ankylostomum duodenale* (*cf. supra*, p. 466). Scurvy is not merely severe anæmia, and Dr Lamb found no ova of the worm in the fæces of the scorbutic patients he examined. As he also pertinently remarks, these Hindoos are not meat-eaters, and yet suffer from scurvy of dietetic origin.

Diagnosis.—As a rule, the spongy state of the gums is a safe criterion between scurvy and other purpuric affections, including the “*morbis maculosus*,” to be presently described.

There is some danger of overlooking the slight forms of scurvy which occur sporadically among the poor of London and other cities. Dr Buzzard remarks that such persons, who probably never brush the teeth, attach no importance to an unhealthy condition of the gums; nor do they notice a slight petechial eruption upon the legs. What they are likely to seek relief for is supposed “rheumatic” complaint attended with muscular weakness. It is important, therefore, to be on the look-out for the other symptoms of scurvy, including the peculiar sallow complexion, when advice is asked for vague pains in the limbs. In out-patients’ practice we are thus able to detect the disease in men who have been living without vegetable food, and find our diagnosis confirmed by the presence of petechiæ and spongy gums.

Treatment.—In the treatment of persons already ill with scurvy, the administration of lime-juice is almost as important as in its prevention, and the presence of diarrhœa does not contra-indicate its use.

If fresh meat, potatoes, cabbage, and salad can be eaten, so much the better; but when the gums and teeth are very tender, the diet must often be limited to milk, beef-tea, and eggs beaten up with wine. On the Continent the yeast of beer is prescribed.

Dr Buzzard says that a daily application of solid nitrate of silver to the gums affords great relief when they are sloughing and bleeding. Washes of chlorine-water, permanganate or chlorate of potash, alum or decoction of oak-bark may also be freely employed locally, and chlorate of potash is also useful internally to subdue the stomatitis.* For the hard swellings in the legs, friction with soap-suds is said to have been used with success in the Turkish hospitals during the Crimean War, and scorbutic ulcers were dressed with lint soaked in lemon-juice, or with the bruised leaves of succulent herbs, such as the house-leek.

Infantile scurvy.—This is an occasional consequence of bad feeding, as is now established by the observations of Dr Cheadle and Sir Thomas Barlow. It often occurs with rickets, and when thus complicated has been described by German writers under the title of “acute rickets,” by Sir Thomas Smith as hæmorrhagic periostitis, and by Dr Gee as “osteal or periosteal cachexia.” The fact that young children are often given fruit and potatoes, with other improper food, probably saves many of them from scurvy. Beside the other symptoms, effusion of blood among the muscles of the thighs, under the periosteum, or at the junction of the epiphyses with the shafts of the long bones is particularly common (see Barlow’s paper ‘*Med.-Chir. Trans.*,’ 1883). This condition has often been mistaken for infantile syphilis. The gums are seldom spongy, perhaps from the absence or fewness of teeth, for the child is usually under eighteen months old.

* I have been induced to try the oxygenated muriate of potash (*i. e.* Potassic chlorate), and have found it efficacious in the true scurvy, cases of which sometimes occur among the poor in consequence of improper diet.”—‘*Medical Notes and Reflexions*,’ by John Ferriar, of Manchester, 1810, vol. iii, p. 250.

Dr Eustace Smith recommends the administration of orange-juice and the liquor of raw meat together with free ventilation of the nursery and taking the child out in the open air whenever the weather is suitable. Cod-liver oil is also useful. Dr Baly recommended potatoes suitably cooked, and this recommendation is endorsed by Dr Cheadle, who finds raw meat juice better than iron, and wine or cream better than cod-liver oil.

PURPURA.*—This affection, “the purples,” consists in the formation of spots of hæmorrhage in and beneath the skin, and occurs in many different diseases:—In scurvy constantly; in hæmophilia; with rapid and multiple sarcoma (vol. i, p. 89); in Hodgkin’s disease, splenic leuchæmia, and Addison’s idiopathic (“pernicious”) anæmia; occasionally in the course of infective endocarditis; as a complication of Erythema multiforme, particularly in cases of rheumatic fever; and lastly in severe cases of measles, of scarlatina, and variola (vol. i, pp. 164, 191, 197).

In all these cases cutaneous hæmorrhage is secondary, and occasional. But there is a condition in which it is the constant and primary or idiopathic; for at present no cause can be discovered.

In Germany this malady is known as the “*Morbus maculosus Werlhofii*.” It was described by Werlhof, a physician to George III in Hanover, who died in 1767. In his ‘*Opera Medica*,’ collected by Wichmann, a well-marked case is recorded; the patient was a girl previously healthy, who also had epistaxis, hæmatemesis, attacks of syncope, and a small and very rapid pulse; she ultimately recovered.

No doubt Werlhof would not have exactly limited his morbus maculosus to what we now regard as primary or essential purpura; nor can we even now tell that it will not hereafter be broken up according as it is due to different causes. The distinction, however, though generally accepted between “*P. simplex*,” confined to the skin, and “*P. hæmorrhagica*,” with bleeding from various mucous membranes and internal ecchymosis, is artificial and unnecessary, for “simple” cases of purpura are apt to pass into the “hæmorrhagic” form.

Some cases of purpura are due to the administration of iodide of potassium, although the corresponding sodium-salt seldom produces a similar effect. This toxic or “iodic purpura” appears to be in all respects like the ordinary form of the disease. Descriptions of it, with copious references, are given in papers by Dr Stephen Mackenzie (in the ‘*Medical Times and Gazette*’ for 1879) and by Dr Duffy (in the ‘*Dublin Med. Journ.*’ for 1880). In one case—that of a syphilitic infant, five months old—it is said to have directly followed a single dose of two and a half grains of iodide of potassium; but it is generally not seen until the salt has been taken for some days or for several weeks. In most cases, should the patient have discontinued the iodide, a fresh crop appears as soon as he attempts to resume it.

The venom of snakes produces a similar effect, and perhaps the purpura of Variola, Typhus, Plague, and other specific fevers, and of acute atrophy of the liver, may be included under the same toxic category.

The purpura which follows the severe kinds of anæmia forms a separate natural group, that of Scurvy another, and that of Rheumatic Erythema a third.

In a collection of 200 cases of purpura from the wards of the London Hospital, Dr Stephen Mackenzie found no fewer than 61 attributable to

* *Synonyms*.—Purpura simplex et hæmorrhagica—Idiopathic purpura—Morbus maculosus Werlhofii.—*Germ.* Blutfleckenkrankheit.

true rheumatism, and 10 to Bright's disease, while all other secondary cases were so distributed that less than 10 could be assigned to any primary condition; and 68 were unexplained or idiopathic.

Schönlein's *Peliosis rheumatica* was not, as described by him, purpura at all, but papular Erythema. Erythema, however, is often hæmorrhagic, especially in Rheumatic fever (vol. i, p. 487).

Ætiology.—Idiopathic purpura is more common about the age of puberty than at any other period of life; females are said to be more subject to it than males, and this applies particularly to children. A case occurred in a man of sixty-four at Guy's Hospital in 1900.

The patient is often well nourished and fresh coloured, having been apparently in good health up to the time when the spots appear on the skin, or when hæmorrhage begins from some mucous surface. Sometimes, however, he is anæmic and weakly, or half starved, or only lately recovered from an acute disease, such as enteric fever. Immermann remarks that in these cases the purpura usually shows itself when the appetite is returning and when the first attempts to stand are being made. Occasionally there is a short prodromal stage of malaise, anorexia, and headache, lasting for a few days.

Symptoms.—In the milder cases the legs are alone affected by purpura, and in more severe ones the spots appear there earlier than anywhere else, the forearms being the next parts to be attacked, and the face suffering last, or not at all. But sometimes ecchymoses come out simultaneously over the whole surface in untold numbers.

The differences of colour chiefly depend (as Dr Hyde Salter long ago pointed out) on the depth in the skin at which the blood is extravasated. Thus the more superficial ecchymoses which are seen through only a thin layer of tissue, appear bright red and sharply defined; the deeper ones are of a purple hue and fade off gradually at their edges; they are also generally larger, because the meshes of the tissue in which they lie are more open. The minute bright red spots look like the papules of dermatitis, but are at once distinguished by not fading on pressure. Occasionally the cuticle is raised into a dark bleb by blood poured out upon the surface of the rete mucosum. The spots bear no definite relation to the hair-sacs, such as has been described in scorbutus; nor do we so often see large subcutaneous vibices; but the eyelids are sometimes surrounded by broad purple rings. Sometimes retinal hæmorrhage occurs; Dr Goodhart met with a case in a child four years old.

Epistaxis is the most frequent and usually the earliest hæmorrhage from a mucous membrane. Blood is also frequently effused in the stomach, the intestines, the urinary passages, or the uterus; and occasionally in the bronchial tubes. It may ooze from the gums, and collect round the bases of the teeth in dark red or black crusts; but when these crusts are removed, the gingival tissues are never found to be swollen, spongy, nor of a purple-red colour, as in scorbutus; they are either perfectly normal in appearance, or more or less anæmic. Occasionally hæmorrhage takes place into the muscular tissue of the tongue.

In the more severe cases of purpura, when there has been a considerable loss of blood, the patient may rapidly pass into a condition of extreme anæmia, with waxy pallor of the skin, a rapid feeble pulse, and liability to faint on the slightest exertion, even on attempting to sit up. In such cases, moderate fever may be present, as in all other forms of grave anæmia.

It has been said that the blood is deficient in coagulating power, but this seems to be a mistake. There is always diminution of red corpuscles. Immermann estimated the proportion of leucocytes in a severe case at Basle; during the first few days it was normal, but afterwards there was a slight excess, as is usual after all kinds of hæmorrhage. Laache, in Christiania, however, found leucocytosis from the first. In a recent case, the number of red discs and the amount of hæmoglobin were both considerably lowered, but there was no excess of leucocytes.

Course and event.—As a rule, purpura ends in recovery. In some cases fresh spots may cease to come out after a few days, the old ones fade and disappear, the mucous membranes cease to bleed, the anæmia is quickly repaired, and within three or four weeks the patient is as well as ever. If he should get up too early it often happens that a new crop of spots may be seen upon his legs within an hour or two after his feet are first allowed to touch the ground; but these soon undergo absorption in their turn. Sometimes, however, the disease runs on for several weeks, or it may recur again and again, with intervals of many months, during which the health appears to be perfectly good.

Beside the ordinary cases of idiopathic purpura which are confined to the legs, and readily cured by laxatives and arsenic or steel, we occasionally see more severe cases in previously healthy young patients. Two of these died under the writer's care in Guy's Hospital.

One was a healthy lad aged fourteen, whose case is published in the 'Pathological Transactions' for 1884. The disease came on without known cause, and ended fatally in about ten days. While lying almost unconscious the day before his death, a house-fly settled on his face, and before it could be brushed off had left a bleeding mark. Here micrococci were found in venous thrombi after death.

The other case occurred in a robust young man aged twenty-two. The onset was unexpected, and the symptoms well marked. Severe epistaxis was effectually stopped by the inflated bag, and active treatment checked the hæmorrhage from the bowels, stomach, and kidneys. But all the ill symptoms returned, and he died on the fourteenth day. Here micro-organisms were looked for by the writer and by Mr Cheyne, who had seen the former case, but none were found; nor were they in Dr Wickham Legg's two cases ('St Barth. Hosp. Rep.' 1884; and 'Path. Trans.' 1885).

In older patients such cases are not uncommon, and prove fatal by hæmorrhage into the bladder, or by decomposition of the blood in the mouth, or by extravasation of blood on the brain.

In a recent case in Clinical Ward under Dr Hale White an elderly man died suddenly of apoplexy after a week's illness of purpura. There were extensive ecchymoses over the whole body and in the mucous membrane, particularly in the mouth, the œsophagus, stomach, and the bladder, but the lungs were quite unaffected, though the trachea showed numerous petechiæ. Of the solid viscera the kidney only was hæmorrhagic, but blood was effused under the pericardium. Lastly, there was extensive extravasation of blood on the brain, which filled both ventricles.

Anatomy.—After death the mucosa of the stomach, intestine, uterus, kidneys, and bladder is generally found spotted with ecchymoses, as are also the pleura, the pericardium, the arachnoid, the peritoneum, and even the tissue of the lungs and the medulla of the bones.

A man aged thirty-four was admitted into Guy's Hospital for purpura, and appeared to be doing well, when he became insensible and paralysed on the right side. Afterwards there was loss of power in the left limbs also, and he died on about the twentieth day of his illness. A quantity of blood was found extravasated on the left hemisphere of the brain beneath the pia mater, and also within the ventricular cavities.

Another case, which occurred in 1871, was that of a woman aged twenty, who while in the hospital for phthisis, was attacked with a severe form of purpura, and with epistaxis.

At the end of about a week she became delirious for some hours on two successive days. Then the bleeding ceased, and the spots disappeared, but she sank gradually a fortnight later with diarrhœa. The dura mater over each hemisphere was found lined with a uniform layer of blood, which was yellowish in tint, and almost membranous.—C. H. F.

Occasionally sloughing ensues as the result of purpura. Thus a girl nine years old was admitted into Guy's Hospital for gangrene of the external genitalia and purpura. So far as could be learned she had not suffered from any one of the contagious exanthemata. There was a foetid discharge, and she died in four or five days. The bladder, the vagina, the uterus, and the Fallopian tubes were all found intensely inflamed and covered with spots of hæmorrhage. Some years later a man aged twenty-three died after an illness of eight days' duration, which began with purpura of the right thigh. The lower end of the ileum, for about one foot of its length, was of a purple colour, its coats thickened and infiltrated with exudation, its serous surface coated with lymph, and its mucous membrane slightly excoriated. In another case, recorded by Zimmermann, several intestinal ulcers formed and sloughed through into the peritoneum, setting up fatal peritonitis. (See also Dr Legg's second case above referred to.)

In a few instances pigment has been found staining the deeper organs after death. A striking example of this remarkable result was recorded by Dr Hindenlang, from Prof. Bäumlér's wards. An apparently healthy post-man, who had gone safely through the campaign of 1870, was suddenly attacked with subcutaneous hæmorrhage in several parts of the body, and died two months afterwards with all the symptoms of purpura. The lymph-glands and the liver showed deep brown pigmentation, and also the pancreas and kidneys. The amount of iron in these organs was much increased, and there can be no doubt that the pigment was directly derived from extravasated blood, though no crystals of hæmatoidin were discovered ('Virchow's Archiv,' Bd. lxxix, 1880). Dr Hindenlang refers to three somewhat similar cases recorded by Orth, Tillmanns, and Wm. Müller. The pathological relation of this pigmentation to the deposit of iron in the liver observed in severe idiopathic anæmia (p. 781) is important, and shows the intimate connection between hæmatolysis and purpura.

Pathology.—Whatever the ultimate cause of Purpura, it is probable that the immediate cause is rupture of a blood-vessel from disease of its walls. Immermann suggests that the purpura which occurs during convalescence from fever is possibly due to the circumstance that the recovery of the minute vessels is sometimes retarded beyond the time at which the volume of the blood is restored and the heart regains its vigour. Dr Thin ('Med.-Chir. Trans.,' lxii) has described the minute blood-vessels as obviously altered and disorganised within the area of a bulla caused by the administration of iodide of potassium; and he supposes that purpura would follow a further stage of the process.

In a case recorded by Dr Wilson Fox, in the 'Med.-Chir. Rev.' for 1865, the arterioles and capillaries of the skin in the neighbourhood of purpuric patches were brittle, with a glistening waxy look, and assumed an intense reddish-brown colour with iodine. The patient, a man aged thirty-three, had suffered from a syphilitic eruption, which followed an indurated chancre at five months' interval. He had taken iodide of potassium for some time, but not continuously. The adrenal bodies and the intestinal mucous membrane were lardaceous.

Unna has recently reinvestigated the condition of the blood-vessels in cutaneous purpura by modern histological methods, and finds that the hæmorrhage takes place from the *venules*, at the junction of the cutis vera

with the subcutaneous connective tissue. It is probable that there is always actual rupture of the vessels—not diapedesis of red corpuscles; but the exact lesion which leads to the rupture is still unknown.

In the purpura of ulcerative endocarditis there is no doubt that the cause of the hæmorrhage is embolism of the small arteries, as in the retina and the kidneys. The arteries are sometimes the seat of lardaceous degeneration.

It does not appear that either senile atheroma or endarteritis obliterans is the cause of multiple minute hæmorrhages. Septicæmia and septic embolism produce ecchymoses of the viscera and serous membranes, and, possibly, variola and scarlatina act in a similar way.

In a case of the writer's, in 1883, referred to above, Mr Watson Cheyne found strings of micrococci in the tissues. He had previously found bacteria in a case of Dr Russell's, of Carlisle ('Path. Trans.,' vol. xxxv, p. 408), as Letzerich did in 1889 (quoted by Dr Stephen Burt, of Boston, 'Med. and Surg. Journ.,' Nov. 1, 1900). In many other instances no bacteria have been found; but Flügge, in his 'Mikro-organismen,' 1896, describes *Bac. hæmorrhagus* of Kolb, *B. hæmorrh. septicus* of Babes, and *B. hæmorrh. venenosus* of Tizzoni and Giovannini. It is doubtful whether any of these is pathognomonic, and whether all cases of purpura depend on the same cause, bacterial or toxic.

The condition of the blood is that of secondary anæmia from hæmorrhage; according to Cabot the blood-platelets are much diminished.

Diagnosis.—The diagnosis of the morbus maculosus rests upon the exclusion of the various diseases above mentioned, which may give rise to symptomatic purpura. The possibility of the sporadic occurrence of scurvy must not be forgotten, but even mild scorbutic cases are generally distinguished by positive characters—the debility and anæmia that precede the cutaneous lesions, the swollen and spongy state of the gums, the brawny induration in the hams, and the formation of each of the spots round the mouth of a hair-sac. In cases of purpura, hæmorrhages from the mucous surfaces are generally much more profuse than they are in scorbutus. Malignant sarcomatous growths must always be carefully sought for, the heart and the urine must be examined, and the state of the spleen and lymphatic glands must be investigated. In fact, the diagnosis of idiopathic purpura, like that of idiopathic anæmia, can only be certainly established after a *post-mortem* examination.

Treatment.—There is reason to believe that certain medicines are capable of preventing the formation of fresh spots of purpura and of averting the hæmorrhage from mucous membranes, which is the most serious part of the disease. Of these *arsenic* is one. Dr Habershon recommended it in the 'Guy's Hospital Reports' for 1857; it has since been commonly employed in our wards, and in one case at least it succeeded at once, when many other drugs had failed. Turpentine is strongly recommended by Dr Gee, and Dr Fagge believed it to be the most efficient hæmostatic in some instances; while he found ergot, acetate of lead, or gallic acid succeed better in others. The objection to turpentine, beside its nauseous taste, is that it may possibly favour hæmorrhage from the kidneys. Immermann says that it is important not to treat the consecutive anæmia by ferruginous preparations for some time, and that their too early administration has led to a relapse. Chlorate of potash is often of service, but is not so certain a remedy as in cases of local hæmorrhagic stomatitis (p. 297). The patient

should be strictly confined to bed in a cool room; he should have a light milk diet, and his bowels should be kept well open. In severe cases stimulants must be freely given. Dr Eustace Smith recommends in previously healthy children oil of turpentine in castor-oil as a drastic purgative.

Dr Mackenzie justly remarks that, before treating purpura, we should endeavour to ascertain its cause. But excluding febrile, toxic, and embolic cases and scurvy, there remain a large number which we must treat empirically. He finds turpentine (in two-drop doses, with tincture of quillaia or mucilage, three times a day) the most often useful. As in the writer's experience, he has been disappointed by ergot and other astringents. Dr A. E. Wright, of Netley, recommends calcium chloride in scruple or ten-grain doses every three hours, with a view to increase the coagulability of the blood; but this seems to act better in other forms of hæmorrhage than in idiopathic purpura.

In the writer's practice he has found that arsenic and tincture of steel in full doses are both valuable, turpentine in some cases even more so; but when these remedies are resisted the case is nearly hopeless.

Henoch's purpura.—In 1874 a peculiar form of purpura occurring in children was first fully described by Henoch. Dr Mackenzie cites a case recorded by Willan in 1804. The peculiarity is the association of cutaneous purpura with multiple synovitis and hæmorrhagic gastro-enteritis. Severe colic with vomiting and hæmatemesis is followed by painful swelling of the joints and ecchymoses on the surface; or the synovitis may appear first. It is extremely apt to recur, and may thus last for months. There is little or no pyrexia, and salicylates are said to be useless. The prognosis is, as a rule, good; but Osler lost three out of eleven cases.

HÆMOPHILIA.*—For about a hundred years it has been known that in certain families the males during successive generations are liable to protracted and sometimes fatal hæmorrhage after injuries of no great severity. In 1803 an American physician, Dr J. C. Otto, gave an account in the 'Medical Repository of New York' (1803), of such a family of "bleeders," in which this habit had appeared again and again for seventy or eighty years. In 1784 Sir William Fordyce had recorded the case of a Northamptonshire family affected in a similar way. A valuable monograph on the disease was published by Dr Wickham Legg in 1872.

Ætiology.—The isolated occurrence of hæmophilia is exceedingly rare. When a "bleeder" is born of healthy parents, some or all of their subsequent male children are also affected; and probably the disease had already existed in a grandfather or some other ancestor.

The preponderance of males among those who suffer from hæmophilia is nearly as thirteen to one. Moreover, when it does affect women, hæmophilia is much less severe, and scarcely ever fatal: its signs are often limited to the occurrence of cutaneous ecchymoses, spontaneously or after slight injuries—or, in other cases, to epistaxis, menorrhagia, or excessive post-partum hæmorrhage. Immermann, however, quotes an exceptional in-

* *Synonyms.*—Hæmorrhaphilia (Schönlein), a barbarous word, intended to mean "love of bleeding" (*αιμορραγία, φιλία*)—Hæmorrhagic diathesis.—*Fr.* Hémophilie.—*Germ.* Bluterkrankheit. Dr Wickham Legg mentions the occurrence of *Hemophil* as a proper name in Ford's 'Broken Heart' (1633). It there is applied to a thrasonical courtier, who boasts of carnage he has made in the field (Act i, sc. 2); and no doubt means "a lover of bloodshed."

stance, recorded by Reinert, of a family of sons and daughters, in which the daughters alone were bleeders, while the sons were all free.

With such rare exceptions, the disorder is inherited through women, but affects males alone. The sons in a hæmorrhagic family do not all suffer; and if any escape, their children are almost always exempt; even those sons who are affected, if they live to beget offspring, rarely transmit their infirmity to their sons. But their daughters, though they have not themselves shown a sign of hæmophilia, are almost certain to transmit it to their male children. The same curious law of heredity is seen in some cases of gout, and still more constantly in the transmission of colour blindness.

At Tenna, in the Grisons, there were once two families, not known to be related to one another, in which hæmophilia had existed for a century. In 1855 the females of these families determined to renounce marriage, and in 1879 Immermann was able to state, on the authority of Dr Hörsli, of Tüsis, that there was no longer a well-marked example of hæmophilia in the village.

It is said that hæmophilic families are unusually large, the average number of children being as high as nine.

Hæmophilia occurs indifferently in those who are well nourished or thin, of dark, ruddy, or light complexion; but those subject to it are said to have a thin, delicate, and transparent skin, with full subcutaneous veins.

The disease has repeatedly been observed in Jews, and a large proportion of recorded cases have occurred in nations of Teutonic as compared with those of Latin origin.

Course and symptoms.—Hæmophilia does not usually manifest itself at the time of birth, when the navel-string is severed; but about the end of the first year, or at least before the close of the second, definite symptoms generally appear. The latest recorded age at which hæmophilia showed itself in a serious form appears to be the twenty-second year.

The hæmorrhage which marks hæmophilia may be traumatic or spontaneous, but the distinction is only partially applicable, since slight injuries are very apt to be forgotten. Thus spots and patches of effused blood in and beneath the skin may sometimes be traced to the pressure of the clothes. But there is no doubt that they sometimes arise independently of any such cause, especially when they are so small as to be termed petechiæ. Indeed, successive crops of cutaneous purpura may be observed in this disease, exactly as in the "*morbus maculosus*."

Spontaneous hæmorrhage from the mucous surfaces is sometimes preceded by throbbing of the heart and arteries, redness and heat of the cheeks, ears, and lips, headache, giddiness, restlessness, and irritability of sight and hearing. Epistaxis is the most common form of mucous hæmorrhage, especially in children; according to Grandidier it is four times as frequent as any other. Next in order of occurrence comes bleeding from the gums and mouth; this, however, may be in part traumatic, for Dr Legg mentions that some patients cannot use an ordinary tooth-brush without drawing blood. Again, there may be hæmorrhage from the stomach, the intestines, the lungs, the urinary passages, the female genitalia, or even the lachrymal caruncle. Blood does not often escape from the unbroken skin, but cases are on record in which it has oozed from the finger-tips or the ears.

In addition to these spontaneous or *quasi*-spontaneous hæmorrhages are those which are directly traumatic. Even slight superficial scratches, such

as would scarcely be noticed in a healthy subject, may bleed so as to endanger life. There are, however, considerable differences as to the amount of injury that can be borne, not only in different hæmophilic patients, but in the same patient at different periods. One cut may cause but slight loss of blood, whereas there may be the greatest difficulty in checking the oozing from a precisely similar injury on a later occasion. The operation of ritual circumcision has several times proved fatal. So have venesection, the application of leeches or cupping-glasses, and, above all, the extraction of a tooth. Indeed, although Dr Legg says he has seen a tooth drawn without there being any remarkable amount of hæmorrhage, both he and all other writers are agreed that extraction is a hazardous operation in those who are "bleeders." The slight punctures made for vaccination appear not to give rise to much bleeding.

The risk of hæmorrhage from a wound continues until it is completely healed; for a thin cicatrix has been known to give way after having formed.

The deeper structures may also be the seat of large extravasations of blood which are almost peculiar to hæmophilia. A subcutaneous tumour may be formed, the size of an apple or even of a child's head. It sometimes seems to rise spontaneously, but very often it is due to some slight injury. Thus, in a case of Sir William Jenner's, cited by Dr Legg, the fall of an india-rubber air-ball upon the thigh filled the connective tissue with blood from the knee to the trochanters.

The seats of hæmorrhage are generally of a black or dark blue colour, surrounded by a zone of red; sometimes very hard, sometimes fluctuating. They are often painful, hot or tender to the touch, and in some cases they suppurate, and discharge a mass of altered blood with shreds of broken-down tissue. If they are punctured, dangerous bleeding follows; but if left alone they slowly subside, and at last disappear.

In hæmophilia the effusion of blood into any one of the large serous cavities seems to be rare. Immermann cites two cases in which the peritoneum was the seat of hæmorrhage, and four in which it occurred in the cerebral membranes; but in at least three of these latter there had been a fall or a blow upon the head. Extravasation into the pleural sac or into the pericardium is very rare; but in a case of hæmophilia Dr Goodhart found the right pleura closed by adhesions which were in part stained of a deep orange colour.

Articular affection.—One of the most remarkable features of hæmophilia is effusion into one or more of the joints, especially the knees; and most often in the case of boys between the seventh and the fourteenth year. It is sometimes the direct result of a blow or of a long walk; but Dr Legg says that the most common cause is exposure to cold or the occurrence of damp and chilly weather, and that it is most frequently seen at the beginning of spring or at the end of autumn. The swelling looks precisely like that of rheumatic or traumatic synovitis; but it is due to extravasation of blood, not to serous effusion into the joint. No doubt after a time the cartilages show signs of chronic inflammation, and the projecting folds of synovial membrane are thickened and swollen. This was markedly the case in the knee of a boy who was under Mr Bryant's care at Guy's Hospital in 1880, and in whom the joint had been more or less swollen for three years; but in the same patient there were other joints in which after death all the structures were found healthy, except that they were stained by orange-coloured pigment, and covered with stringy masses of ochre-

brown fibrin. Even when a great quantity of blood is poured out into a knee-joint, no discoloration is visible through the skin.

The usual course of the articular affections of hæmophilia is slowly to subside under treatment, but to return again and again at intervals of months or years. After this repeated articular hæmorrhage, the synovial membrane becomes stained with hæmatoidin, the cartilage opaque and thin, adhesions form, and the condition is not unlike that of the slighter cases of osteo-arthritis. Dr Legg refers to several preparations showing this condition in the museum of St Bartholomew's Hospital, No. 740.

Diagnosis.—It is not difficult to recognise hæmophilia in confirmed cases; but one must keep in mind the possibility that an effusion into the joint may be due to this cause, even when it occurs in a person who may not be anæmic, who exhibits no purpuric spots, and who does not tell us he is a bleeder. The real nature of the less marked examples, such as occur in women, could never be determined without the history of family predisposition. Many women have a tendency to bruise very readily, and others are subject to the recurrence of spontaneous hæmorrhages, especially "hæmatidrosis," in which blood oozes from the mouth of the hair-sacs or sweat-glands; but this is not true hæmophilia.

In the 'Medical Times and Gazette' for 1871 Dr Legg recorded two cases, in women, of a "hæmorrhagic diathesis," in which the resemblance to hæmophilia was nearly complete, there being a great liability to hæmorrhage from slight wounds, and also to epistaxis, menorrhagia, and purpura. In each patient the disorder had been present for some years, but in neither of them did it exist before puberty. This last circumstance could hardly exclude hæmophilia, because many of the slight forms which are seen in women seem not to be recognisable during childhood; but what appeared to be conclusive was that each patient had borne male children who were not bleeders, and that no family history of hæmophilia could be elicited.

Pathology.—The nature and cause of hæmophilia are quite unknown. Coagulation is very slow; but there is no proof of any abnormal condition of the blood, except as the result of the continuance of hæmorrhage. So, again, the fatty change sometimes found in the substance of the heart is clearly consecutive to the anæmia which commonly precedes death. Several observers have noticed a peculiar thinness and transparency of the arteries—not only of the aorta and pulmonary artery, but also of such branches as the temporals and radials. Others, however, have failed to discover anything of the kind; and the most that can be said is, that although not itself the cause of the symptoms of the disease, it may possibly point to the existence of a similar abnormality of the capillaries, and that this may really be the cause.*

Dr Legg suggests that hæmophilia may depend upon a backwardness of growth or imperfect development of the vascular system generally. Immermann also speaks of a "hypoplasia" of the vessels; but he thinks that the essential cause of hæmophilia is a disproportion between the capacity of the circulatory apparatus and the volume of the blood. He imagines that in hæmophilic males there is an actual overgrowth of the blood itself, and he ingeniously endeavours to account for the transmission of the disease by

* See a paper by Dr Percy Kidd ('Med.-Chir. Trans.,' vol. lxi, p. 243), and Dr Legg's account of four other cases ('Path. Trans.,' xxxvi, p. 490); also Dr Theodore Acland's description of the state of the thymus (ibid., p. 491).

females who themselves are not bleeders by supposing that they have the thin vessels, without the excess of blood. Sir William Jenner's authority may be quoted ('Brit. Med. Journ.,' 1876, vol. ii) for the view that there is in hæmophilia "a tendency to plethora of the smaller vessels." He remarks that it is when the patient is looking his best, that injuries have the worst effect and that spontaneous hæmorrhage is most likely to occur.

It seems generally agreed that "bleeders" are not ill-formed, and that they show no sign of disease of the heart or blood-vessels. In fact, they are not recognisable until hæmorrhage occurs.

Prognosis.—In some exceptional cases of hæmophilia the hæmorrhagic tendency may cease during childhood, and never return; but the larger number of those who are affected die before they are eight years old. Grandidier (quoted by Eustace Smith) found that, of 150 boys who were bleeders, more than half died under seven years of age, and only nineteen reached twenty-one. When adult life has been reached, the danger is less, but it is by no means at an end, for fatal bleeding has been known to occur as late as fifty or sixty years of age.

Dr Legg gives a better prognosis than some others who have seen many cases of this strange disease, and believes that with sufficient care bleeders may avoid injuries and attain middle life.

Their habitual condition, even when they have long suffered from the disease and are perhaps still troubled with the articular complication, is not generally one of permanent anæmia; they often have as much colour in the face and lips as other people. They may of course be blanched for a time by a profuse loss of blood, for the quantity poured out is sometimes enormous. One case is related in which, after the extraction of a tooth, half a gallon was lost in less than twenty-four hours, and often oozing goes on at the rate of three or four pints in the twenty-four hours for several days together. When the source of the hæmorrhage is visible, it seems to come not from a single vessel, but from the whole surface, as from a saturated sponge. Jenner thought it was venous rather than arterial or capillary in origin. If the bleeding goes on, the patient becomes pallid, pulseless, delirious, and unconscious, and death is often preceded by convulsions. But sometimes, when his vital powers are reduced to the lowest ebb, the oozing, which may have resisted all treatment, ceases spontaneously; he remains apparently on the brink of dissolution for several days, and then slowly revives.

Treatment.—In the diet of bleeders, Jenner recommended "a considerable proportion of white meats." An aperient dose of sulphate of magnesia may be given every week, and a mercurial purge every third week. Dr Legg has found that after taking iron, patients have been less liable to spontaneous bleeding and have lost less blood after wounds; and advises the tincture of steel and cod-liver oil. The patient must wear flannel, and avoid all risks of injury. A warm and dry but, if possible, bracing climate is desirable.

When actual hæmorrhage is going on, there appears to be no styptic so valuable as the perchloride of iron; a strong solution may be applied to the mucous membrane of the rectum, if that is the seat of hæmorrhage; and when the socket of a tooth bleeds after extraction it may be checked by the introduction of solid crystals of the salt.

DISEASES OF THE SKIN

“His native beauty is a lily-white,
Which still some other-coloured stream infecteth,
Least like itself; with divers stainings dight,
The inward disposition it detecteth:
If white, it argues wet; if purple, fire;
If black, a heavy cheer and fixed desire;
Youthful and blithe if suited in a rosy tire.”

PH. FLETCHER: *The Purple Island*.

INTRODUCTORY CHAPTER

General pathology and anatomy—Classification: examples—Nomenclature—Historical sketch—The elementary anatomical lesions—The local distribution of cutaneous affections: in depth (bathymetric) and over the surface (regional)—Symmetry—The circumstances: age, occupation, and habits of the patient, season, history of the malady, subjective symptoms, disturbance of other organs, effect of previous treatment—Arrangement followed.

It is now well known that diseases of the skin differ in their origin and significance no less than diseases of the tongue, the eye, or the bladder. Some of them are symptomatic of specific febrile diseases, of which they form a part, and are important for the sake of diagnosis. Others are produced by certain articles of food, by poisons, or by drugs. Some are examples of pathological processes which are familiar in other organs, as cancer, hypertrophy, atrophy, hæmorrhage, pigmentation; but the majority belong to the wide-spread morbid process called inflammation.

We have seen that although this term may still be used to denote the reaction of a living tissue to injury, and although the four Galenical signs of inflammation, and the fifth modern sign of pyrexia, are still useful as pointing to the fact of exudation of plasma and leucocytes from the blood, yet the different kinds of inflammation vary greatly in origin and results. Serous exudation, chronic interstitial inflammation, and suppuration form three very different pathological groups. The first is often difficult to distinguish from dropsy, the second is closely related to atrophy and cicatrization, and the third is the direct result of infection by pyogenic microbes.

In the case of the skin it is important to recognise not only external irritants, and internal mineral or vegetable poisons or animal toxins, but also the effect of streptococci and staphylococci in causing common suppura-

tion, that of the bacilli of tubercle and leprosy in causing granulomata, the effects of various parasitic fungi, and the results of syphilis and other specific infections.

Dermatitis, however, is often independent of any ascertained irritant or infection, and therefore must at present be called primary or idiopathic. In this respect it is most nearly allied to the inflammations which affect the bronchial mucous membrane, the stomach, the intestines, and the urinary tract. Closely resembling these mucous membranes in its origin and development, its general anatomy, its vascular and nervous relations, and its glandular apparatus, the skin has also a close pathological alliance with them.

Anatomy.—The histology and the physical and vital properties of the skin are now well ascertained. The condensed, highly vascular, and very sensitive cutis, with its papillæ and touch-corpuscles, is continuous below with the much looser connective tissue of the subcutaneous fascia, arranged in bundles with large open spaces, which are filled with adipose tissue, except in certain regions, as the eyelids, the ear, and the genital organs.

Above, it is covered by the epithelium, epidermis, or ectoderm, of hypoblastic origin, non-vascular, but traversed by minute nerve-branches. The deeper part, *rete mucosum*, or Malpighian layer, is inseparably attached to the papillary layer of the cutis, where a single layer of clear, nucleated cells (*stratum lucidum*) closely follow the papillary surface. Another layer, the *stratum granulosum* of Langerhans, intervenes between the epiderm and the derm only in certain regions. The uppermost cells of the Malpighian layer are granular and flattened, then come some more transparent, and the rest of its thickness is made up of large polygonal minutely granular cells, with large nuclei. They are connected together by interlocking processes, which led Max Schultze to give them the name of prickle-cells.

The superficial horny layer of epidermis (the scarf skin or *cuticula*) is readily separated from the Malpighian layer. It is quite without sensation, and serves only to protect the parts beneath.

The human skin is everywhere impermeable to water, owing to the sebaceous secretion which lubricates the horny cuticle; but it can be traversed by oily matters, and by minutely divided insoluble powders rubbed into the surface with fatty compounds, either mineral, like vaseline and other kinds of paraffin, or vegetable, like olive oil, or animal, like lard, lanolin, and spermaceti.

The skin is contractile, elastic, and extremely resistant. When over-stretched, the papillary layer gives way without rupture of the epidermis, and thus smooth and slightly depressed white marks are made, which are a kind of superficial scar. They are known as *lineæ atrophicæ*, and may be seen in women after pregnancy (*lineæ gravidarum*), or on the breasts after lactation, and on the abdomen after ascites, or on the flanks, thighs, and hips after anasarca.

The horny cuticle is thickest on the palms and soles, thinnest on the face, and thinner on the back, trunk, shoulders, and buttocks than on the limbs. The cutis or corium is thickest on the shoulders, and thick on the scalp, face, and trunk. The whole skin is thinnest on the eyelids, the genitals and perinæum, and the lips.

The hairs are thickest and deepest rooted in the eyelashes and the nasal and anal vibrissæ and the beard; absent only on the palms and soles, the penis, and the prolabium. The sebaceous glands are appendages of the

hair, two usually opening into each hair-sac. The sudoriparous or coiled glands are believed, notwithstanding some evidence against the current doctrine, to secrete the sweat. They are largest in the skin of the palms and soles.

Classification.—Affections of the skin are best studied as examples of general morbid processes, modified by the peculiarities of the affected tissue. Any arrangement of diseases is useful so far as it helps the memory to retain useful facts; any arrangement is useless or mischievous if it pretend to be a universal or “natural” or “scientific” system. Diseases are not natural objects; they are physiological states, which we sometimes define by their cause, as lead palsy and scabies, sometimes by their histology, as sclerosis of the cord and epithelial cancer of the lip, sometimes by their constancy in transmission, as measles and typhus, sometimes by the presence of a microphyte, as tuberculosis, or of a microzoon, as malaria, and sometimes by more or less constant concurrence of symptoms, as chorea and epilepsy.

The following examples show how cutaneous disorders may be classified on different principles, each of which is useful in its place.

I. *Diseases of the Skin regarded as Anatomical Conditions of certain Layers, Organs, or Regions.*

A. *Bathymetric Distribution.*

The horny cuticle—Psoriasis, ichthyosis, cornu, clavus, sudamina, xerodermia; all of them conditions secondary to errors of formation in the cutis or the cutaneous glands.

The Malpighian rete and papillæ—Eczema, scabies, secondary syphilis, impetigo, exanthems, erysipelas; Erythema, pemphigus, psoriasis, lichen planus not leaving scars.

The deeper cutis and subcutaneous fascia—Zona, acne, variola, vaccinia, varicella, phlegmonous erysipelas, carbuncle, late syphilis, lupus, lepra, carcinoma leaving scars.

Elephantiasis, xanthelasma, sclerodermia not ulcerating.

B. *Distribution to Organs.*

Sweat-glands—Sudamina.

Sebaceous glands—Miliun, comedo, acne, lupus eryth. Seborrhœa sicca.

Hair sacs—Ringworm, favus, area, sycosis, furunculus.

Nails—Onychia, onycho-mycosis, psoriasis, atrophy (lineæ transversæ).

C. *Surface Distribution.*

Scalp—Eczema (especially impetigo), phthiriasis, favus, ringworm, area.

Face—Dermatitis from exposure, ephelis, rashes of smallpox, measles, and syphilis, erysipelas, lupus, acne.

Forehead—Supra-orbital zona, chloasma, acne frontalis.

Eyelids—Xanthelasma, milium and sebaceous cysts.

Nose—Lupus, gutta rosea.

Bridge of nose and cheeks—Lupus erythematosus.

Nose and lips—Impetigo of children.

Upper lip—Symptomatic herpes.

- Lower lip—Flat-celled epithelial cancer.
 Beard—Sycosis.
 Ears—Eczema, xanthelasma tuberosum, tophi.
 Neck, front—Intertrigo.
 nucha—Furunculi.
 Shoulders and back—Prurigo pedicularis, acne, carbuncle, lichen marginatus.
 Chest—Rashes of scarlatina, syphilis and other exanthems; tinea versicolor.
 Breasts—Intertrigo, scabies, pre-cancerous dermatitis.
 Abdomen—Rash of enterica, typhus, and other exanthems.
 Sides of trunk—Zona.
 Genitals—Eczema, scabies, elephantiasis scroti, herpes præputialis, tinea marginata, soft chancre, and primary syphilis.
 Nates—Scabies (in children), congenital syphilis, furunculi, intertrigo.
 Elbows, extensor side—Psoriasis, xanthelasma tuberosum.
 Forearms and back of hand—Erythema, hydroa (erythema iris), lichen planus.
 flexor side—Eczema, xanthelasma planum.
 Wrist and between fingers and toes—Scabies.
 Fingers and toes—Pernio.
 Palms and soles—Syphilis (especially squamous and bullous forms).
 Axilla and groin—Eczema.
 Knees, extensor side—Psoriasis.
 flexor side—Eczema.
 inner condyle—Lichen planus.
 Legs—Chronic dermatitis, varicose and syphilitic ulcers, elephantiasis, erythema nodosum, purpura.

II. *Classification of Diseases of the Skin regarded as Physiological Processes.*

(Pathological Arrangement.)

Acute inflammations:

- Diffuse, *e. g.* scarlatina, morbilli, syphilis, roseola.
 With venous congestion—Erythema nodosum.
 With œdema—Urticaria.
 With necrosis—Furunculus, anthrax.
 Localised in papules—Enterica, syphilis, eczema, prurigo, lichen.
 in vesicles—Eczema, zona, variola, varicella, scabies, herpes.
 in blebs—Pemphigus, scabies, rupia, pemphigus neonatorum.
 in pustules—Impetigo, variola, scabies, syphilis, sycosis, acne.
 Desquamating during involution—Scarlatina, eczema squamosum, &c.

Chronic inflammations:

- With over-production of epidermis—Psoriasis.
 With fatty degeneration—Xanthelasma.
 With hypertrophy—Elephantiasis (Arabum), molluscum fibrosum, &c.
 With œdema—Elephantiasis (lymphatic), œdema durum.
 With venous congestion—Gutta rosea, pernio.
 With ulceration—Lupus, syphilis, lepra.
 New growths—Xanthelasma, lupus, lepra, tertiary syphilis, cheloid, cancer.
 Atrophy—The senile skin, lineæ gravidarum.
 Hypertrophy—Ichthyosis, cornu cutaneum, clavus, verruca.

Hæmorrhage—Traumatic (flea-bites), typhus, scurvy, erythema (peliosis).
 Pigmentation—Syphilitic maculæ, melasma, chloasma, icterus, ephelis.
 Congenital malformations—Ichthyosis, cutaneous nævus.

Neurosis—Pruritus.

Anomalies of secretion :

Increased or diminished—Seborrhœa, xerodermia.

Hyperidrosis, anidrosis, chromidrosis, &c.

Obstructed—Molluscum, comedo, milium, acne, sudamina.

III. *Diseases of the Skin regarded as the Result of Antecedents.*

(Ætiological Classification.)

- Due to the sun, wind, &c.—“Eczema” solare, and traumatic dermatitis.
- Due to the irritation of friction and sweat—Intertrigo.
- Due to the irritation of scratching—Eczema, scabies, urticaria, and prurigo.
- Due to local applications—Mercury, mustard, arnica, antimony, &c.
- Due to animal parasites—Scabies, prurigo pedicularis, impetigo capitis.
- Due to sporozoa—Paget’s disease of the nipple (?), molluscum (?).
- Due to vegetable parasites (Fungi)—Favus, ringworm, &c.
- Due to bacteria—Seborrhœa sicca corporis.
- Due to pyogenic cocci—Pustular eczema, ecthyma, furunculi, and cutaneous suppuration in general.
- Due to specific bacteria—Tuberculosis, including Lupus—Leprosy—Actinomycosis—Enteric rash, Seborrhœic eczema.
- Due to the unknown contagia of syphilis, scarlatina, morbilli, typhus.
- Due to pneumonia (herpes)—to rheumatism (erythema, peliosis).
- Due to the presence of poisons in the blood—The rashes caused by belladonna, copaiba, shell-fish, &c. ; bromide acne ; hydroa from iodide.
- Due to the poison of uræmia—Various forms of dermatitis (vol. ii, p. 623).
- Due to disturbance of innervation—Zona, sclerodermia (?), Bullous lesions.

IV. *Diseases of the Skin as they specially affect Different Ages.*

- Infancy—Syphilis, nævi, eczema, intertrigo, impetigo capitis.
- Childhood—Scarlatina, morbilli, rubella, erythema, ecthyma, molluscum sebaceum, eczema of face and scalp, ringworm and favus, pernio.
- Adolescence—Lupus, acne, erythema nodosum.
- Early adult life—Erythema, secondary rashes, psoriasis, sycosis, lupus.
- Old age—Rodent ulcer, epithelial cancer, prurigo pedicularis, chronic eczema.

Nomenclature.—The binominal terminology which once extended to the whole of medicine was founded upon a misleading analogy between natural organisms like plants and animals, and heterogeneous objects like diseases. It has, unfortunately, persisted in dermatology, after the genera and species, the classes and orders of Cullen and Sauvage and other compilers of artificial systems of medicine had long been abandoned ; and after being deservedly discredited, has of late years revived, to the detriment of scientific medicine. Next to a false pathology and fruitless attempts at classification, nothing has been more injurious to the rational study of cutaneous diseases than a cumbrous, pedantic, and often barbarous nomenclature.

A good name should have the following characters:—(1) It must consist of a single word. (2) It must be distinctive, and easily recognised both by the eye and the ear. (3) It must be capable of forming an adjective. Less essential points are (4) that it should be short and, if possible, familiar, of Latin or Greek origin, or capable of easy reproduction in the former tongue, and as classical and euphonious as may be. (5) It should be unmeaning, or at least arbitrary and conventional in its application; or, if descriptive, should apply only to some obvious and constant feature of the malady. (6) It must be generally accepted, and, therefore, new names should chiefly be introduced by simplifying or correcting old ones.

Historical sketch.—The fact that many eruptions on the skin are closely attendant upon febrile and other general disorders early attracted notice; and the humoral pathology which pervaded medicine from classical times until almost the present day afforded a ready explanation of their occurrence. Hence cutaneous diseases were long regarded as mere symptoms of some hypothetical “dyscrasia” (*i.e.* ill-mixing) of the four Galenical humours—the blood which was formed by the liver, the phlegm secreted by the pituitary gland, the bile by the gall, and the black bile by the spleen. From due mixture of these humours arose the four natural temperaments: sanguine, in which the blood was predominant; phlegmatic or pituitous; bilious or choleric; atrabilious or melancholic; and from their ill-mixture resulted such “ill-humours” as scurvy, scrofula, gout (*gutta*), and rheumatism (*rheuma*).

When the doctrine of the four humours was given up, cutaneous diseases were ascribed to equally hypothetical “disorders of the blood;” then, when better knowledge of the chemistry and morphology of the blood began to stand in the way of so easy an explanation, they were explained as being due to “diatheses” or tendencies, of which the eruption was at once the evidence and the effect. Explanation by assumed causes still took the place of inquiry into anatomical and clinical facts.

The skin was supposed to be a kind of chart or index to the “state of the blood,” as the tongue to the state of the stomach, and eruptions are still so regarded by popular opinion.

During the eighteenth century the causes and indications for treatment of an eruption were readily determined by learned physicians to be due to a strumous cachexia, or a scorbutic state of the blood, or to vitiated humours from obstruction of the primæ viæ, or to lues venerea, but the actual physical conditions of the skin were scarcely noticed. It was the great merit of Willan, who began to write in 1808—a merit shared in some degree by his predecessor, Plenck, of Vienna (1783)—that he accurately described the *anatomy* of the morbid skin. His “orders,” the *elementary lesions* of later dermatologists, are the alphabet of the subject, and correspond with the “physical signs” of the diseases of the lungs introduced by Auenbrugger and Laennec.

From the English or anatomical school of Willan and Bateman (1813) sprang the French school of dermatology, which may be generally described as *ætiological* in aim. Biett, its founder, was a pupil of Willan, and introduced his system into France (1833). He had the merit of perceiving that syphilis does not merely act along with other predisposing causes in producing diseases of the skin, but that it has, as its direct consequence, definite, constant, and recognisable lesions, the knowledge of which is all-important for diagnosis and for cure. He was succeeded by Cazenave

(1843), Devergie (1854), and other systematic writers, who continued the work of clinical investigation and accurate description. Unfortunately Biett's success in tracing certain cutaneous affections to syphilis led to the formation of similar groups of "scrofulides" and "maladies dartreuses."

The attempt to define cutaneous diseases by their true *nature and cause* instead of by anatomical lesions had been already made by Alibert, a contemporary of Biett. His eloquence and power of picturesque description had much influence, which was increased by the publication of a magnificent atlas of plates, illustrating his *Arbre des Dermatoses* (1814). His pathology, however, was erroneous, his descriptions superficial, his nomenclature inaccurate, capricious, and unclassical. The same attempt to explain rather than to investigate, and to supply the nature and causes of disease by hypothesis when proof is absent, pervades the voluminous writings of Bazin (1853-70), who carried the hypothesis of "diathesis" to its extreme limit. The same principles were illustrated in the interesting lectures of Professor Hardy (1858-64), so long connected with the great hospital of St Louis.*

Meanwhile, another school had risen at Vienna which was guided by the pathological doctrines of Rokitansky. Its founder was Ferdinand Hebra, whose writings have done more than those of any other man to put the study of dermatology on a sound basis and to extend its limits.†

The pathological school of Vienna represented by Hebra, the diathetic school of Paris represented by Hardy, and the anatomical and therapeutical school of England represented by Erasmus Wilson (1847-67), have all changed during the last twenty years. Histological investigation by the improved methods of the last twenty years has thrown much light upon the morbid processes of the skin, and the study of bacteria has revolutionised the pathology of many affections.

In England during the long blank after Willan and Bateman's work, the late Erasmus Wilson alone made valuable contributions to dermatology; but of late a new school has sprung up and has produced such excellent works as those of Dr Liveing and Dr Crocker, besides the numerous important papers of Mr Hutchinson and many younger dermatologists, to whose contributions in the 'British Journal of Dermatology' and elsewhere the writer is greatly indebted.

In France a new generation has sprung up, well versed in German and English literature, accurate, laborious, and scientific; and Besnier, his colleagues and disciples, have restored the credit of French dermatology.

In Germany the labours of Kaposi, Pick, and other disciples of Hebra have borne excellent fruit; while Lassar, Unna, and many others have opened fresh lines of research in histology, pathology, and treatment.

America has made important contributions to dermatology during the last twenty years, and under such leaders as Duhring, White, and Bulkley, has become a worthy rival of the best schools of the old world.

I. THE ANATOMICAL LESIONS.—Before entering upon the description of

* Since these lines were first written, M. Hardy has returned to his dermatological studies, and has published a systematic treatise on the subject.

† His 'Acute Exantheme und Hautkrankheiten' was the third volume of the series of text-books of 'Pathology and Therapeutics' superintended by Virchow, and was published in parts between 1860 and 1874. Much of the latter part is written by Hebra's son-in-law, Moritz Kohn, now better known as Kaposi. The translation into English for the New Sydenham Society (1866-75), begun by Dr Fagge and the present writer, was completed by Mr Waren Tay.

the several diseases to which the skin is subject, it will be well to refer to the elementary lesions, as they have been called, the morbid anatomy which can be seen and felt so as to constitute the solid basis of fact, like the anatomical changes in the heart and lungs which are detected by the hearing.

1. *Hyperæmia* or *Congestion*.—(a) Mere over-fulness of the vessels from paralysis of the vaso-motor nerves with redness and heat, but without the exudation and tissue changes which accompany inflammation. This hyperæmic blush, readily produced in the physiological laboratory, is rarely seen as an uncomplicated morbid condition (*e. g.* Trousseau's *tache cérébrale*, see vol. i, p. 803).

(b) *Active or arterial irritative hyperæmia*, varying in colour from brilliant scarlet to rose pink, and combined with heat, tingling, or other sensations. Such an early stage of inflammation is often called "erythema." The local swelling and the subjective symptoms distinguish it from the non-inflammatory hyperæmia just described.

(c) *Passive or venous congestive hyperæmia*, dependent upon retarded circulation and distended venules. The colour is purple, bluish, or livid, the surface is cold, and there are no painful sensations. This passive congestion, frequently seen as the result of thrombosis, and also in long-standing affections of the heart and lungs, is often associated with the more chronic forms of inflammation in which œdema is present, and connective-tissue overgrowth is apt to result. The best example of this condition in the skin is in the congestive erythema of a chilblain.

2. *Pimple* or *Papule*.—A small, solid elevation of the skin. Under this name more than one pathological lesion is included.

(a) The acute, minute, inflammatory papule, more or less pointed, bright red, and very early seen with a lens to contain a minute drop of exudation. It is either abortive or ends in a vesicle or pustule.

(b) The chronic large inflammatory papule, never showing liquid exudation, but apt to become covered with minute scales. Sometimes, as in psoriasis, these papules increase so as to form a raised patch, and then become covered with scales, and sometimes they coalesce with the same result, as in lichen planus. More often the papules remain discrete and without scales, as in prurigo and some forms of secondary syphilis.

(c) A solid non-inflammatory papule formed by true hypertrophy of the normal papillæ of the cutis. Such papillary growths produce the minute multiple warts which occasionally occur in immense numbers over wide surfaces of the body; large local warts and condylomata are much more common.

(d) Solid elevations of the skin, which may be called false papules; such as the heaped-up scales at the orifice of a hair-sac which form the so-called "lichen or pityriasis pilaris," and a sebaceous gland occluded by its own secretion, which is called a "comedo."

3. *Vesicle*.—A visible cavity in the skin filled with transparent liquid. In almost all cases the vesicle is inflammatory, and the liquid is exuded plasma, consisting of water, salts, albumen, and a few leucocytes with only a trace of fibrinogen. Where the epidermis is thin, the vesicles rupture almost as soon as they form; but where it is thick, as in the palm and sole, they grow and coalesce into large bladders. Broad and flat vesicles, as those of zona, are distinguished from the smaller and more closely-packed vesicles of eczema. The vesicles of smallpox are remarkable not

only for their size and depth, but for the exudation being so effused into the meshes of the papillæ and Malpighian layer that the cavity is "pocketed," and shows a central depression or *umbilicus*.

Non-inflammatory vesicles consist of retained excretion either of sweat-glands (sudamina) or of mucous glands. The latter are almost the only vesicles seen on the mucous membranes, for under the moisture and friction of the mouth, though inflammatory vesicles form, they are scarcely ever seen before they burst.

4. *Pustule*.—A cutaneous abscess, that is, a cavity in the skin containing inflammatory exudations, water, salts, albumen, and abundance of dead leucocytes in a state of fatty degeneration, with usually only traces of fibrinogen. The distinction between a vesicle and a pustule is therefore often one of time only, and rests upon the abundance of the corpuscular element in the exudation; but while most vesicles become pustules, the exudation remains serous in many cases of eczema. Again, in contagious impetigo, in furunculi, and in some other cases, the first visible exudation is already opaque, yellow, and purulent.

5. *Bulla* or *Bleb* is the name given to a very large vesicle. It is, as a rule, inflammatory and of essentially the same pathology as a vesicle or pustule. It contains at first transparent serum, but this usually becomes more or less completely purulent. There are also almost always shreds of fibrin to be seen. The anatomical distinction asserted between ordinary inflammatory bullæ and those of pemphigus will be referred to in the chapter on the latter disease.

6. *Scab* or *Crust*.—A dried-up concretion of the contents of a vesicle, pustule, or bleb. Its form depends upon the inflammatory process ceasing, otherwise fresh exudation succeeds, and no dried-up mass is allowed to form. The size of a scab will always depend upon that of a pustule or bleb which formed it, its thickness upon the amount of fibrin and leucocytes in the exudation. Its colour is often characteristic; light brown or yellow when the exudation is serous, deeper yellow (compared by the elder anatomists to honey, whence the term *Porrigio favosa*), or greenish yellow in some cases when the pus is thick, red or almost black when the exudation contains erythrocytes.

7. *Scale* (*squama*).—A dry flake of epidermic cells. When scales form in moderate amount and of small size as the result of inflammation which has passed, they are described as *furfuraceous*; when large, adherent, imbricated, and glistening silver-white from the refraction of air enclosed in the spaces, scales have the characteristic appearance seen in psoriasis. Large, thin, and very abundant scales, which have been compared with dry hop leaves, and sometimes termed *squames*, are almost characteristic of pityriasis rubra. Beside the true epidermic scales, desquamation often consists of dried-up sebum or of dried exudation mixed with epidermis. The microscope distinguishes the amorphous fatty material of the former and the leucocytes of the latter from the flat horny cells of true scales.

8. *Wheal* (*pomphos*).—A flat solid elevation of the skin much larger than a papule, and of ephemeral duration. Such wheals may be either traumatic, as from the lash of a whip, or idiopathic; they are the characteristic effects of the poison of the stinging-nettle and of one form of erythema, hence called urticaria. They are formed by acute œdema of the skin producing local anæmia from pressure.

9. *Scratch-mark*.—An injury to the skin of linear form and curved out-

line, usually marked by dried-up blood, and having a definite relation to the range of the patient's hands. They are of diagnostic value as proofs of pruritus.

10. *Raw*.—A surface which has lost its horny layer of epidermis so that the moist and living Malpighian layer is exposed, from which more or less exudation oozes. Such a raw weeping surface is characteristically seen after the blister formed by cantharides has been broken. It also results from the rapid rupture of a number of vesicles as in the kind of dermatitis called *Eczema madidans*.

11. *Chap (rima)*.—A crack or fissure which goes through the epidermis to its Malpighian layer or to the vascular papillæ beneath. These rimæ or *rhagades* sometimes extend very deeply, are apt to bleed, and are always extremely painful.

12. *Sore (ulcus)*.—The result of destruction by inflammation, which has reached below the Malpighian layer and has destroyed the papillæ; characterised by the absence of any trace of epidermis, by the granulations which cover its floor and by the pus in which they are bathed.

13. *Scar (cicatrix)*.—The result of the healing process after an injury or disease which has been deep enough to destroy the papillæ of the part. Accordingly the presence of a cicatrix, however superficial and slight, shows that the preceding process reached the deep layer of the cutis.

14. *Nodule*.—A solid elevation of the skin, larger than a papule and seated in its deep layer. The nodule was formerly called a tubercle, but the word "tubercle" should never be applied except with its present pathological meaning. A *node* is a large nodule, and there is no reason for restricting the term to syphilitic nodes or *gummata*.

15. *Stain (macula)*.—A patch of increased pigmentation of the skin, either the result of long-continued hyperæmia or occurring independently as a primary increase of pigment.

16. *Hæmorrhage (ecchymosis)*.—When a blood-vessel of the cutis vera gives way, a dark red or purple mark is produced, which (like the macula) does not disappear on pressure. When small, the punctiform spots resemble flea-bites, and are hence called *petechiæ*; larger extravasations, particularly when elongated in form, resemble the bruises caused by a stick, and are termed *vibices*.

The mode of invasion should be noticed, whether by successive foci appearing and coalescing, or by a spreading *serpiginous* border. The earliest and most characteristic anatomical lesions will generally be found in this advancing edge.

II. DISTRIBUTION.—After determining the morbid anatomy of a disease of the skin, the next step is to notice its distribution.

The various organs of the body have each their own characters in disease, so that inflammation, hæmorrhage, fatty degeneration, and cancer vary not only in their local varieties, but in their local frequency. The same pathological predilection for certain regions is marked in most affections of the skin, and greatly helps in diagnosis. Thus psoriasis of a flexor surface is as rare as phthisis of the base of a lung, and zona of the forearm or leg as rare as cancer of the small intestine. One cause of this difference is the thickness, vascularity, and abundance of hair-sacs or sweat-glands; another is exposure to air, sun, friction, or moisture; but in many cases

no cause can be assigned for the local distribution of the dermatoses any more than for the geographical distribution of animals and plants.

(A) *Distribution in depth*.—In its pathology the skin does not follow the anatomical and embryological division into epidermis and cutis vera. It may be pathologically divided into three layers :

(1) The *horny layer of epidermis* or *cuticle*, dead scales, the only affections of which are increased growth, atrophy, dryness, desquamation, and other results which really depend on perverted growth in the living layer of cells which lies immediately beneath it. Lesions of the cuticle, hair, and nails are, therefore, all secondary lesions.

(2) The *living Malpighian layer* of the epidermis, together with the *papillary layer* of the cutis. These two tissues are constantly and inseparably united in their pathology. A blister does not lift up the epiderm from the derm, but the horny from the Malpighian layer of the epidermis. Inflammation of this combined layer constitutes the enormous group of diseases which come under the head of superficial dermatitis. Affections confined to this part never leave scars.

(3) The *deep layer of cutis* with the *subcutaneous connective tissue*.—Inflammation or new growths beginning below the papillæ seldom spread upwards ; but are prone to spread to the subcutaneous tissue and not to stop until they reach subjacent muscle or bone or deep fascia. The deep affections of the skin which lie in this region are less numerous, but more severe, than those of the superficial layer, and always leave cicatrices when they are cured.

(4) Lastly come the deep cutaneous affections which particularly affect the *sweat-glands*, the *sebaceous glands*, the *hair-sacs*, or the *nail-beds*.

(B) *Distribution over the surface*.—The earliest attempts at classification of skin diseases were between affections of the scalp and of the trunk. But in Willan and Bateman's system this regional distribution did not receive due consideration ; and it has met with still less at the hands of French and German dermatologists. Even in the best descriptions of Hebra and his successors it is sometimes impossible to learn what part of the body is most often affected by a particular disorder. The fact is that very few diseases of the skin are indiscriminate in their extent, while many are at least as definitely and exclusively fixed to certain localities as the lesions of enteric fever in the intestine, or of tabes in the spinal cord. The skin is not uniform in its structure, the relative thickness of its layers, its vascularity, its nervous supply, or the distribution of its glands. Its different parts are variously protected both by natural and artificial coverings, they are variously exposed to injuries, to irritants, and to moisture. It is, therefore, not surprising that their diseases differ so greatly.

It must be remembered that in childhood the several regions of the skin are not yet completely differentiated, and hence the local distribution of its diseases is less strictly adhered to than in adults. We find precisely the same rule in the localisation of pneumonia, of malignant disease, and of tubercle in children.

The principal dermatological regions are :—The hairy scalp, beard, axilla and pubes ; the face ; the orifices of the eyes, mouth, nose and ears ; the front of the neck and chest ; the abdomen and genitals ; the back of the neck, shoulders and loins ; the outer side of the arm and forearm and back of hand ; the bend of the elbow and wrist ; palm of hand and sole of foot ; the buttocks, outer side of thigh and leg, and dorsum of foot ; the inner

side of the thigh and ham ; the fingers and the toes ; the anus, perineum, scrotum or vulva.

We must here stop for a moment to explain the meaning of the word *symmetry* in distribution. Universal eruptions, like that of scarlatina, are in one sense symmetrical ; but only because the human body is itself bilaterally symmetrical. Nor is it enough that the same disease should be found in both right and left members, as is acute rheumatism, to make it symmetrical. Symmetrical distribution means that exactly the corresponding parts on the right and left side are simultaneously affected, both ears, both elbows, the back of each hand, the under surface of each wrist, the popliteal space on each thigh, or the sole of each foot.

Beside bilateral symmetry, we also see examples of serial symmetry in pathology, when the same condition is seen on the elbow and the knee, the wrist and the ankle, the palm and the sole.

Bilateral symmetry is often well marked in dermatology. It is absent in diseases of the brain and eyes, present in many cases of pneumonia and most of phthisis, present in Bright's disease and in Addison's disease of the adrenals, absent in the testes and ovaries.

III. CIRCUMSTANCES.—The third group of common characters of dermatoses includes the natural history and surroundings of a case. Such facts help in diagnosis, they throw light on pathology and causation, and they frequently supply hints for treatment. We have to consider :

1. The *age* and *sex* of the patient. Some affections, like prurigo pedicularis, are scarcely seen except in the aged skin ; others, like impetigo, are extremely common before puberty and extremely rare afterwards ; while true acne begins, with rare exceptions, at the period of puberty. Syphilis is more common in men, and rodent ulcer and epithelioma ; lupus erythematosus is more common in women, and xanthelasma and scleroderma.

2. *Condition of other organs*, and particularly the state of the stomach and bowels, the urine, and the temperature.

3. *Occupation*, especially when parts of the skin are exposed to cold, to great heat, to wet, or to chemical or mechanical irritation.

4. *Intercourse* with other persons affected with a similar disease.

5. *Clothing*.—Some cutaneous maladies are caused by the friction of under-garments, or by their being saturated with sweat, or being coloured with deleterious dyes, or conveying pediculi or streptococci.

6. *Cleanliness*.—Phthiriasis and tinea versicolor are the result of dirt, and the same is true of some forms of eczema, intertrigo, and acne. But as a rule it is remarkable how little local mischief is done by uncleanly habits and neglect of the skin, and how often soap and water irritate it.

7. *Climate*, season, temperature, moisture or dryness of the air, sun, frost, and wind. Many diseases of the skin, as of other organs, which were formerly supposed to be endemic, are not really so ; but some are peculiar to hot countries, and others, notably leprosy, have, within historical times, become more restricted in their range. Exotic diseases will be only briefly described or merely mentioned, and the reader referred to accounts by those who write from personal observation.

8. *History* of the malady, of its duration, and the manner of its onset ; particularly, when obtainable, a knowledge of the primary lesion. The fact of recurrence is also of great importance. Patients often forget previous attacks.

9. *Subjective symptoms*, as pain or discomfort, itching, burning, smarting, tenderness, or neuralgic pains, alleviation by exposure to the air, or by covering, by heat or cold, by the application of water or oil, by pressure or by friction.

10. *Concomitant symptoms*: Pyrexia, insomnia, jaundice, albuminuria, and the varied signs of past or present syphilitic or tuberculous disease.

11. Lastly, the effect of previous treatment, whether positive or negative, is sometimes a great help, not only in avoiding therapeutical errors, but in deciding doubtful diagnoses.

Diseases of the skin should be arranged (as they should be named) like diseases of other organs, *i. e.* for convenience, either alphabetically or otherwise.* The order followed in the present volume is to begin with the most common affections, the superficial forms of dermatitis, eczema and its allies, psoriasis and its allies, erythema and its allies. Then will follow affections of the hair-sacs and cutaneous glands, and the tineæ. Next comes a chapter on the deep inflammations and the hypertrophic conditions which result therefrom. Closely allied to the deep chronic forms of dermatitis are the important and well-defined bacterial diseases known as lupus and leprosy, and the chapter which treats of these two subjects is naturally followed by one on tumours and new growths. Then comes a short section on abnormalities of the cutaneous pigment and of cutaneous innervation, and the subject concludes with a chapter on the practical diagnosis of diseases of the skin in general.

* The attempt to form a natural or scientific classification of diseases is in the writer's judgment a mistake; and nowhere more impossible and injurious than in this department of medicine (cf. 'Guy's Hosp. Reports' for 1877, series iii, vol. xxii, p. 151). In America Dr Duhring has given the great weight of his judgment in the opposite direction, but neither do his arguments appear to be conclusive, nor has the classification he proposes (a modification of Hebra's) been more generally accepted than its predecessors.

ECZEMA*

AND COMMON SUPERFICIAL DERMATITIS

“Her wrinkled skin, as rough as maple rind,
So scabby was, that 'twould have loathed all womankind.”

SPENSER.

Definition—Willan's—Hebra's—Its distinction from other forms of dermatitis—Histology—Anatomical lesions—Distribution and local varieties—General symptoms and course—Ætiology—Diagnosis—Prognosis—General treatment—Local applications—Diet and regimen, baths, etc.—Internal remedies—Special treatment of local varieties.

Impetigo—Its relation to eczema—to pediculi—to contagion—Its treatment.

The most important of diseases of the skin, from its frequency, its obstinacy and the misery it occasions, is the affection now universally known as eczema (ἐκζέμα), the “outbreak” or “eruption,” as the Greek physicians called it.* In its commonest form it is familiar to the profession and the public, and cannot escape instant recognition, but under many circumstances it is difficult to diagnose, and opinions have differed widely as to its limits, its pathology, its definition, and the extent to which dermatoses bearing other names are allied to or identical with it.

Definition.—Willan classed eczema among *vesicular* diseases, and this is a proof of his acumen and judgment; for, although the vesicles of eczema are so small, so short-lived and so speedily supplanted by weeping surfaces or scales, that one may see hundreds of cases before the vesicular stage can be demonstrated, yet there is no doubt that vesicles are the most characteristic and, if not constant, form the most nearly constant anatomical lesion of eczema.

Hebra first stated that eczema can be produced at will, that it is in fact identical with *common superficial dermatitis* from ordinary irritants. As a result of this important statement, Hebra not only described under eczema much of erythema, intertrigo, the pustular form of dermatitis

* *Synonyms.*—Moist Tetter—Common superficial dermatitis.—*Fr.* Eczème.—*Germ.* Ekzem.—*Die nässende Flechte.*

† The word is used as of common application by Dioscorides in his ‘*Materia Medica.*’

known as impetigo, and most cases of papular dermatitis previously classed under various species of lichen and strophulus, but he boldly included scabies itself as also a common inflammation of the skin and therefore a true eczema. Most dermatologists have more or less followed Hebra in extending the bounds of eczema far beyond the definitions of Willan and Bateman.

But pathologically true as was Hebra's doctrine, it has become clear that for clinical purposes we must seek again to narrow the definition of the word eczema. Inflammation, the reaction of the living tissues to injury, is the key-note of pathology. If to the doctrine of inflammation we add that of degeneration and new growths, of parasites and of contagia, almost the whole range of modern pathology is covered. It is quite true that the vast majority of diseases of the skin, like those of the rest of the body, are inflammatory, but for prognosis and cure we need much more than this elementary fact. Inflammation is not one and the same thing. In other words, the irritants or causes of inflammation vary greatly, and the reaction of different tissues to the same irritant vary also. Acute inflammation due to purely chemical or thermal or mechanical irritation is one thing—that due to streptococcus pyogenes or other suppurative microbes is another. The tubercle bacillus produces its own form of tissue growth, that of leprosy quite another. Hence we must expect to find dermatitis in many different forms.

Hebra himself had too much sagacity and practical sense to be led far astray by his own reform.

(1) Syphilitic diseases are most of them undoubtedly inflammations of the skin, but however closely they may approach in symptoms and appearances to some forms of eczema, they are fundamentally different in origin, prognosis, and treatment. Scarlatina is a dermatitis not unlike some stages of eczema. Variola and varicella often approach impetigo still more closely in appearance, but they are clearly separated by our knowledge of their ætiology, by their combination with definite symptoms in other organs than the skin, and by their course.

(2) Scabies, again, is distinguished from all other forms of dermatitis, not by the pathological process, but by the peculiarity of the irritating agent, by the consequent characteristic distribution, and by the special mode of treatment.

(3) The superficial dermatitis of Erythema, of exanthems and of toxic origin are manifestly distinct in origin and course, and with these must go Erysipelas and Zona.

(4) The part of vegetable parasites in exerting superficial dermatitis is not yet fully known. Very little inflammation is caused by the fungi of ringworm and only one so-called Eczema is due to this cause (*E. marginatum*). Pyogenic bacteria complicate eczema as we shall see (p. 847), and micrococci are probably the specific cause of some forms of seborrhœic dermatitis.

(5) Lastly, we must separate from the true eczema diseases like psoriasis, which, though undoubtedly inflammatory, are special in their characters, in their anatomy, in their chemical products, in their results, and (above all) which cannot be produced or even simulated by an external irritation. In other words, they are not "common superficial dermatitis," such as results from the natural reaction of the healthy skin against a common mechanical, thermal, or chemical irritant.

But now comes a more fundamental definition of the term, which is absolutely necessary for those practical objects which are the end and justification of all refinements of nomenclature. If we call eczema common superficial dermatitis, and assert with Hebra that we can produce eczema at will by rubbing in an irritant ointment or by exposure to the sun, we run the danger of forgetting what is, after all, a most important character of the disease which we agree to call eczema, whatever else may be included under the name. Undoubtedly "wet tetter" is in the majority of cases *not* the direct and immediate result of local irritation. It is therefore preferable to say that a scorching sun or a mustard plaster will often produce a common superficial weeping dermatitis which is histologically and chemically identical with eczema, which may, if we please, be called artificial or traumatic eczema, but which yet differs from the true disease by the very fact that it is the physiological reaction of the healthy skin to a definite known irritant; that it further differs in its course, in its distribution, in its whole natural history, from idiopathic* or primary eczema, and demands as a consequence a different prognosis and different therapeutics. The distinction, however subtle in theory or difficult to draw in practice, is of real importance.

For instance, the surgeon of a gaol is shown an eruption on a prisoner's arm, which, by every anatomical character, is a "common superficial dermatitis"—is what might have been made by Hebra's eczema-producing liniment. He diagnoses eczema, prophesies the course it will take, its obstinacy, and its probable recurrence, and prescribes what he calls appropriate treatment. But whether verbally correct or not, he has made as great a practical blunder as is possible. The common superficial dermatitis is traumatic; the eczema is not *like* that produced by an irritant, but was actually and designedly so produced; the subject of it is not a patient with a disease, but a skilful impostor who has inflicted injury upon his skin; the course of the eruption will not be guided by the natural history of eczema, but by the will of the patient; it will not recur except by his wish, and will not be cured by "appropriate treatment." Since, therefore, the name which follows a diagnosis should connote as much knowledge as possible in brief, it is much better not to name factitious dermatitis "eczema."

In the same way we should exclude all common superficial dermatitis which is the direct and immediate result of local irritation, for what is remarkable is not that the skin should inflame when irritated, but that the skin of many people is liable to undergo the exact pathological changes produced by irritation *without* any demonstrable irritant.

Eczema, therefore, may be defined as "idiopathic or primary, common, superficial dermatitis." We must, however, fully admit the difficulty or impossibility of drawing a line in every case. We can only classify diseases as they more or less naturally are connected with certain typical forms. At one end of the scale we have purely traumatic dermatitis produced by a demonstrable external irritant, limited to its immediate effects and disappearing not to return when the cause is once removed; at the other end we have dermatitis appearing on parts of the skin which are not exposed to any known irritation, following a distribution which is independent of irritants, recurring without external cause after it has once disappeared, and only curable

* It must be remembered that idiopathic is by most German writers defined so as to include traumatic causes. In this country we usually call a condition idiopathic when it is not caused by injury or any other known condition. No one of course supposes that any disease is without a cause, but for the present a cause undiscovered is no cause at all.

by measures other than those addressed to the local irritation. But in every case of dermatitis, however idiopathic, there is no doubt an *irritans*, if we could only recognise it, and in every case, however traumatic, there is an *irritabile* in the patient's tissues. Inflammation can never be truly "idiopathic," that is, uncaused; for like every other event it depends upon antecedents. No fire, no cantharides or croton oil can produce a pustule or a bleb upon the skin of a corpse. All eczema is common superficial dermatitis; but every common superficial dermatitis has not the characters in its origin, its distribution, and its course, in fact, in its whole natural history, which entitles it to the name of eczema. In order practically to identify eczema we must, therefore, look for the clinical characters to be presently described.

Eczema, again, is dermatitis at the stage of *exudation*: it is well called "moist tetter." Cases of dry eczema no doubt occur, but they are either abortive or residual. When we use the term eczema we imply that the eruption is moist, or will be moist, or has been moist; or that at least it occurs in a person who has previously been, or will hereafter be, subject to another outbreak of the same thing, when exudation will be apparent.

Slight degrees of inflammation, when the result of irritants, fall under the minor degrees of superficial traumatic dermatitis. Slight degrees of idiopathic inflammation which do not reach the stage of exudation—hyperæmia, roseola, erythema of the skin—when not shown to be abortive eczema by their locality and course, belong to very different pathological groups. They may be, first, symptomatic rashes like those of measles and scarlatina, which are true dermatitis with all the characters of inflammation, and followed by desquamation; and under the same head of exanthemata should be also included the roseolar, erythematous, or papular rashes of enterica, cholera, and syphilis. Again, there are superficial forms of dermatitis which differ from eczema in locality, in their local and general symptoms, and in their constantly subacute character. These superficial dermatites, of which erythema nodosum is perhaps the best type, are clinically and pathologically to be separated from eczema, and will be treated of in a separate chapter.

Anatomy.—The pathology of eczema is that of inflammation generally. Its signs are the four Galenical characters of pain, heat, redness, and swelling, to which we now add a fifth, pyrexia or febrile reaction. Of its cause we know no more than of inflammation in other parts. Traumatic inflammation follows injury or local death of a tissue; idiopathic inflammation we assume must follow some corresponding lesion, but of its nature we are ignorant. The order of events is vaso-motor paralysis, dilatation of the small arteries and capillaries, stagnation of the blood-stream, diapedesis of leucocytes through the stomata of the capillaries, and exudation of the plasma or liquor sanguinis.

If a section of eczematous skin be made, the cuticle or horny epidermis is found unaffected, the Malpighian layer swollen, the papillæ œdematous, with dilated blood-vessels and multitudes of leucocytes clustered round them; while the deep layer of the cutis and the subcutaneous tissues are unaffected. Looking at the living skin we see, so soon as a sense of slight irritation with some pain of a tingling or smarting character has drawn the patient's attention to the spot, that there is already an inflammatory blush. This usually has from the beginning a brighter, more arterial hue than the

rose-coloured tint of true erythema. A more important distinction is that the eczematous blush is diffused and fades off at the edge; while it is scarcely ever disposed in blotches, circumscribed or mottled patches, or figures of definite outline. The swelling from oedema is very slight. On close inspection, particularly if a lens be used, one can see that the apparently uniform redness is produced by a number of isolated deeper-coloured points. In this and in other respects the early stage of eczema resembles scarlatina as true erythema resembles measles.

Before long, but never without a precedent stage of hyperæmia, there appear minute vesicles. Frequently, however, they are preceded by little red elevations, which for some time show no bright transparent spot of fluid, and these inflammatory papules may appear early and continue for a long time before becoming vesicular. Such papular forms of eczema must be regarded as abortive, and very seldom will a careful scrutiny fail to discover liquid exudation at one period or another of the case.

Soon after the vesicles have formed, the remarkably thin roof of the cuticle ruptures and they run together, forming a raw weeping surface, *eczema madidans*; or they may previously have acquired more or less purulent contents before their thicker roof bursts. Such pustular forms of eczema usually produce, not weeping surfaces, but more or less extensive scabs, though intermediate stages are very frequent. In the most typical form of eczema the weeping stage continues until a great abundance of clear watery exudation is poured out. The liquid consists chiefly of serum, to which the salines give its irritating property and the albumen its characteristic effect in stiffening linen. On the raw weeping surface it is easy to distinguish more injected points which mark the seat of ruptured vesicles. This *état ponctué*, as the French writers call it, is very characteristic, and may be sometimes seen before and even after the moist stage.

The involution of eczema is accomplished by the exudation diminishing, and at last drying up; the weeping ceases, or scabs take the place of pustules. Finally, the cuticle again covers the abraded surface and a branny desquamation, formerly described as *psoriasis diffusa* and also as *eczema squamosum*, covers the lately inflamed parts. The itching still continues and is sometimes troublesome up to the very last.

In chronic eczema the skin becomes decidedly thickened, a result which is readily appreciated on pinching up a fold. It is constantly covered with branny desquamation, acquires a deep red instead of a bright scarlet colour, and in certain parts is marked by deep fissures or rhagades, which often penetrate to the true skin and give rise to bleeding and excessive pain. This *eczema rimosum* is most frequent in the palms or the soles, and in its hæmorrhagic form on the nipples and the lips.

Distribution.—One of the distinctions between eczema or idiopathic dermatitis and that which is traumatic in origin is that, while the latter corresponds more or less exactly to the irritant, typical eczema has its own peculiar laws of local distribution. Speaking generally, it is a disease of the thinner parts of the skin, of the flexures of the joints, and of the head and the limbs rather than of the trunk.

Eczema, as above defined, is an extremely symmetrical disease, more so than any other affection of the skin excepting psoriasis and scabies.

Head.—The most characteristic locality for eczema is behind each *auricle*, not only because it is so frequently seen here when it affects other

parts, but also because this spot is but little liable to other diseases. The *face* is in children the favourite seat of the pustular form of eczema (which we shall presently describe as *impetigo*). The rough skin of slight eczema and the weeping of the serous form is less common, but often seen on the lips, nostrils, and cheeks. In adults the face is still oftener the seat of ordinary eczema, coming next in frequency to the limbs. On the *scalp*, *impetigo* is the commonest affection in childhood, but ordinary eczema is comparatively rare except on the bald scalp of infancy or age. Eczema does not frequently affect the skin which is covered by the *beard*, and when it does is not usually remarkable for obstinacy; but sometimes the inflammation can be unmistakably seen to penetrate the hair-sacs and there become a deep instead of a superficial dermatitis. Its clinical features and treatment are then so different that it is properly known as a separate disease, non-parasitic *sycosis*. It may in like manner spread to the sacs of the *eyelashes* and become localised as what used to be called *tinea tarsi*.

Trunk.—The *neck* is very frequently the seat of eczema, especially the front and sides. The shoulders, back, and loins are but rarely affected; and the same statement applies to the gluteal region, so frequently the seat of isolated pustules, not only from scabies, but in the *impetigo* and *ecthyma* of children, and from suppuration of the hair-sacs in this region. The flanks, though covered with soft and delicate skin, are not often affected with eczema, which when present has usually spread from the axillæ or from the abdomen; and the same applies to the chest. In women, however, eczema of the *breast* is common, either as *eczema intertrigo* beginning in the fold under the mammæ, or as true eczema of the nipple. The *abdomen* is more often the seat of eczema than other parts of the trunk, especially of that variety which begins at the *navel*.

The *genital organs* by the thinness of the skin are readily disposed to dermatitis, but are certainly less often affected with ordinary eczema than either the face or the limbs. Sometimes the inflammation begins as *intertrigo* (chafing) of the scrotum and thigh, especially common in infants, or it is an acute weeping eczema which extends to the abdomen, thighs, and other parts as well, or it is a chronic and extremely pruriginous eczema of the vulva or scrotum. The last form even more frequently affects the neighbourhood of the *anus*, particularly in elderly persons; and the cleft of the nates is extremely liable to eczema *intertrigo*, particularly in fat persons, under the irritation of riding or of long walking and free perspiration. In many cases eczema of the anus, perinæum, genital organs, and thighs forms a well-marked local variety, which must be carefully distinguished from the so-called "*eczema marginatum*" of the same regions, to be afterwards described as a form of *tinea*.

Limbs.—On the *arms*, eczema is scarcely ever seen over the deltoid muscle, and though common in the axilla, particularly its anterior fold, is much less so than on the elbow. The bend of the elbow is probably, next to the face and ears, the most frequent seat of ordinary eczema, and if we were to exclude cases occurring under puberty even that exception need not be made. The skin covering the biceps cubiti and the flexors of the front of the forearm usually participates in eczema of the elbow, and this local form of eczema is one of the most constantly and accurately symmetrical.

A relatively uncommon form of eczema occupies the external aspect of the arms. It is a dry, circumscribed, seborrhœic eczema, is more often seen in adults than in children, and in men than in women; and affects the

outer side of both forearms from an inch or more below the point of the elbow down to the wrist. It is extremely symmetrical, and often affects the skin of the upper arm which covers the triceps, though without spreading to either shoulder or elbow. It will be again mentioned under *seborrhœa corporis*.

The back of the hand is seldom the seat of eczema, though the affection will sometimes spread from the arm as far as the knuckles, and this region is sometimes the seat of the dry chronic circumscribed dermatitis which will be described as "single-patch eczema."

The *fingers*, especially their clefts, are often the seat of eczema, but in most cases this can be traced to a local cause of irritation. The thick skin of the *palm* seems little adapted to eczematous inflammation, but it is frequently the seat of a characteristic chronic dermatitis, painful, disabling, symmetrical, and obstinate; from the absence of vesicles and the presence of deep fissures this local variety has received the name of *eczema rimosum*. This is, however, either of traumatic origin, or at least unassociated with eczema of other parts, and is curable by local applications alone. Eczema of the matrix of the *nails* is almost always part of eczema manuum. It proves a long period of dermatitis, and its presence is, therefore, a point of diagnosis in distinguishing eczema from scabies. The consequent malformation of the nail is generally marked by transverse grooves, and by less thickening than in the far more rare psoriasis unguium.

In the *lower extremities* eczema of the groin and inner part of the thigh is very common in adults, and is often associated with the same condition of the axilla or of the abdomen and genitals. The outer side of the thigh is not often the seat of eczema, and the patella, like the olecranon, is practically exempt; but the popliteal space is almost as favourite a seat of the disease as the bend of the elbow, and the inflammation spreads thence more or less extensively over the thighs and legs.

Below the knee eczema is on the whole less frequent, and most often appears in one of three forms. (1) Eczema of the calf and peroneal region of an ordinary weeping and irritable type is now and then seen, affecting both legs symmetrically. (2) Eczema of the same region is less frequently seborrhœic: dry, circumscribed, marginate, with yellowish tint and branny desquamation. (3) Varicose "eczema," a local dermatitis often unconnected with eczema of other parts, is obviously the result of ordinary irritation acting upon a skin congested by varicose veins. It affects the inner side of the leg from the internal malleolus upward, that is to say, from the point where, as Hilton showed, the last considerable anastomosis takes place between the superficial saphenous and the deep posterior tibial veins. This form of dermatitis is known by its purplish tint and frequent association with ulcers as well as by its locality.

The *foot* is less often affected with eczema than the hand, but follows its serial homologue very closely. When not of traumatic origin and not due to scabies, that is to say, when dermatitis of the foot is true eczema, it either affects the dorsum in association with eczema of the outer side of the leg, or the sole as a chronic eczema rimosum much resembling that of the hand, or it is an eczema-intertrigo digitorum sometimes leading to deep clefts between the toes. This last form is as common as eczema of the fingers, if not more so. It is rarely associated with the disease in other localities, and must be treated entirely by topical measures.

Chronic eczema of the soles sometimes assumes the characters of hyper-

trophic dermatitis, with accumulation of horny epithelium a quarter or even half an inch thick, very painful, and of a sickening caseous odour. This is quite distinct from the somewhat similar condition occasionally produced by syphilis.

Symptoms and natural history.—Eczema provokes extreme itching, more perhaps than any other cutaneous disease except prurigo and scabies. Indeed, although we see some cases of eczema which are almost free from irritation, yet in others the pruritus seems to be at least as intense, constant, and obstinate a symptom as in the worst cases of those diseases. Itching is usually less in the weeping than in the dry, papular, or chronic and scaly conditions, and it is rare in the pustular form of eczema. It is most intense in ordinary eczema of children, and in that of old persons, and of all local varieties is most constant and most severe in eczema ani et vulvæ.

Smarting, tingling, and some amount of local tenderness are common symptoms of the more acute and ordinary forms of eczema, and are associated with a peculiar sense of burning and tension. There is slight febrile movement at the onset of the disease and particularly when large surfaces are invaded at once; but even when the thermometer shows no appreciable elevation of temperature, there is thirst, loss of appetite, and a slightly furred tongue. The mucous membranes are unaffected; there is no foundation for such names as eczematous gastritis, enteritis or bronchitis. The pathology of the digestive, pulmonary, and urinary mucous tracts is quite different from that of the skin, and we have no right to assume an eczema of membranes which we cannot see, when we are unable to demonstrate eczema of those which we can. The writer has never seen eczema of the lips spread to the mouth or tongue, eczema of the anus to the rectum, eczema of the eyelids to the conjunctiva, or eczema of the penis to the urethra and bladder.

Except for anorexia produced by the slight pyrexia at the onset of eczema, the appetite and digestion are, as a rule, unaffected in this disease, nor are the bowels either constipated or relaxed. Dyspepsia is so common that many persons suffering from eczema are also dyspeptic, but there is no reason to regard the latter condition as the result of the former, or *vice versâ*. One would beforehand expect the urine to be affected when large surfaces of the skin are inflamed, but there is no satisfactory evidence of this being the case. The fact that in the large proportion of cases the patients are not confined to bed, or even to the house, makes it difficult to obtain observations of the amount or average condition of the urine; but as far as we know at present, the urine is unaffected by eczema.

Course.—Eczema is very rarely acute in origin or development; even in what appear to be the most acute cases it will usually be found that the patient has been subject to previous attacks, and that in the intervals small patches of the disease linger behind the ear or on the face or hands or some other isolated part. And if an acute origin is rare, a subsidence by crisis is unknown. After a more or less gradual subsidence, fresh smaller outbreaks occur again and again, so that with scarcely an exception, however acutely the attack of eczema may appear to begin, its subsequent course is chronic.

Another peculiarity also very characteristic of eczema is its strong tendency to recurrence. It is extremely rare for a person to suffer from a

single attack in the course of his life. Again and again when the disease appears to be in a quiescent condition a fresh outbreak will occur, or one attack will scarcely have passed off when another supervenes. Happily the majority of cases are not lifelong in duration, but they sometimes extend over several years, and it is not unusual for recurrence to take place even after long intervals of complete freedom.

Ætiology.—We have already stated what appears to be the true relation between traumatic dermatitis and idiopathic eczema. When the inflammation directly follows an irritant, is not prolonged after its cessation, does not spread to other than the irritated region, does not recur without fresh irritation, and does not follow the local distribution of eczema, then it is best called common superficial dermatitis of traumatic origin. But some skins, whether by natural stability or by habit, are insensible to sun and friction and sweat, and the other irritants which in ordinary persons produce inflammation. In others a hot day, or bathing in sea-water, or an east wind, or a long walk, will produce “eczema solare” or “eczema intertrigo” or some other form of local traumatic dermatitis—which when once established becomes chronic, persists long after its original cause has ceased to act, and localises itself in the bends of the joints, and in the symmetrical positions which have been above described as characteristic of eczema. Admitting, therefore, local irritation of various kinds as an exciting cause of eczema, we must also admit a certain proneness of the skin to inflammation, and in probably half our cases this predisposition causes the disease without our being able to fix upon any adequate irritant.

It has been widely supposed that we are to seek the predisposing, and in most cases the efficient cause of eczema in a diathesis or disposition of the whole body, which can be recognised independently of the presence of the eczema, and which produces other recognisable diseases. This diathesis has been called dartrous, arthritic, and gouty, while the ever-ready assumption of a strumous disposition has been invoked when the other failed. The reader is referred to the works of Bazin and Hardy, of Gigot-Suard, and other French writers, for their exposition of the dartrous doctrine; while all that can be said for the more particularly gouty relations of eczema will be found in the writings of Erasmus Wilson, Hutchinson, and the late Dr Tilbury Fox. It is only right to add that Besnier and most of the other leaders of French dermatology have more or less completely abandoned the doctrines above mentioned. The fact is that the doctrine of temperaments and diatheses is a mere residuum of the exploded physiology of Galen. Although persons with gout are often subject to very irritable and obstinate eczema, in the vast majority of cases of eczema there is no reason for the belief that gout, that is, the deposit of uric acid in the joints, has been present in the patient or his immediate relatives; there is no pathological connection between gout and true rheumatism, arthritis deformans, or gonorrhœal arthritis, and none of the latter forms of multiple arthritis have any demonstrable connection with eczema; eczema rarely co-exists with psoriasis, pemphigus, or other supposed manifestations of the dartrous diathesis; no one can give an intelligible account of the characters by which this predisposition can be recognised; there is no evidence that eczema has more than accidental connection with diseases of other parts of the body, or that it is anything but a common superficial

dermatitis; and lastly, the diathetic hypothesis is practically misleading in prognosis and treatment, no less than scientifically unsound.*

The efficient cause of eczema, apart from local irritation, is as much and as little unknown as the efficient cause of bronchitis or of cystitis. All we can say is that in some persons the skin is naturally sensitive, or delicate, or irritable. Such persons are, in other respects, like their neighbours, and the predisposition of their skin to inflammation can only be prophesied after the event.

General eczema is sometimes set up by an accidental irritation, and this is probably the explanation of the undoubted though rare occurrence of vaccination leading to eczema: much more frequently their relation is purely accidental. It appears to be waste of time to discuss the vague speculations, at once unscientific and unpractical, which ascribe eczema to such common disorders as dyspepsia, or to such *idola theatri* as constitutional predisposition, assimilative debility, nervous debility, perverted innervation, renal inadequacy, strumous cachexia, scurvy in the blood, or acidity of the *primæ viæ*. There are, however, some forms of superficial dermatitis usually classed as eczema which modern research enables us to separate from the primary and the traumatic cases.

Pustular eczema.—In the pustular forms of eczema, strepto- and staphylococci are found with the exuded leucocytes, and not infrequently we can see that the change from serous exudation to suppuration is due to accidental inoculation with pyogenic cocci.

Whether the most common forms of papular, vesicular, and weeping eczema are of bacterial origin is very doubtful, though Unna believes he has shown it to be so.

Seborrhæic eczema.—There is, however, one kind of dry eczema, with circumscribed margin and chronic course, with comparatively little irritation and without pyrexia, which this eminent dermatologist has shown to be often connected with a seborrhæic state of the head ("pityriasis capitis"), and a corresponding affection of the sebaceous glands of the trunk and limbs. It usually spreads from the scalp to the face, and thence to the shoulders and back, or sometimes to the chest, and on the limbs takes the outer aspect by preference. This kind of dermatitis is certainly of bacterial origin, and the recognition of the fact marks an important step in the theory and practice of dermatology.

Among the secondary or "traumatic" kinds of eczema, or, as we prefer to put it, of common dermatitis, the following ætiological varieties are of practical importance.

E. solare—or rather, *E. a calore*—which is not only confined to the exposed face or arms, but may affect covered parts by spreading or by heat, just as similar dermatitis may be produced by the heat of forges, smelting-rooms, bakehouses, or by sitting in front of the fire.

E. a luce complicates eczema from heat, but also occurs from mere exposure to light—as to the reflection from a river or a calm sea, when there is no direct sunshine; or exposure to white snowfields beneath an overcast sky. I have seen a vagabond Hungarian deeply "sunburnt" in winter when no sunshine had appeared for weeks. On this subject Dr Robert Bowes' observations and experiments are most important ('Journal of Der-

* The writer may be allowed to refer to papers on this subject in the 'British and Foreign Medico-Chirurgical Review' for January, 1874, and in the 'Guy's Hospital Reports' for 1880.

matology,' vol. v, p. 237). The dermatitis caused by the X-rays is now a frequent example of this kind of "eczema."

E. a sudore has been already referred to as a local variety of dermatitis of irritative origin.

E. ab unguibus is a term which denotes the important fact that many forms of so-called eczema are really dermatitis set up by scratching and rubbing to relieve itching. This applies to the dermatitis of *Prurigo e pediculis*, to the eczema (and secondary infective impetigo) of the scalp from pediculi, to the dermatitis of the anus from pruritus ani, and to much of the "prurigo," "strophulus," and "urticaria" of early childhood, which depend upon their scratching to relieve the irritation of fleas and other parasites.

Whether in most of these cases there is no primary local lesion, but only disordered sensation, is difficult to prove or to disprove. Pruritus of cerebral or peripheral nervous origin is possibly the primary condition which leads to scratching and so to local traumatic eczema in old age; and it may be connected with degenerative changes in the senile skin.

There is no doubt that eczema is in some cases *hereditary*, but it is certainly much less so than psoriasis, and in the great majority of cases there is no reason to admit hereditary predisposition.

The exudation of eczema is not contagious so long as it is transparent. When purulent it shares in the infective characters of pyogenic microbes, and the pustular dermatitis which we shall presently describe as impetigo capitis is markedly contagious.

Extent, &c.—Eczema affects both *sexes* indifferently. It is common at all *ages*, but differs in its most frequent characters. In the infant it is of the ordinary weeping and vesicular kind. The same form is seen in older children, but much more frequent in them is impetigo, or pustular eczema, which is comparatively rare before the first dentition and after puberty. In adults the commonest form is ordinary weeping eczema of the limbs and face. In old age, the dry, very chronic, and extremely pruriginous forms are the most frequent and characteristic.

Eczema appears to be universal over the globe. It is certainly not more frequent where true gout is prevalent, as in London, than where it is rare, as in Vienna and New York.

The traumatic forms of eczema naturally occur in those *occupations* where the hands are exposed to constant irritation. Hence arises the eczema of the hands which has long been recognised as frequent in washerwomen, grocers, and other hand-workers.

It is a popular opinion that skin diseases generally, and particularly eczema, as the most common of them, are most prone to occur at certain seasons—the spring and fall. Like most popular beliefs, this was not founded upon experience, but chiefly upon theory, and partly perhaps upon analogy. The period of change in the seasons seems "naturally" to be the most likely period for change in the human economy, and changes are proverbially dangerous. It is possible also that the insalubrity of southern Europe in the autumn, from the prevalence of malaria, led to a belief in the same insalubrity in northern countries, while even in England malaria was far from uncommon until quite recent times. However this may be, neither autumn nor winter are particularly eczematous seasons, and if one meets with eczema of the ordinary irritative and inflammatory kind

more often in the spring than at other times, this may be fairly attributed to the dry east winds which then prevail.

Diagnosis.—Keeping to the definition of eczema as above stated, the only difficulty is on the one hand to distinguish between idiopathic and traumatic dermatitis, or rather to detect the decided and efficient prevalence of a traumatic cause; and secondly, to draw the line between eczema and certain other forms of superficial dermatitis, the distinctive characters of which, and the justification for their separation, will be considered under each head. The distinction between eczema and intertrigo, eczema and impetigo capitis, and even eczema and scabies, depends on their ætiology and the prognosis and treatment which follow when this is ascertained. The dry, scaly eczema associated with seborrhœa capitis above mentioned is a form to be recognised for the same reason, as are varicose dermatitis and the traumatic dermatitis set up by the patient's own scratching. There remains eczema in the restricted sense of the term characterised by being *common*, that is to say, the same as is produced by ordinary mechanical or chemical irritants, *idiopathic*, that is to say, not directly co-extensive with irritation, *moist* from visible inflammatory exudation, *symmetrical*, *selecting* certain favourite parts of the skin, and *prone to recur* after disappearance.

From *scarlatina* and other rashes, eczema differs in being never truly universal, in its moisture, and in being unaccompanied by marked pyrexia. From *erysipelas* it is also distinguished by the colour, the minute vesicles, the locality, and the absence of a defined margin and of œdema.* *Erythema* is more rosy in tint, and though it may form papules or bullæ, never shows the small vesicles or the weeping surface of eczema: moreover, its distribution is different, and it is never chronic in its course. Eczema has no resemblance to *psoriasis* except in very old cases of the latter disease, when the scales have disappeared and the original locality is obscured. *Syphilitic* dermatitis is multiform, and does not itch.

Prognosis.—Eczema is almost always amenable to treatment, that is to say, we scarcely ever see a case in which no improvement can be produced, and still more rarely one which finally resists all therapeutical measures. Moreover, it is scarcely ever dangerous to life.

There are, however, exceptions to each of these statements. In the outbreak of acute vesicular and weeping eczema, whether primary, or, as far more often happens, occurring in the course of a chronic or nearly cured attack, we can do little or nothing to stop its violence. Abortive treatment is unfortunately rarely successful in any acute disease. Again, in some cases eczema, though treated until very little remains, cannot be driven entirely away, but remains in a quiescent state here and there, to burst forth again after a longer or shorter interval.

Lastly, though it is remarkable how little the general health is affected even by very extensive, troublesome, and long-continued eczema, yet occasionally, in infants, or in aged persons, broken rest and loss of appetite cause wasting and muscular weakness, which may at last end fatally. The only cases of the kind which have occurred in the writer's practice were, first, an infant which became emaciated, pale, and unable to take the breast, and secondly, an old gentleman considerably over seventy, who after being much relieved by constant tepid baths and other treatment from an almost

* An exception occasionally occurs when eczema affects the eyelids.

universal and extremely irritable eczema, sank rapidly and died without albuminuria or other evidence of organic disease. On the other hand, everyone must have seen scores of infants who appeared worn to a skeleton and almost moribund through severe eczema, but who nevertheless, under persevering treatment, completely recovered. Extensive eczema in a person over seventy, especially if complicated with gout or with chronic Bright's disease, should always suggest a guarded prognosis.

Treatment.—In the first place it is our duty to treat and, if possible, to cure every case of eczema as quickly as we can. The supposed danger of driving in eczematous eruptions upon internal organs appears to be without any foundation. This doctrine arose partly from theoretical views of the sympathy of organs, partly from the well-known fact that cutaneous hyperæmia diminishes or disappears during acute febrile affections, partly from observing the benefit of counter-irritation of the skin in synovitis or bronchitis, and possibly, as Hebra taught, from the difficulty of curing some cases. In the eczema of children one may again and again observe that as soon as the cutaneous exudation is checked and the irritation subsides, their general health begins to mend. The only caution needful is to be very careful to ascertain the condition of the heart, the arteries, and the kidneys in aged persons suffering from eczema, lest the treatment or cure of their cutaneous disease should be credited with the fatal result which is really the consequence of degenerated viscera.

Prophylaxis and general protective treatment.—The irritants which excite or keep up and renew eczema are chiefly mechanical, thermal, and contact with water. Of mechanical irritants the most important are rough clothing, friction against adjacent parts of skin (*intertrigo*), prolonged contact with decomposing sweat, and also with various chemical irritants which are incidental to certain trades. But the most difficult irritation to get rid of is that which is the result of the disease itself. Eczema always itches, and itching is sure to produce scratching. Hence our first attempt is to prevent this, by persuasion of an adult patient, by muffling the hands of infants, and by such local applications as will at least relieve the intolerable irritation.

With the same object we forbid the application of either cold or heat, the latter for its immediate, and the former for its consecutive effects on the circulation of the part. Contact with air is, in many cases, a decided stimulant, and one important use of the various ointments with which the eczematous skin is smeared is to protect it from the air. With weeping eczema we obtain the same end by covering it with wetted rags or muslin wrapping, or by dusting it with absorbent powders.

Even more important as a cause of irritation is moisture. It is not that mere contact with water is an irritant; probably a faintly alkaline or very weak saline solution with a little colloid material, such as gum, size, or oatmeal added, is the least irritating medium with which an inflamed and excoriated skin can come in contact. A continuous bath, even of ordinary water, is a most useful and perfectly safe means of treatment in some cases of very general eczema, with profuse exudation and great irritability. To Hebra is due the merit of proving that patients can be kept continuously in a bath of suitable temperature, not only for hours, but for days—indeed, for an indefinite period, without leaving it for any purpose whatever. The writer saw this plan carried out at Vienna, and has more than once adopted

it himself. The practical difficulties are obvious, and it is fortunately not often that we need resort to it.

But though *continuous* contact with water is by no means irritating, bathing and washing mean *intermittent* wetting of the skin. The change from dry to wet, from higher to lower temperature, and the reverse change on withdrawing the eczematous surface from the bath, the necessary friction of the towel, the saline constituents of most waters, and most of all, the evaporation, which even great care cannot entirely prevent, and which, after careless washing, goes on abundantly from the half-dried surface—these form altogether a most efficient series of irritations.

It is even possible that the frequent and systematic cleansing of the skin from dead epidermis and from sebaceous secretion, which is the result of the artificial condition of extreme cleanliness to which modern civilised society more and more tends, may itself render the skin more susceptible to slight irritants, and certainly with tender skins the use of soaps, of nail brushes, of rough towels, and of flesh gloves, may sometimes aid in exciting dermatitis—a small set off against the advantage to general health of mind and body to which a clean and active skin undoubtedly conduces.* Important rules of treatment in eczema, therefore, are that the inflamed parts must not be washed with either hot or very cold water, must not be washed frequently, and must be very carefully dried after washing with soft, dry, and warm towels. In order to prevent the “chapping” of the hands, which is so common in children during the winter, it is important to take care that they are thoroughly dried on towels which are not already damp. In large schools for poor children, a good plan is to make them dry their hands by dipping and rubbing them in a tub full of bran, instead of upon towels, which are sure to be wet for all but the first comers. They should also be given olive oil, or any other neutral fatty compound, to rub into the hands after washing, when there is the least appearance of dermatitis. With severe and chronic eczema rimosum of the hands it is necessary absolutely to forbid washing, and to protect from contact with air or moisture by ointment and a well-fitting kid glove. The best plan in chronic eczema is to advise that once a week, or more frequently as may be thought safe, a complete warm bath and thorough cleansing of the whole surface should be used, taking care to keep the skin immersed from the time of its being first wetted, to dry it thoroughly when the washing is over, and to use inunction afterwards with olive oil, vaseline, or benzoated lard. Washing, with unscented, pure yellow soap and lukewarm water, if done seldom and followed by careful drying and inunction, is less injurious to an eczematous skin than more frequent and careless ablution with no soap at all. For the exceedingly irritable skin of the face and hands it is sometimes desirable, however, absolutely to forbid all contact with water, and cleansing can then be accomplished by friction with dry and stale bread crumbs, or by wiping with lint dipped in olive oil. When eczema affects the scalp, the best cleansing agent is yolk of an egg diluted with water.

In obstinate cases it is necessary to forbid washing altogether, and to keep the eczematous surface constantly covered with ointment. Any scales, crusts, or accidental impurities must be removed with oil and cotton-wool.

Poultices are almost always injurious, and scarcely less so is the modified form which follows the application of water dressing, a piece of lint

* See an amusing article by Hebra on the dangerous consequences of being overmuch clean, translated in the ‘London Medical Record,’ March 15th, 1877.

dipped in water or lotion, and closely covered with oiled silk or gutta percha. The impermeable covering soon raises the temperature, and the result is the combined warmth and moisture of a poultice—most valuable for relaxing tension, promoting suppuration, and relieving deep-seated inflammation, but most injurious in superficial dermatitis.

Eczema, even in its acute form is not benefited by the direct abstraction of heat as with ice or by cold baths; and intermittent application of cold proves worse than useless from the reaction which ensues. Eczema in the majority of cases which come before us has passed its acute or sub-acute stage, and irritation rather than heat is the common symptom. It is, however, always well for patients with eczema to avoid the heat of the sun or exposure to fires or to the heated atmosphere of crowded rooms. The affected parts should not be covered with thick woollen garments, and the patient should be lightly covered at night; the bedroom should be well ventilated, the temperature kept somewhat low, and much relief is experienced by keeping the feet or arms uncovered except with a thin rag dipped in lead lotion or powdered with zinc oxide.

Local medicinal treatment.—We next come to the treatment of eczema by chemical applications. Our object is, first, to diminish the hyperæmia and exudation by *astringents*; secondly, to diminish irritability and to prevent scratching by *sedatives*; thirdly, to substitute for a chronic and interminable process of inflammation a more directly traumatic, acute, and self-limiting process, or else, it may be, by less stimulus to produce an effect short of this but serving to quicken the natural process of physiological repair. Applications which have this effect have been called *stimulants* or *alteratives*.

The most powerful chemical astringent which can conveniently be used is probably lead. (See a paper by Dr Payne in the 'St Thomas's Hospital Reports' for 1878.) Salts of copper, zinc, and iron, nitrate of silver, boracic acid and borax are also efficient astringents. So are gallic acid, tannin, and similar vegetable preparations, though these are less applicable to the skin than to mucous membranes.

As local sedatives we may use belladonna, opium, chloroform, hydrocyanic acid, but these are generally unsuitable to eczema on account of its extent, and raw, denuded surface, both which characters make absorption too probable for these narcotics to be safe. More efficient as remedies against itching, and free from any but local action, are preparations of zinc, which combine antiphlogistic and antipruriginous qualities. Dilute solutions of carbolic acid, 2 per cent. in water, 1 in 20 in oil, are very useful. Weak tarry preparations are also efficacious, especially in the drier forms of eczema; as diluted oil of cade (juniper tar) or liquor carbonis picis with vaseline in the proportion of a drachm or half a drachm to an ounce, or in the proportion of ℥xx to an ounce of camphor or chloroform water.

For chronic and no longer very irritable eczema more stimulant applications are necessary; sometimes stronger tarry preparations, unguentum picis liquidæ or pyrogallic acid. These are most useful in limited patches of scaly and very chronic dermatitis with much thickening of the skin; those, in fact, which approach most nearly in appearance and pathology to psoriasis. The colour and smell are objectionable, but the staining of both skin and clothes by chrysarobin ointment is much more unpleasant. Ichthyol, resorcin, and similar local remedies are sometimes indicated, particu-

larly in dry and irritable cases ; but ichthyol is a disagreeable application ; and Dr Crocker finds thiol, which has no smell, to be equally useful.

A still more energetic method, introduced by Dr Anderson, of Glasgow, is painting the eczematous surface with liquor potassæ. This must be done with much caution, for it gives rise to considerable pain, though in many cases this is less complained of than might be expected ; but the writer can bear witness to the efficacy and safety of the treatment when applied tentatively on limited surfaces of old and obstinate eczema, especially of the dry kind. With moist secreting surfaces of unusual obstinacy one finds more useful the application of a solution of nitrate of silver varying from a scruple to as much as a drachm to the ounce. It must be occasionally painted on, not kept in constant contact, and often proves most efficient as an astringent and a sedative, as well as an alterative.

With the same object a twenty per cent. solution of salicylic acid in ether, has been used with good effect, painted on the affected part and allowed to dry.

But often there is too much active inflammation for us to venture on such treatment, and more generally applicable alteratives are the various preparations of mercury, corrosive sublimate in solution, white precipitate ointment, red oxide ointment, and dilute nitrate of mercury ointment. Mercury in some form is particularly adapted to pustular forms of eczema, and is seldom suitable to those which profusely secrete serum. Lotio nigra is an old remedy, recently revived with success by Dr White, of Boston.

Most often, however, the cases of eczema which come before us combine the characters of irritation, itching, and chronicity, so that for perhaps the majority of cases, at least if we include suppurative forms, there is no more useful preparation than such a combination of zinc, lead, and mercury as forms the unguentum metallorum of the Guy's Pharmacopœia.* This may be varied by substituting the red oxide for the nitrate of mercury ointment, and by varying the proportion of the three constituents ; often again lead and zinc act better without mercury, or the carbonate better than the alkaline acetate of lead. Another variation which sometimes gives better results is the combination of the oleates of lead, zinc, and mercury.

In cases of seborrhœic eczema especially, sulphur is a very useful application, but the sulphur ointment of the Pharmacopœia should be diluted for this purpose.

Whatever be the chemical application used, it is important to decide whether the vehicle should be watery or oleaginous. An excellent general rule was that of the late Dr Hughes Bennett, of Edinburgh ; for dry affections of the skin, use ointments ; for moist, use lotions. If an ointment is applied to a profusely secreting eczema the drug and its vehicle are washed away by free exudation and never reach the subjacent skin. Lotions, on the other hand, have but little power of penetrating the epidermis, and if carefully watched will be seen to run from the surface, which is greasy by its natural sebaceous secretion. With raw surfaces which do not secrete profusely, either lotions or ointments may be appropriately used. Practical considerations teach us that lotions are better suited to diseases of exposed parts like the face and hands, that they are readily applied to young children, that they are more efficiently used by persons confined to bed or by

* R. Ung. zinci, Ung. plumbi acet., Ung. hyd. nitr., ãã partes æquales : misce.

women living indoors than by those who are engaged in active work, that they are more cleanly and more pleasant to most people, but that they also give more trouble and demand more time in their application; and lastly, that in the summer, when the skin is frequently covered with sweat, they are particularly grateful and efficient. We must remember that lotions should in most cases be used with exposed skin or with the surface only covered by a thin rag into which the lotion has soaked. If applied in the morning and covered up till night they speedily become water dressings, and probably in less than an hour mere applications of wet rags with no further therapeutical power.

On the whole, therefore, notwithstanding the rule quoted above and the fact that eczema is pre-eminently a moist tetter, it will be found that with the majority of our out-patients, whether private or at the hospital, ointments are practically the more eligible vehicle. It is important to make sure that the lard or other oleaginous material is not in the least rancid, and that it is free from salt. The addition of benzoic acid as ordered in the British Pharmacopœia makes as good a vehicle in most cases as can be wished. The mineral oils have the advantage of not decomposing, and for some reason ointments made up of vaseline (soft paraffin) suit certain cases of eczema better than those prepared with animal fats.

A method of applying remedies, such as zinc, lead, and tarry preparations, in an oily vehicle, introduced by Unna, consists in strips of muslin soaked in a mixture of suet, wax, and paraffin as the basis for any mineral we desire to apply. These *Salben-mulle* are closely applied to any part affected, and insure continuous action of the drug employed.

A somewhat similar plan is to impregnate caoutchouc with drugs, so as to form a closely adherent and impervious application to the eczematous skin. This has, however, the disadvantages of all impervious plasters, that the part often becomes too hot and moist.

Unmedicated oily applications, vaseline, cold cream, olive oil, have in themselves the good effect of protecting from air and of softening rough, harsh skin, inspissated sebum and dried secretions. Lanolin is better fitted for psoriasis and other affections in which it is desired to rub the oily vehicle thoroughly into the skin; and it mixes with water as other oily preparations do not. Glycerine, from its strong affinity with water, is well known to be a direct stimulus to a nerve-trunk. It is, except when very dilute, a decided irritant in eczema, and has far from the same soothing effects as cold cream or zinc ointment, in cases of intertrigo, chilblains, and eczema solare. In very small quantities, however, it may be added to lotions with the view of preventing evaporation. Glycerine of starch is a much more soothing preparation, and, thickly smeared over the surface, it often suits acute weeping eczema very well.

Weak alkaline lotions have often been recommended to relieve the burning pain and irritation of acute eczema, and they were extensively used by Professor Hardy at St Louis. But in the very cases of acute weeping eczema in children to which such treatment seems applicable, the parts are so excessively tender that even a 1 per cent. solution of bicarbonate of soda is ill borne, so that in such cases the lead lotion (liq. plumbi subacetatis dilutus of the British Pharmacopœia) is more useful. At all events, if soda is used at all it should be in quantities only just sufficient to react to test paper, and the water in which it is dissolved should also contain a little oatmeal or size, and should be tepid or warm.

A weak solution of Resorcin (a phenol-derivative soluble in water and in oil) is often better borne, and proves more beneficial than alkaline lotions: one, one and a half up to two per cent. is the safe strength to use.

Quite apart from the ordinary use of a lotion, the whole object of which is to keep the part continually wet, is that of a solution which, when painted on, is allowed to dry. For this purpose nitrate of silver or other strong astringent solutions may be used; or zinc or lead rubbed up with oil may be painted over a dry, irritable, eczematous surface so as to exclude the air.

Another useful method is to suspend insoluble powders like oxide of zinc, starch, or sulphate of lime or bismuth in water by help of a little mucilage or tragacanth, without, however, attempting to form a perfect emulsion. The milky liquid is applied freely with a large camel-hair brush or sponge, and is allowed to dry over the weeping surface, and in some cases of irritable and profusely secreting eczema, as also in pemphigus, this is found to be the most effective application. Care must be taken not to have too much of the colloid ingredient, or hard cakes are apt to form which crack and become painful. In fact, chalk or gypsum shaken up with water and applied like whitewash is sometimes the simplest and pleasantest method.

Lastly, we may apply our remedies directly as dry powders. In this way oxide of zinc, chalk, bismuth, calamine, and other fine insoluble powders may be used, and such applications are usually better than starch. They dry up discharges, protect from the air, and are often the best applications in cases of intertrigo. On the other hand, they are unsuitable for pustular eczema, where they would form massive and troublesome crusts.

It must, however, be admitted that nothing but experience, tact, and previous knowledge of particular cases will guide one aright in the selection either of appropriate astringents, of the strength of the application, or of the kind of vehicle. Some patients assure one (and prove right again and again) that they cannot bear any kind of ointment. With others all lotions are apt to produce pustules, or even boils. Not infrequently, especially in the acute stage of eczema, an inert powder or unmedicated vaseline, according as the surface is moist or dry, will do more good than anything else. In all cases we should remember that the ointments are not to be rubbed in, but gently smeared on the skin, and afterwards kept in continual contact by well-adjusted soft linen bandages; that the lotions should never be allowed to get hot, and must be frequently renewed; and that the strength of our applications should be small in the acuter stages, and greater as the case becomes inveterate.

The writer believes that the majority of cases of eczema can be cured by well-directed local measures of the kind above indicated; but it must be admitted that Hebra undervalued the treatment by internal measures, which undoubtedly holds an important though a secondary place.

Diet.—In the acuter stages of eczema the patient should be put on almost fever diet, and should be encouraged to drink freely of any cooling beverage. He should take no stimulants or meat, and eat sparingly, chiefly of bread, milky dishes, green vegetables, and ripe or stewed fruit.

In ordinary chronic eczema no such strict diet is necessary. It is, however, usual to forbid certain articles of food, and the experience of patients shows that, at least in some persons, one or all of these really aggravate the disease, chiefly perhaps by producing thirst, increased heat of the skin, and more scratching. The kinds of food referred to are salt meats of all kinds, including ham and cured fish, curries, cheese, pepper, spices, and

other hot condiments. The stronger wines and malt liquors are also usually forbidden, but although in the necessarily generalised treatment of hospital out-patients this is doubtless good advice, there does not appear to be any evidence that the moderate use of ale or wine with food does harm in eczema or any other affection of the skin—except in cases where, independently of dermatitis, even moderate stimulants provoke symptoms of gout or dyspepsia, with flushing of the face. In many patients, especially those in middle and later life, wine or beer with the principal meal of the day helps digestion, and certainly does no harm to the eczema, while a little spirit and water at bedtime will favour sleep, and in that respect prove a useful adjunct to other treatment. Sometimes, however, even weak whisky and water produces heat and discomfort after retiring to bed, and must then of course be interdicted. In almost all cases a somewhat free supply of unstimulating diluents should be taken between meals, and a glass of water while dressing of a morning, and again the last thing at night, is almost always useful.

There is no good reason, theoretical or empirical, for a patient with eczema to abstain from sugar; but there is every reason for him to abstain from hot soup or hot tea. He should also dress lightly, and lie lightly covered at night, and avoid hot rooms and proximity to the fire.

Air, places, and water.—In chronic and obstinate diseases like eczema, patients frequently ask whether change of air would do them good. They are usually recommended to go into the country if they live in town, or go to the seaside if they live in the country, or, if they can afford it, to visit Scotland or Switzerland, or some more distant resort. The only point on which one may speak with confidence as to the effects of air and climate upon eczema is that, just as it is aggravated by the east winds of an English spring, so it is more difficult to cure in the eastern counties, and is often favourably influenced by removal to the moist and soft air of the western Highlands, of Devonshire, or of Ireland. As one sees in many other diseases, it is the change which does the good, and this is most apparent when the change is from an unfavourable climate. Secondly, there is no doubt that in many cases of the more irritable forms of eczema sea-air proves a decided irritant. It is only now and then, in chronic non-pruriginous eczema or in the impetigo of childhood, that sea-air and even sea-bathing do good instead of harm.

With respect to baths generally we have already sufficiently insisted upon the evil effects of frequent contact with water; but there is no doubt that in the very chronic and intractable forms of eczema saline and sulphurous baths act beneficially, probably like the stimulant and alterative applications above described. When the period has arrived for their use it is difficult to say, and each case must be judged by the tact and experience of the physician. A single bath may bring back in all its virulence an eczema which had nearly disappeared. Long-standing dryness, thickening of the skin, and absence of excessive irritability are the features which should generally weigh with us in advising or permitting this mode of treatment. The baths best adapted for the purpose are perhaps those of Harrogate.

Internal treatment.—Lastly, we come to the treatment of eczema by drugs. While we could better dispense with this group of remedies than with the others, we should often fail for want of them, or the success of our treatment would at least be less rapid and complete.

In the acute stage of eczema with profuse exudation and much irritation, it is the practice of the French school to purge freely, and most English physicians adopt the same plan, though perhaps less systematically. Saline laxatives appear to be the most useful in these cases. The old-fashioned white mixture of sulphate and carbonate of magnesia taken three times a day, or the pleasanter combination of Epsom salts with carbonate of soda in peppermint or cinnamon water, are useful and popular medicines. A seidlitz powder or a dose of Rochelle salts or Carlsbad salts every morning is suitable for less acute cases. Often it is sufficient for the patient to take a daily morning draught of Püllna, Friedrichshall, Apenta, or Hungarian bitter water. Of these three Friedrichshall is, perhaps, most often suitable, particularly when the eczema occurs in a gouty subject. Sometimes, however, it is less efficient than a seidlitz powder, and occasionally it produces much griping without satisfactory result. In such cases it may be changed for the Hunyadi Janos with advantage; and some find that this Hungarian bitter water agrees better with women than with men. Whichever form of laxative is selected, it should be taken with a draught of warm water early in the morning and on an empty stomach. Such a dose should give one or two loose motions after breakfast without griping or subsequent irritability, whereas even larger doses, if undiluted with water or taken with the stomach already full, are more slowly absorbed and produce more frequent and less effectual irritation. In cases of eczema in which the patient has other independent evidence of gout, it is well to combine with moderate laxatives the exhibition of a pill containing colchicum and aloes or rhubarb, every or every other night. In persons who have lived freely and who are subject to hepatic dyspepsia, beside restricted diet both in food and drink, and moderate laxatives, it is important to prescribe small doses of mercury, either a single grain of blue pill with a little nux vomica and rhubarb before dinner, or two, three, or four grains with an equal quantity of the compound rhubarb pill every other night or twice a week.

We have seen above how important a point it is to relieve the itching of eczema, not only for the comfort of the patient, but to secure the physiological rest which the night should bring to all inflammatory processes, and also to save him from the serious aggravation of his disease which scratching and rubbing the eczematous parts infallibly cause. Moreover, it is at night (even during sleep) that the irritation, increased by the warmth of the bed, reaches its maximum, and that the self-control of the patient is weakened or abolished. Beside the various measures above mentioned for securing coolness, protection from the air, and such help as local sedatives can give, it is often necessary to call in the aid of internal narcotics. Of these opium and its preparations should generally be avoided. Unless given in large doses they are apt to increase rather than to quell the irritation of the surface; they also check secretion and bind up the bowels. It is therefore better to prescribe chloral hydrate or bromide of potassium, or the two together.

Chloral should be avoided with old people, and with patients who may have disease of the heart or atheromatous arteries. On the other hand, it is well adapted to young children, and we have found syrup of chloral the most harmless and useful sedative in cases of infantile eczema. The safest plan is to give a moderate dose when the child is put to bed, and repeat it towards midnight, and, if necessary, again towards morning. Fifteen or twenty drops (about two or three grains) may be given with

perfect safety to a child of six months old; half a drachm twice or even thrice repeated in the night may, if necessary, be given to a child of twelve or eighteen months; and after infancy, say from two to five or six years old, half a drachm, or for older children a drachm of the syrup may be given at bedtime with safety. Again and again this treatment has been followed by the best results. In the first place the child gets rest, and in the morning is ready for food, and all its organs have profited by the natural refreshment of sleep. Next, the skin has been free from fresh irritation, and instead of being marked with the little sufferer's nails, is paler and less angry than before. All the processes of repair have had opportunity to go on; the habit of pruritus is broken for the time, and the nervous apparatus concerned has escaped from a vicious circle of irritation, itching, scratching, and increased irritation.

The bromides are unsuitable to infants from their bulk and disagreeable saline taste, but with older children five or ten grains of the bromide of potassium may be sometimes added with advantage to the chloral draught if suitably covered with syrup of lemon or orange. With adults nocturnal irritation is not usually so severe as with children, and a draught of bromide of potassium or ammonium, with or without the addition of chloral hydrate, is usually sufficient when a sedative is required. Fifteen or twenty grains of ammonium bromide, with ten of the potassium salt, and twenty drops of aromatic spirits of ammonia in an ounce of camphor- or chloroform-water, forms an effectual and not unpleasant sleeping draught.

In some cases, especially in old persons, neither bromide, nor chloral, nor a combination of them acts well. Henbane may then be prescribed with advantage, but in doses of not less than a drachm of the tincture, either alone or with a little compound tincture of chloroform in peppermint water, or the tincture of henbane may be combined with that of hop. Indeed, in old persons where the irritation is not severe, and the want of sleep is rather dependent on general conditions of their age than upon pain or pruritus, two drachms of tinctura lupuli in camphor water may take the place of the whisky and water which the ascetic habits of the patient may render distasteful, or which habits of the opposite kind may render too agreeable. In the happily rare cases of severe and intractable pruriginous eczema in aged persons we are sometimes compelled to resort to repeated doses of belladonna or cannabis indica, or to chloroform inhalations, as the only means of obtaining rest. Such patients will sometimes get needful sleep in a warm bath.

The exhibition of antimony has been recommended in the acuter forms of eczema, and in the writer's experience has sometimes proved useful by subduing vascular excitement and hastening the passage of the acute into the chronic stage of the disease.

Women affected with eczema are often very anæmic, and then steel must be added to the laxative medicine. There is no better combination for this purpose than that of sulphate of iron, given in doses gradually increased from two to five or even ten grains, with half a drachm or more of sulphate of magnesia, five or ten drops of dilute sulphuric acid in peppermint, cinnamon, or chloroform water. Along with laxatives it is usual in cases of chronic eczema to prescribe acetate of potash and other diuretics. Their action is somewhat uncertain, more so than digitalis, squill, or the resin of copaiba; but salines, and especially those of potash, have other actions beside that upon the kidneys, and in ordinary cases of eczema with much

secretion and extensive inflammation, citrate of potash or acetate of potash is often found more beneficial as well as more agreeable than the alkaline carbonates.

In many cases of eczema, especially in children, the patient is thin and pale, with a poor appetite, and a frequent and feeble pulse. It is in these cases that a little wine or malt liquor is not only admissible but often extremely useful; and here the exhibition of iron finds its proper place. For children the syrup of the phosphate, the saccharine carbonate, or the citrate of iron and quinine, are all valuable remedies. For infants steel wine is also a popular and valuable remedy, but after eight or ten years old effectual doses are too large to be convenient. In cases of great anæmia in children, especially where there is diarrhœa, no preparation of iron is so useful as the tinct. ferri perchlor., guarded, if necessary, by twice the number of drops of glycerine, or its taste concealed by a little syrup. It would almost seem as if the astringent quality of the drug had an effect upon the profuse secretion of the eczema. The result, at all events, is often striking as well as beneficial.

With the exception of iron, the group of so-called tonics are not generally indicated in the treatment of eczema. Quinine, however, has a very distinct effect, particularly in the case of infants, children, and persons below adult age, in preventing itching. Half a grain of sulphate of quinine may be given for this purpose to a child a year old an hour before bedtime, a grain if a year older, and as much as five grains to a boy or girl of fifteen. This effect of quinine was well known to Dr Fagge, and it has been independently and strongly recommended by Dr Eustace Smith in his work on 'Disease in Children.' The latter writer also recommends guaiacum in the treatment of eczema, especially where there is reason to suspect a disposition to gout.

There remains a drug which, in England especially, has been very largely and often far too indiscriminately used in the treatment of eczema, as of other diseases of the skin, namely, arsenic. It is undoubtedly a therapeutical agent of the utmost value in psoriasis, in pemphigus, and in certain other cutaneous diseases to be afterwards described, and no one of experience can doubt its efficacy in certain cases of chronic deforming arthritis, neuralgia, idiopathic anæmia, leuchæmia, and anæmia lymphatica; but, like all powerful medicines, it is powerful for evil as well as for good. In the acute stages of eczema, in most cases where there is extensive and active inflammation, and in most cases accompanied by severe pruritus, arsenic is decidedly injurious. In other cases, however, its success is so marked, that, in spite of its frequent failures, it has never lost a certain reputation in the treatment of eczema.

The first indication for the exhibition of arsenic is that the eczema must be in a chronic condition—the greatest benefit is obtained in cases which have persisted for years. Secondly, the more dry and scaly the surface, the more infiltrated and indurated the skin, the less there is of active inflammation and irritation, and the less disturbance of the stomach and intestines, the more likely is arsenic to be beneficial. As a rule, children with eczema do not need it, but some of the most striking instances of its value are in very obstinate and long-continued cases in young patients.

A boy of fourteen had been subject from five years old to what, by his own and his mother's testimony, was really uninterrupted eczema, spreading from time to time with excessive violence from its favourite seats over almost the whole body, but never absent from the scalp, the ears, and the limbs. When he was taken into the hospital there was dry scaly eczema of the head, face, and neck, and the hair was very thin. There was

eczema rimosum of the ears and axillæ, papular dermatitis of the arms and back, and *eczema rubrum madidans* of the abdomen, genitals, perinæum, nates, and thighs. The only parts of the whole surface free from the disease were the palms, the soles, and the shoulder. He was thin, worn, and miserable, and the whole skin was so deeply pigmented that he looked like a mulatto; but the urine was perfectly healthy, and he had no other disease than this severe dermatitis. He was carefully treated with zinc and lead ointment and unguentum metallorum as he had been before while an out-patient; but he was also given arsenic in steadily increasing doses, from three drops up to fifteen three times a day. Under this treatment the inflammation gradually subsided, and at the end of five weeks it was reduced to a little ordinary *eczema* of the arms. This also gradually disappeared. Meantime he had become a stout, healthy-looking lad. He has from time to time appeared again with slight return of *eczema*, chiefly in the scalp and arms; but it has never in the least approached its former severity, and the skin generally, instead of being thick, rough, hard, and infiltrated, with almost entire absence of subcutaneous fat, is now smooth, soft, plump, and elastic, while his head is covered with a thick growth of hair.

In prescribing arsenic the following rules will be found useful:—To begin with a small dose, and gradually but steadily increase it until either obvious benefit results, or the physiological action of the drug is shown by itching of the eyes or slight nausea. When these occur the arsenic should be at once stopped, and then resumed in somewhat smaller proportion, and if necessary again cautiously increased. Secondly, it should always be given either with, or immediately after food, and sufficiently diluted with water. There appears to be no advantage in any other form of the drug over Fowler's solution. The arseniate of soda may be given in somewhat larger doses, but is probably converted into the same form during digestion. The liq. arsenici hydrochloricus is useful if we wish to combine it with perchloride of iron. The "Asiatic pills" of Vienna are in every respect less eligible; and there does not seem to be sufficient advantage in substituting subcutaneous or intra-muscular injection of solution of arsenic, to make up for the pain, the discomfort, and the expense of this method. Lately, cacodylate of sodium has been introduced, administered by this method or by the mouth; and it certainly avoids the irritation of other arsenical preparations. Indeed, such large doses can be taken without producing toxic symptoms that one cannot but doubt whether some of the therapeutical value of the drug is not also diminished.

Troublesome and difficult to treat as many cases of *eczema* are, sometimes rebellious to the very treatment which in apparently similar cases has proved effectual, and always liable to relapses which are most trying both to patient and physician, it is nevertheless very rare for us to fail in at least relieving the miseries of an attack, and in a great majority of cases we may be fairly said to cure a disease which, without skilled treatment, would linger on almost indefinitely. Hebra concludes one of the most masterly and original chapters in his great work by saying that he who having once decided upon his plan of treatment, follows it out with patience and determination, will attain his object sooner than he who often changes the measures that he uses. We may venture to add that while keeping steadily in view the broad principles of treatment based upon rational pathology and tested by experience, the most successful practitioner will be he who knows how to vary their application in accordance with the perpetually varying needs of each individual patient.

There remain certain practical points in the treatment of local varieties of *eczema* which must be briefly mentioned.

Eczema of the ears is one of the commonest local forms of *eczema*, and

is sometimes extremely troublesome. Ointments will be found almost always to suit better than lotions—lead, zinc, or equal parts of the two, or in some cases weak carbolic oil, 1 in 40. When extremely moist, powders suspended in thin gum are better than dry powders, which are almost sure to form thick crusts and produce bleeding.

Chronic *eczema of the meatus* may cause deafness by swelling or the accumulation of its products. This must be treated by syringing with soap and water and, if necessary, application of an alkaline wash followed by unguentum plumbi or unguentum metallorum made soft by an equal part of carbolic oil.

Eczema of the scalp is complicated by the presence of hair and of sebaceous secretion. It often depends on primary seborrhœa capitis, and is apt to be infected by staphylococci or streptococci, and thus become pustular. The hair should always be kept short, but shaving is unnecessary. Unguentum metallorum is commonly a good application. In the drier form of eczema of the scalp with scarcely any exudation, which is often combined with seborrhœa sicca under the name of pityriasis capitis, tarry applications are most efficient, and none is better than liquor picis carbonis, either diluted to form a lotion, or, as we have found better, with vaseline in the proportion of a drachm or half a drachm to the ounce. Impetigo capitis will be presently considered separately.

Eczema of the eyelids and adjacent parts is apt to cause considerable inflammatory œdema, which resembles erysipelas; but the colour, undefined edge, and the absence of marked febrile symptoms, together with the almost certain presence of ordinary eczema in other parts, distinguish the two. It is best treated by mild lead, zinc, or boracic ointments.

Eczema of the lips is sometimes confined to that part and has then a peculiar aspect, there being very little serous or purulent secretion, great swelling, deep cracks, thin scabs, and considerable hæmorrhage. In chronic cases, large thin scales, partly epithelial and partly dry secretion, are formed, which have led to its being called psoriasis labialis. The difficulty is to keep the parts from movement. Very mild ointments, vaseline or lanolin with zinc, or yellow oxide of mercury, or honey with borax will be found useful. Deep and painful fissures should be touched with nitrate of silver either in strong solution or (what is less painful) with a pointed pencil.

Eczema of the palms is usually bilateral and confined to these parts, or it may persist here after it has disappeared from the rest of the body. As above mentioned, it is often directly dependent upon irritants. Having made sure that the case is not one of syphilis, the first and essential point of treatment is to protect the hand from contact with all other irritants, and especially with soap and water. For this purpose scabs, scales, and crusts should be carefully removed with sweet oil, or if necessary by poulticing. The cleansed surface should then be anointed with unguentum metallorum, and thin rags covered with the same ointment should be closely applied to each affected part. A thin kid glove should be worn over the whole, and the dressings should be changed night and morning only. At the end of a week the improvement will generally be striking, or if not it will be due to some neglect of the patient in uncovering his hands or in washing them. If the parts are very irritable it is better to use diluted white precipitate or yellow oxide ointment, or occasionally unmedicated vaseline will be most effectual of all. In chronic indolent cases, on the other hand, a little of the red oxide ointment will often stimulate

most usefully. If there is great accumulation of epidermis it must be removed with soft soap, and then Hebra's diachylon ointment be applied. Deep and painful fissures should be touched at once with lunar caustic.

Chronic *eczema of the sole* is not nearly so common. It must be carefully distinguished from a syphilodermia of those parts by the greater pain, deeper fissures, and more exclusive range. Sometimes it is accompanied by enormous hypertrophy of the epidermis, with deep bleeding cracks and horrible odour. Such cases may be cured by the application of salicylic acid in an ointment (3ss ad ʒj) until the horny masses are removed, and then assiduous treatment with the *empl. plumbi*.

Eczema of the matrix of the nail is still more local than eczema of the palms. It is comparatively rare as a complication of ordinary eczema, and a precisely similar inflammation of the matrix of several nails is sometimes seen where there is no other evidence of its eczematous character. The ill-formed nail may be scraped, but its removal is unnecessary and only possible under deep narcosis. The grooves around it should be carefully anointed with some form of mercurial ointment.

Eczema of the mamma is not infrequent as a local variety of intertrigo. It begins in the lower part of the breast, where it comes in contact with the adjacent skin, and is most common in stout women with pendulous breasts. The surface is raw, weeping, and irritable. The treatment suitable is extreme cleanliness and the application of drying powders, such as fuller's-earth, bismuth or zinc, with separation of the inflamed surfaces by a bandage supporting the breast. So-called eczema of the nipple is a separate disease (*v. p.* 846).

Eczema of the anus, perinæum, and genitals is sometimes confined to the immediate neighbourhood of the rectum. This eczema ani, the prurigo podicis of Willan, the lichen podicis of Hardy, is, as these names imply, most frequently dry and papular, and is apt to be intolerably itching; the irritation is sometimes most severe, especially while in bed, while the disturbance of the rest, and the remarkable effect of mental depression which is common to most of the disorders of this region, make it sometimes a truly miserable complaint. The scratching of the patient produces a serous exudation or even hæmorrhage, and in chronic cases much thickening of the skin. French writers describe it as sometimes associated with a profuse and almost paroxysmal discharge of mucus from the rectum. This form of eczema is most common in elderly persons and is often associated with portal congestion and hæmorrhoids. In children it most commonly depends upon the presence of threadworms. Occasionally it is started by fissure of the anus, and disappears when this has been cured by division of the sphincter.

The scrotum and penis are frequently the seat of eczema, most often of the weeping form. Eczema vulvæ closely resembles eczema ani in its symptoms and, like it, most frequently affects persons beyond middle life. It is sometimes associated with, and probably dependent upon, diabetes, and sometimes appears clearly due to inflammation, new growths, or degenerative changes in the uterus or bladder. Eczema of the anus, perinæum, or genitals often proves very rebellious, and leads to great thickening and induration of the parts affected. Borax lotion or lead ointment, according to the degree of moisture, relieve, perhaps, more frequently than other applications, but this is one of the forms in which one must be content with tentative measures in each patient. In some obstinate cases a drying lotion of nitrate of silver proves effectual when other means fail.

For the *intertrigo* of infants, finely powdered starch and oxide of zinc or chalk is the best application. When it affects the fold of the nates in adults, it is better treated by extreme cleanliness and the application of vaseline or diluted white precipitate ointment. Glycerine to most skins proves an irritant rather than a healer.

"Eczema" of the legs due to *varicose veins* must be treated like varicose ulcers, by elevation and bandaging. An old-fashioned flannel bandage often proves a cheap and efficient method. Martin's elastic bandage has frequently the most valuable results, but in wearing it or an elastic stocking care should be taken that the pressure is not too great.

So-called "*eczema marginatum*" of the thighs is essentially a form of ringworm, and will be described under that head.

PAGET'S DISEASE OF THE NIPPLE was formerly described as eczema. Its characters were graphically delineated by Sir James Paget in the 'St Bartholomew's Hospital Reports' for 1874. In his original fifteen cases, cancer developed as a sequela within two years, marked by retracted nipple and its other symptoms.

The appearance of the affected nipples is at first that of superficial dermatitis, with an intensely red, raw surface, minutely granular, and pouring out "a copious, clear, yellowish, viscid exudation." The border is more sharply defined than is usual with eczema, and it is said to be thickened from the first, feeling, as Mr Henry Morris puts it, like a penny in a fold of cloth. In any case this induration of the edge is present in the later stages of the malady, and is the precursor of invasion of the ducts and the whole glandular tissue by ordinary alveolar carcinoma. The supervention of cancer has sometimes been delayed until more than two—six or even ten—years have elapsed after the eczema appeared; so that it is doubtful whether the cancerous growth should be regarded as more than a frequent result of the chronic cutaneous irritation.

A parallel case has been reported by Dr Crocker, where a patch of eczema of the scrotum in a man of sixty ended in the development of cancerous nodules ('Path. Trans.,' 1890); and the process is familiar to surgeons in the case of cancer of the vulva, glans penis, of the lower lip, and of the tongue, following chronic and apparently innocent inflammations of the same parts.

The anatomy of the disease has been investigated by Butlin, Thin, Bowlby, and other histologists ('Med.-Chir. Proc.,' May 12th, 1891), and many cases have been published on the Continent and in the United States (see 'American Journal of Med. Sc.,' July, 1884).*

The patients are usually women who have arrived at, or passed, the climacteric age. There is at first no special pain, and but little irritation.

It is most important to treat every case of eczema of the nipple as early and sedulously as possible, even when it does not offer the non-characteristic features above described; but when the raised and indurated edge is present, the best plan is probably complete destruction of the diseased surface by caustic.

* M. Darier brought before the Congress of Dermatology which met at Paris in 1889, remarkable microscopical observations of the presence of psorosperms (*coccidia*) in the epithelial cells of this disease, before as well as after cancerous changes begin. The animal parasite is described by Dr Louis Wickham in his *Thèse de Paris* (1890) as recognisable in an early stage by appropriate staining in the substance of the cells, and as subsequently becoming encysted, glistening, and readily seen. The true nature of this appearance is, however, far from decided.

PUSTULAR DERMATITIS—*Impetigo capitis*, *Porrigo*, *pustular eczema*.—This is one of the most frequent diseases in children. It was known to our forefathers as “scald-head,” but has happily become far less common than it was when children’s heads were more neglected than at present, and especially when the bad habit prevailed of covering the scalp with caps and linen hoods, indoors as well as out, by night as well as by day. Probably the majority of cases are due to the irritation of *pediculi capitis*, but there remain a large number where no such cause can be found, and where a similar eruption upon the face or other parts establishes its independent character. These forms of dermatitis resemble eczema; for they are superficial and never leave scars; they are often associated with ordinary characteristic eczema of the ears, the limbs or the trunk, and the same child may be affected at one time with what will be called impetigo of the face or scalp, and at another with eczema of the same parts; or in an infant with ordinary eczema of the scalp the dermatitis will be seen to become more pustular as the hair grows thicker over the head, until it has assumed all the characters of the *porrigo favosa* of older writers. Nor does the fact that this dermatitis of the scalp is often dependent upon dirt, lice, and other irritants, prevent our regarding it as true eczema, if the principles above laid down (p. 823) are correct. That something beside a traumatic cause, an *irritabile* as well as an *irritans*, is necessary for the production of impetigo, is proved by the fact that some children and most adults may have *pediculi capitis* for many years, and may even suffer from the irritation and yet be free from impetigo.

The accident which transforms a local eczema into impetigo is infection by a pyogenic microbe. The pus of impetigo is found to have been conveyed by infection from a whitlow or other source of suppuration on a child’s fingers. Or it may be transferred from one scalp to another by changing caps. Schoolboys, again, may acquire impetigo of the neck and chest by mutual contact in a football “scrum,” or by interchange of jerseys. The contagion of impetigo does not seem to be always the same: most often staphylococci, sometimes streptococci, and sometimes other pyogenic microbes are present.

We have already mentioned the best treatment for impetigo of the scalp when associated with ordinary eczema. The children who are the subjects of it are often rosy, plump, and in every way healthy, though here as in other cases it is necessary to judge by the trunk and limbs as well as by the face. If, notwithstanding fat cheeks and ruddy complexion, the child is found to have flat shoulders and nates, thin arms and thighs, disproportioned knees, and ill-developed pectoral muscles, his impetigo should be treated not only with ung. plumb. acet. or diluted mercurial ointment, but also by careful attention to diet, by Gregory’s powder, with or without grey powder, and when the digestive disorder is corrected, by cod-liver oil.

Impetigo affecting the scalp or face alone, without ordinary eczema, and in a healthy child, is happily not difficult of cure. Indeed, apart from the purely pustular secretion, from the eruption being discrete and with a defined margin, and from the absence of severe itching, these typical cases of impetigo are separated from eczema by the fact that they are not prone to recur. Zinc ointment is the popular remedy for the eruption, but its efficacy is much increased by the addition of equal parts of white precipitate ointment or by the substitution of unguentum metallorum (p. 836, *note*). The hair should be cut short, but there is no need to shave it, and the

parents may be assured that it will grow all the better afterwards. It is only in extremely rare cases after the inflammation has penetrated to the hair-sacs, owing to a deeper suppuration of the scalp from too strong local irritants, that the hair-sacs are destroyed, and a bald cicatricial patch results. Such an event is more often seen in impetigo of the scalp in an adult than in the far commoner disease in children. When, as is usually the case, the scabs are thick and massive, they should be removed first by poulticing. In circumscribed cases the bread and water poultice may be used, but where the whole scalp is covered it should be anointed with linseed oil and a large linseed poultice be then applied. For circumscribed and strongly adherent crusts, soft soap or even liquor potassæ may be necessary. Great patience and gentleness should be used in removing the scabs, or the child will suffer considerable pain, and the cure will be retarded.

Contagious porrigo has been separately described, and some authors have laid much stress on its distinction both from pustular eczema and from impetigo from pediculi; but no sharp line can be drawn. All impetigo is more or less contagious, and the degree of contagion varies here as elsewhere according to the pyogenic organism. The most virulent of all is the pus of a gonorrhœa; but probably even it is of varying degrees of activity, when we consider the frequency of the urethral inflammation compared with the comparative rarity of gonorrhœal ophthalmia. Leucorrhœa is supposed as a rule to be non-contagious, and no doubt with justice, but the most experienced surgeons admit the possibility of infection from an idiopathic and apparently innocent discharge. Again, the pus of boils is extremely contagious, and is often the source of what is called ecthyma. The pus of scabies, too, is contagious. So also a whitlow on a child's finger may cause by contagion impetigo of the hand, of the nates, and sometimes of the scalp. Impetigo often secretes pus of a most actively contagious kind, the proof being not only in the outbreak of similar pustules on other parts of the child's body, especially the fingers and the nates, but also in the spread of the disease to other children of the same household; so that in some cases a whole family or a whole street may be infected from a single case.

Purulent dermatitis is no doubt always associated with the presence of staphylococci, streptococci, or other pyogenic microbes. But what part is taken by bacteria in the pathology of eczema is still undecided. In 1891 Welch, of Baltimore, described an abundant coccus, with a white pure cultivation, which he named *Staphylococcus epidermidis albus*, probably normal; and Unna has also described the morococcus as constant in eczema but also found in psoriasis. At present no microbe has been found which is itself distinctive, which is present in all cases of vesicular or weeping eczema and in no other condition. (See Dr T. C. Gilchrist's report on the bacteriology of eczema, 'Johns Hopkins Hosp. Reports,' 1899, vol. ix, p. 419; and Galloway and Eyre's paper in the 'Brit. Journ. Derm.' for September, 1900, vol. xii, p. 307.)

DERMATITIS PRODUCED BY EPIZOA

“Occupet extremum scabies.”—HORACE.

SCABIES—*Importance of the disease—Its nature and history—The acarus—The superficial dermatitis it produces—The distribution of the parasite and of the inflammation—Diagnosis—Treatment.*

PHTHIRIASIS—*Pediculi capitis—Pediculi pubis—Pediculi vestimentorum—Their effects on the skin—Treatment of impetigo and prurigo pedicularis.*

SCABIES.*—The last variety of common superficial dermatitis is one which may be called traumatic or parasitic eczema, for it is pathologically identical with eczema and impetigo as described in the last chapter. But it depends directly and exclusively on the presence of an irritant, namely the invasion of the skin by a parasitic insect (or rather mite). Hence its distribution, ætiology, prognosis, and treatment are different from those of idiopathic or true eczema as above defined; and on clinical grounds—which should always decide classification—we accordingly place it apart.

This curious disorder is extremely common, almost as common as phthiriasis over the whole globe, and has been well known from the earliest times, though its origin has only recently been discovered. It was called Scabies by the Romans, though no doubt the term was applied to many itching disorders of the skin in men and animals which were not due to the acarus. It is more common in Scotland than in England, and more common in England than in America, where, however, it appears to be on the increase. Scabies is very common in lepers, in whom it has been described as a variety under the term Norwegian Itch.

Though once scarcely accounted worthy of a place in nosology, and though without the interest of danger, scabies is one of the most important diseases from a scientific point of view; for, if this were the place to enter fully into its history, we should find that it illustrates the whole progress of scientific medicine,—the ancient method which still survives of inventing explanations instead of investigating circumstances, the fallacy of ascribing results to causes of which the existence has never been proved, the survival of doctrines in pathology which have long been exploded in physiology, the value of apparently useless knowledge, the bearing of pure sciences like zoology upon practical therapeutics, the nature of inflammation and the relation between an irritant and an irritable tissue,

* *Synonyms.*—Greek, ψώρα.—Lat. Scabies, psora.—Fr. La gale.—Germ. Die Krätze.

the radiation of sensations, the psychology of pruritus, and the importance of a patient's nails in the production of cutaneous lesions. Finally, scabies is the typical example of a disease which is now as fully known as it is, perhaps, possible for us to know any disease—of which we know the pathology and the cause, of which we can explain the symptoms, which we can diagnose with certainty, in which the hypothetical *vis medicatrix naturæ* is utterly powerless, but which we can cure by definite and rational means, quickly, safely, and permanently.

Scabies, like the affections which have hitherto occupied us, is a superficial dermatitis: in the character of its lesions it may even be called a common superficial dermatitis, for they do not essentially differ from those which will be produced by any common mechanical or chemical irritant of sufficient energy, and are exactly comparable in their anatomy to the vesicles of eczema, the papules of lichen or prurigo, the bullæ of pemphigus, and the pustules of impetigo. Hebra, therefore, since as we have seen he called all common superficial dermatitis of traumatic origin "eczema," logically describes scabies as a form of eczema. But, as explained on a previous page (p. 822), eczema is not a mere traumatic dermatitis, and scabies must be separated from all other diseases because its cause, its prognosis, and, above all, its treatment, are totally different. We may define it as a superficial dermatitis, of various degrees of severity but always accompanied with pruritus, which results from the invasion of the skin by a parasitic acarus and from the scratching which ensues.

History.—The living cause of this disease is the female itch-mite now known as the *Sarcoptes hominis*, formerly as the *Acarus scabiei*, belonging to the acarine division of the class Arachnida. It is possible that the Greeks were acquainted with the acarus, for Aristotle describes *φθείρες* (i. e. pediculi) as coming out of little pimples which contain no pus ('Hist. Anim.,' v, 138). He applies the special term *ἀκαρί* to the cheese-mite (*ibid.* v, 144).

The acarus scabiei was recognised in the sixteenth century. Thus Rabelais (1483—1553): "*Mais d'ou me vient ce ciron icy entre ces deux doigtz?*" Ingrassias (1570—1580) described "lice which burrowed under the skin," and Hebra quotes a passage from the famous French surgeon Ambrose Paré, living at the same time (1570—1590): "*Les cirons sont petits animaux toujours cachez sous le cuir, sous lequel ils se trainent, rampent et le rongent petit à petit, excitant une fascheuse demangeaison et gratelle.*" In Thos. Moufet's 'Insectorum Theatrum' (Lond., 1634) he writes: "*Latine pediculi, gallice ces cirons. . . Anglice mites in caseo, foliis, ligno arido, atque cera; sed in homine whealewormes dicuntur. Sunt pediculi, subter manuum crurumque et pedum cutem serpentes, et pustulas ibidem excitantes aqua plenas: tam parva animalia ut vix visu perspicaci discerni valeant.*" This account seems to have been taken from an Arabian writer, Abenzoar (Ebn Zohr).

The itch-mite.—The parasite has four pairs of legs (which at once distinguish it from parasitic insects), and is clothed in a chitinous integument furnished with abundant bristles. The male acari, which are much the smaller in size and fewer in numbers, live upon the surface of the body, but do not burrow. The female, after impregnation, digs her way into the integument, forming a straight, curved or sinuous *cuniculus* (mite-burrow, *Milbengang*, *sillon* or "run") which is visible to the naked eye as a slightly raised ridge, with a dark depression at one end (the entrance clogged with

dirt) and a papule or small vesicle at the other deeper end, where the parasite lies.* A lens of low power shows these characters more clearly; but it is comparatively rare to see the runs perfectly well developed, for they are injured by the patient's scratching, by friction, and by dirt. When fresh they are best seen in the soft skin between the fingers and on the ulnar and flexor side of the wrist, still better when present in the skin of the prepuce and penis, or in that of the mammary gland in women. In children their locality is less certain, and they are much less easily found.

With good light and a little dexterity the burrow may be laid open by a needle from the entrance to its blind extremity, and the acarus, a minute white grain just visible to the naked eye, extracted. It generally clings to the point of the needle, but a microscopic slide with a drop of water, glycerine, or liquor potassæ should be ready to receive it. The needle should be sharp, stout, and not too long or elastic; some prefer the broader needle of the oculist. Another plan is to excise the parasite, burrow and all, by means of a sharp pair of scissors curved on the flat. The winding passage can then be demonstrated, with the acarus, its black granular fæces, and often with a row of oval eggs in chitinous cells, which are laid one by one as the acarus bores deeper into the skin. Sometimes the scissors fails to secure the parasite, but its presence is proved by that of one or more of its ova.

The dermatitis.—The bristles of the acarus produce irritation which in most cases is intense, equal to that of the most irritable eczema or the worst kinds of prurigo; but often it is comparatively slight, only annoying the patient after he is warm in bed, when the skin is more vascular, the papillæ more sensitive, and possibly the acarus more lively, while the patient has nothing to divert his attention from his own sensations. The degree of inflammation also varies extremely, and cannot always be explained by the more or less severe scratching of the patient. As above stated, there is usually a small vesicle formed at the end of each run; but beside these, large vesicles, bullæ, and pustules frequently follow, first on the hands and then (probably through transfer of pus and serum by the patient's fingers) on various other parts of the body, where runs and acari cannot be discovered.

Small acuminate papules are very characteristic; and not less so are the scratch marks, often accompanied, especially in children, by wheals like those of urticaria. In severe cases of scabies the dermatitis may be intense, both hands and arms swelling as if with phlegmonous erysipelas; or arms, hands, legs, and feet may be the seat of weeping, raw surfaces like those of eczema madidans; the lymph-glands of the axillæ and the groin become swollen and painful, and the excessive itching is at last replaced by the smarting and tingling of acute dermatitis. Frequently, especially in children, the pustules resemble impetigo or ecthyma, and form as they dry up thick scabs and crusts. In chronic cases—for unhappily we often see scabies which has lasted for weeks and months without detection, and has been therefore ineffectually treated—the skin becomes thickened, indurated, hard, scaly, and fissured, resembling the condition of the more chronic forms of dry eczema. Bullæ as large as those of pemphigus are less frequent lesions of scabies, but are not uncommon in children—a case was figured in the 'Guy's Hospital Reports' for 1877. In fact, we may say that any of the

* Compare the figures given by Dr Bristowe in his 'Practice of Medicine' with those of M. Hardy in his recent 'Traité des Maladies de la Peau.' The writer's experience is that short straight runs are more frequent than would appear from either of these figures.

inflammatory lesions of eczema, urticaria, pemphigus, impetigo, ecthyma, and lichen may be more or less perfectly represented. On the other hand, the large, flat, and discrete papules of prurigo, the imbricated scales of psoriasis, and the thin, dry, abundant squames of pityriasis rubra are never simulated by scabies.

Localisation.—The acarus itself infests the thin skin of the hands between the fingers, the flexure of the wrist, particularly its ulnar side, the flexor surface of the front of the forearm less frequently, the foot and ankle occasionally, the axilla and the groin, the genital organs, the inner part of the thigh, and the fold of the nates. But the lesions indirectly caused by its presence have a far more extensive though perfectly definite range; in fact, the local distribution of scabies is so well marked that in a majority of cases a glance is sufficient to identify it. The inflammatory lesions are always present on the hands, except occasionally, when the patients are engaged in some handicraft which leads to the constant immersion of their hands in oily or strong-smelling substances or in metallic solutions, and prevents the development of the parasite. The same result is often seen in private patients, where the hands escape owing to the frequent use of soap and nail-brush. With these exceptions, which are important for diagnosis, the fingers and ulnar side of the wrist may be said to be the favourite seats of scabies, as they are of the acarus. Some lesions will almost always be found upon the prepuce, and the inflammation usually affects not only the thin skin of the genitals, but that of the lower part of the abdomen, at least as high as the umbilicus. The whole of the forearms is very liable to be affected, and the eruption is more general than is the case with eczema. The axillæ seldom escape altogether. The buttocks are almost always more or less affected, and in children nearly constantly, especially at the gluteal fold, but the perinæum and the sacral region usually escape. The toes, feet, and ankles, especially the inner ankle where the skin is thinnest, are very frequent seats of the dermatitis of scabies; less often the knees, but the whole of the inner side of the leg and thigh may share in the inflammation. The back, shoulders, and chest are but little affected, the thick or hairy skin being apparently less favourable to the parasite. The neck, face, ears, and scalp almost invariably escape, in striking contrast to the frequency of eczema and impetigo in these parts; and the only exceptions are in children. Why the skin of the face is shunned is hard to say. It is not more exposed to the air than the hands, it is as thin and delicate and vascular as that of the abdomen, but for some cause it is shunned by the acarus. May it be that the large sebaceous sacs and thick cutis with thin cuticle which are characteristic of the face, as of the shoulders, the chest, and the scalp, furnish a fatty secretion which repels the invader?

It has often been remarked that a line drawn across the waist and arms of a man standing in "the first position," will have below it the regions of scabies acarorum, and above it those of prurigo pedicularis.

In children, localisation of scabies is much less strict than in adults, as is the case with eczema and psoriasis, and probably from the same reason. Only in children is the face affected; the hands frequently escape, and runs are found as well or better on the ankle or in the skin of the sole; the nates, and sometimes the trunk, are often more affected than the limbs.

Diagnosis.—This depends on a recognition, first, of the characters of the dermatitis, next of its very constant localisation, and thirdly of the cuniculi, the ova, or the acarus. The general facies of the disease is so character-

istic that nine out of ten cases will be recognised in a moment when the patient is stripped, but in private practice anyone may be thrown off his guard who is accustomed to diagnose by probabilities rather than by facts. As Sir William Gull said, there are three diseases which we all sometimes overlook,—phthisis and syphilis and itch.

When the inflammation has obscured all trace of the acari, their existence may be proved by removing the crusts, boiling them in solution of potash or soda, and allowing the dissolved mixture to stand in a conical glass. On decanting and removing the deepest layer with a pipette, fragments of the chitinous skeleton may be recognised.

It need not be said that scabies is always *contagious*, and its occurrence in an entire household often leads to its recognition. It is remarkable, however, how cases may remain isolated, and we must remember that impetigo and prurigo (pedicularis), not to mention variola and varicella, may also be contagious. The mode of transference is not always easy to follow; direct contact of hands is probably one method; often the ova are conveyed by clothes or other articles of constant use. Bedfellows seem particularly liable to infection. Lastly, it must be remembered that scabies is frequently a venereal disease, the acarus having first invaded the genital organs; and in otherwise cleanly persons this origin is apt to be overlooked.

Treatment.—Experience long ago discovered that sulphur is good for the itch; it is an effectual poison to the acarus, and all we need is the best method of applying it.

The general practice is inunction of unguentum sulphuris into the affected parts, especially those which are the chief seat of the acarus. The colour of the application may be disguised, but its smell is always unpleasant. Sulphur lotions or sulphur fumigations may be substituted, but neither are so effectual. The best method is for the patient to rub the ointment well in every night, to lie in merino clothing all night, and next morning to wash with hot soap and water, and apply a little dilute ointment to the most irritable parts. For women and children the *ung. sulphuris* of the Pharmacopœia should be diluted; and even in men the addition of an alkali aids its efficiency.

A rapid cure may be effected by first rubbing the skin with soft soap so as to remove crusts and epidermis, and then using thorough sulphurous inunction. In this way patients are cured in a few hours at St Louis on a large scale, and their clothes meanwhile are baked and washed. This last precaution is important, since otherwise the patient may readily reinfect himself from his own clothing. With private patients the disease rarely gains such extension by neglect as to be severe, and its cure, when once the diagnosis is made, is usually quick and easy.

It may, however, happen that the sulphur ointment is itself too irritating; so that, although it kills the acarus, it perpetuates or sets up a fresh and even more severe dermatitis. One often sees these cured but over-treated cases of scabies, and all that is necessary is to recognise their nature.

With children, diluted ointment,—two to one, or equal parts, in infants with much dermatitis one to two, are the best proportions, the dilution being made with benzoated lard or with zinc ointment.

In slight cases, especially in children, balsam of Peru is a pleasant and generally an efficient parasiticide.

PHTHIRIASIS.—Another common parasite, much commoner than the acarus, and indeed universal, except under the constant control of civilised habits, is the pediculus. This is a true insect, with six feet, and does not burrow into the skin like the itch-mite, but clings to the hairs or to the clothing. All animals have their lice as they have their ticks, and the same applies to man in a state of nature.

Impetigo a pediculis.—The *Pediculus capitis* causes a certain degree of irritation, but much less in adults than in children. In the latter subjects pediculi are the common cause of the pustular dermatitis of the scalp, and particularly of the occiput. In all cases of pustular inflammation of the scalp the hair should be carefully searched for pediculi. Equally decisive of the cause is the discovery of the nits, which consist of small triangular cases containing the eggs made of hard material, in colour and consistence like dried size, adhering to the hairs by one side of the triangle, and visible to the naked eye. The impetigo which results from their presence is produced more by the scratching of the patient than by the direct irritation of the lice. It affects the back of the scalp chiefly or exclusively, and is attended with great consecutive swelling of the posterior cervical lymph-glands. Indeed, occipital impetigo in children is almost synonymous with impetigo a pediculis.

The treatment is decisive and efficient. In bad cases, the whole of the long, tangled, and filthy elf-locks should be cut off, and the head washed with soap and water; but in slighter cases it is not necessary to cut the hair at all. The noxious insects are readily destroyed by mercurial washes, but an equally efficient and harmless remedy is the stavesacre ointment (3ij ad ʒj). Common petroleum oil is also a cheap and efficient parasiticide, or if the hair is cut short, as is much the best plan in hospital practice, the white precipitate ointment which cures the disease will also kill the vermin. The egg cases are less easily attacked, and might be sources of future trouble. They must be either combed off, or removed with spirits of wine, or cut off, hair and all. The impetigo which results from pediculi will sometimes heal spontaneously as soon as they are removed, but usually unguentum metallorum, or white precipitate ointment, diluted to 1 in 3, hastens recovery.

The *Pediculus pubis* is a larger and broader kind of louse which infects the pubic hair in adults of both sexes. Though more repulsive it is less frequent and less severe in its effects than the last mentioned species, and is easily cured with white precipitate ointment.

The *Pediculus corporis* or *P. vestimentorum*, or body louse, is somewhat longer and narrower than that of the scalp, and lives in the underclothing, and particularly in the pleats and folds of shirts and petticoats. It also, however, clings to the fine hairs of the skin and fixes its eggs to them, an observation due to Dr Jamieson in 1899.

In children it produces a certain amount of irritation, and an eruption of papules or of wheals (p. 859). In elderly patients it has a more constant, obstinate, and severe effect.

Prurigo senilis a pediculis.—This is a well-characterised and common disease known as prurigo senilis, phthiriasis, or prurigo pedicularis. It is a papular dermatitis of definite clinical characters dependent on the irritation of body-lice, and is only seen in elderly persons. It is a good example of the combination of two conditions—the excitant and the predisposing cause, the *irritans* and *irritable*—to form a constant clinical result.

Phthiriasis is not a sufficient title, for children may be swarming with vermin, and may suffer from urticaria or ecthyma as the result, but are never affected with this form of prurigo; nor is "prurigo senilis" enough, unless we recognise the exciting cause of the disease.

The papules are separate, not spreading over wide surfaces as in eczema, nor collected in more or less rounded patches as in lichen circumscriptus, nor coalescing as in lichen planus. Moreover, they are much larger than in eczematous dermatitis, flat rather than pointed, less red, and more persistent. But what is most characteristic is that before long each of them is capped by a little black crust of dried-up blood, the result of scratching.

Beside these papules, the disease is marked by an extensive series of scratch-marks following the curves which are described by the right or left hand respectively, working from the shoulder. The irritation of scratching not only causes excoriation and hæmorrhage, but sometimes produces wheals like those of urticaria, and raw surfaces which may be properly called traumatic eczema. Both these effects may be absent, but prurigo senilis never lasts long without the whole surface between the papules becoming more or less deeply pigmented, until in some cases the affected parts are as dark as the skin of a mulatto.

The *distribution* of prurigo senilis is as characteristic as its anatomy. It occupies the shoulders, back, and loins, the papules usually stopping abruptly at the waist or the sacral region, and sometimes not spreading below the scapulæ. They may appear over the upper arms, but rarely below the elbow, and never on the hand. They are numerous on the flanks, and in severe cases may cover the whole chest and abdomen. The thighs may share in the disease; but even in the most extensive cases it is generally found that the outlying parts are rather the seat of ordinary dermatitis produced by scratching than of the true papules of the disease. Prurigo senilis never affects the face.

The itching is most severe; and, like all pruritus, it is worst at night and when the patient is warm. The absence of pain and tenderness leads to more reckless scratching than in any other disease. It is the consequence, however, and not the cause of the papules, for we can distinguish the latter from the traumatic dermatitis set up by the former.

The exciting cause of the disease can be found when carefully looked for, especially in the plaits of the underlinen about the neck and waist. It is important to remember that pediculi corporis may exist in old men and women of apparently scrupulous cleanliness.

The whole facies of the disease is so well marked that it can scarcely escape recognition. It affects both sexes. Typical cases are very rare in persons as young as fifty.

The *treatment* is simple and effectual when the disease is once recognised. The most effectual parasiticide is the white precipitate ointment, and if only applied to the shoulders no harm will ensue; when used more freely and extensively it may cause salivation. Inunction, as used with grey ointment in cases of syphilis, is quite unnecessary; it is enough for the parts to be smeared over. Carbolic acid lotion (5 p. c.) or diluted creasote ointment relieves the itching. The clothes must be scalded or fumigated.

PAPULAR FORMS OF CHRONIC SUPERFICIAL DERMATITIS

(PIMPLY TETTERS)

“Ardentes papulæ, atque immundus olentia sudor
Membra sequebatur.”—VIRGIL.

Definition—Relation to common dermatitis and to eczema.

LICHEN.—*Its traditional species—Lichen circumscriptus—Lichen tropicus—Lichen pilaris—Lichen urticatus, etc.—Strophulus—Lichen scrofulosorum—Lichen planus—Lichen acuminatus—Lichen ruber.*

PRURIGO.—*Pruritus—Summer and winter prurigo—Prurigo senilis—Idiopathic Prurigo: Hebra's and milder forms—characters and treatment.*

THE group of diseases to be now described is far from being natural or well defined. They agree with eczema in being inflammatory, in beginning as papules, in affecting only the epidermis and the papillary layer of the cutis, so that they never leave scars, in their essentially chronic course, in the greater itching than pain that they produce, and in their persistence and liability to recur.

But they differ in the following important points:—(1) The inflammation never goes on to the stage of exudation either of serum or of pus; they are all “dry tetters.” Even when, as the result of scratching, common irritative exudation follows, the pustules or raws thus produced are limited by the cause, and only resemble eczema or impetigo for a time. (2) They are much less symmetrical, and rather avoid than choose the favourite places of eczema. Their locality may be said to be undefined, and widely diffused, but they affect rather the trunk and the exposed part of the limbs than their flexures.

It will be convenient to deal with all the traditional “papular diseases” together, different as they are in pathology. Of the whole group only two can be said to be natural and well-defined morbid types, with a characteristic anatomy, course, and habitus: these are *Lichen planus* and *Prurigo*.

Willan, who first strictly defined the term papulæ, admitted three genera of papular diseases: *Strophulus*, *Lichen*, and *Prurigo*, and of these names the latter two are still in general use.

LICHEN.*—The word Lichen (λειχήν, translated by the Latin word *impetigo*) was applied in Greek to any moss or "lichen" that grows on stones or trees, and also to any more or less similar rough "efflorescence" on animals or man.

The word is very commonly used in the Hippocratic and Galenical writings and by later writers, but without stricter definition than the following:—*Lichen* est summæ cutis vitium, ut *psora et lepra*, cum asperitate et levi pruritu; deterius quidem Pruritus, Psora autem et Lepra levius.

Some dermatologists still use this term to denote a papular dermatitis which by subsequent more or less exudation of moisture, or by its symmetry, or by its association with previous or later attacks of ordinary eczema, proves itself to be more properly termed papular or *abortive eczema*. Its pathology, natural history, prognosis, and principles of treatment are precisely those of the early stage of eczema. It most often affects the arms and legs, and the extensor rather than the flexor aspect, and in many cases is identical with Seborrhœic eczema.

Willan's definition of Lichen is: "An extensive eruption of *papulæ*, affecting adults, connected with internal disorder, usually terminating in scurf, recurrent, not contagious." Bazin divided lichen, according to its origin, into parasitic, dartrous, herpetic, scrofulous, and syphilitic—a good example of his classification and pathology.

Lichen circumscriptus† was the name given by Willan and Bateman to a peculiar and characteristic disorder, which will be referred to in a future chapter, under disorders of the sebaceous glands.

The form of lichen described by E. Wilson, and also by Hardy (*lichen circonscrit*), affecting the extensor aspect of the forearm and the back of the hands, and running an acute course, should probably be regarded as eczema papulatum; but the *Lichen circonscrit* of Cazenave was what is now known as *Lichen scrofulosorum* (p. 859).

Lichen tropicus.‡—The writer has seen only three or four cases of this curious affection, so well known in the East and West Indies under the name of *prickly heat*. It occurs also in Australia and on the West Coast of Africa. Its characters are the sudden appearance of the eruption, its almost universal distribution, and the intense irritation it produces. It is said to affect blonde more than swarthy persons, and whites more than blacks. After once attacking a patient, it is apt to return with each hot season until the patient is acclimatised, and, though usually cured by a temperate climate, it sometimes comes before us in England. In the cases referred to the eruption has been entirely papular, with no other lesion but scratch-marks or occasional wheals. The parts most affected were the abdomen, buttocks, and thighs. The face and scalp, the hands and feet, and the genital organs seem to be usually free. Its course and characters are quite unlike *Lichen planus* or *L. ruber*. One must speak doubtfully about a disease of which the personal experience of an English physician is small; but from its acute character, the absence of moisture, and its uneczematous

* *E. g.* *σαρκῶν ἐπαμβρατῆρας ἀγρίαῖς γνάθοις*]

λειχῆνας, ἐξέσθοντας ἀρχαίαν φύσιν.—Æschylus, Choëphori, v, 281.

† *Synonyms*.—*Lichen annulatus* (Wilson)—*L. marginatus* (Liveing)—*L. circinatus*—*Lichen acnéique*—*Eczema flavum*—*Seborrhœa of the trunk* (Duhning).

‡ *Synonyms*.—*Gr.* *ἰδρωα*.—*Lat.* *Papillæ sudoris*.—*Arab.* *Essera* (in part).—*Angl.* *Prickly heat*.—*Dutch*, *Rootvont*.

distribution, it is at present best classed as a form of papular dermatitis, from heat and sweat. The small red papules are frequently associated with sudamina.

It was once supposed of prickly heat, as of other eruptions of the skin, that driving it in by a cold bath was extremely dangerous; but more than sixty years ago Dr James Johnson, who gave a graphic account of it in his own person, justly ridiculed this superstition.

The late Dr Tilbury Fox regarded this disease as essentially an *adenitis* of the sweat-glands, the direct result of excessive heat and perspiration; and Dr Duhring, of Philadelphia, in his excellent text-book of dermatology, calls it *miliaria papulosa* on the same theory. Dr Crocker gives it the same name, and puts it among affections of the sweat-glands.

Whatever its pathology, it has no affinity to any acknowledged form of lichen.

Lichen pilaris was a name applied by Willan to a familiar condition which is, however, not a dermatitis at all. The hair-sacs of the affected part of the skin become filled up with horny cuticle, which forms rough papular projections, hard, pointed, and very characteristic, both in appearance and feeling. They do not occur in places where the hair is long, but are almost exclusively confined to the outer side of the limbs, over the vastus externus most often, but not uncommonly more or less developed on the extensor surface of the arms and legs, on the buttocks, and the shoulders. The condition is most common in the brawny skin of muscular working men, and may be readily removed by soap and water and friction. Occasionally it may be seen on the back, loins, or limbs of delicate children, in girls of only seven or eight years old. This affection was described by Devergie under the equally inappropriate terms *Lichen spinulosus* and *Pityriasis pilaris*. It may be better called *Keratosis pilaris*. Dr Fagge proposed for it the name *Rhinoderma* (from *ῥίμη*, a file, not *ῥίς*, a nose): but, as he himself says, this term has such obvious disadvantages that he prefers Devergie's title.

Occasionally keratosis pilaris is complicated by local inflammation and large flat red papules, or pustules may result, each surrounding a minute hair. These cases have been called *Lichen ruber pilaris* or *sycosis* of the trunk or limbs. Several examples were shown at the Dermatological Society of London in 1885—86. Such cases have probably been often recorded as examples of the rare form of disease, to be presently described, *Lichen ruber* of Hebra: indeed, Hans v. Hebra is convinced that some of his father's typical cases were really inflamed keratosis pilaris. Some authors restrict the term *Lichen pilaris* or *Pityriasis rubra pilaris** to the cases of keratosis which are inflamed; but it is surely better to drop these misused terms altogether. *Keratosis pilaris*, whether inflamed or not, has no relation to "true" Lichen (*L. planus et ruber*), and is naturally associated anatomically with other affections of the hair-sacs.

Willan's *Lichen lividus* is purpura, the petechial spot being perforated by a minute hair.

The *Lichen agrius* of Willan is clearly, from its acute course and the

* To be carefully distinguished from *Pityriasis rubra* of Devergie. *Pityriasis rubra pilaris* is closely allied to, if not identical with, *Lichen ruber acuminatus*, while *P. rubra* is closely allied to, if not identical with, *Dermatitis exfoliativa* (p. 877).

presence of small vesicles filled with a straw-coloured fluid, an acute form of eczema.

Lichen urticatus (Bateman) seems to be nothing but papular erythema combined with urticaria. It corresponds to much of what has been called prurigo infantilis and prurigo æstivalis. It is extremely irritable, and only seen in children. In most of these cases in young subjects the first step in the pathological process is pruritus set up by fleas or other vermin, the second the little patient's scratching, and the third papules and wheals caused by the friction.

Lichen hypertrophicus was a name given by some French writers to what is probably in most cases identical with the rare affection to be described in a later chapter under the name of *Mycosis fungoides*.

Lichen syphiliticus is a bad name for *Syphiloderma papulosum*. It has been described in the chapter on lues (vol. i, p. 343).

The term *Strophulus*,* applied by Willan and Bateman to certain papular eruptions in infants, is now deservedly abandoned. Willan defines lichen as a papular eruption occurring in adults, so that the original distinction between the two diseases was merely one of age.

Green remarked that "strophulus differs from lichen in no essential particular, a circumstance that might warrant us in discussing the two diseases under one and the same head" ('Compendium of Diseases of the Skin,' 1836, p. 174). This author points out the difference in age of the patients, the more frequent intermissions of strophulus, and its milder character. Rayer regarded strophulus as "infantile lichen," but Wilson described them separately. Most authors admit that the papules so closely resemble those of lichen as to appear identical with that disease. They are, indeed, only modified by the age of the subject.

Strophulus, therefore, may be regarded as a synonym either for infantile urticaria ("*Lichen urticatus*"), or for papular dermatitis from sweat or other irritants occurring in children.

Strophulus albidus, however, is not a form of dermatitis, but identical with *milium*,—a variety of comedo which will be mentioned among affections of the sebaceous glands.

Strophulus intertinctus and *S. confertus* may be called infantile papular dermatitis of more or less acute form, and in most cases may fairly be termed eczema.

Strophulus volaticus, with its acute course and slight maculæ following the patches, is a typical form of *erythema papulatum*. Bazin and Hardy were unable to class these papular eruptions of infants among the chronic inflammations which they ascribe to the dartrous diathesis. The former writer placed them under it among the scrofulides, the latter among what he ingeniously calls "Maladies cutanées accidentelles." The *Strophulus prurigineux* of these authors seems to be identical with infantile *Lichen urticatus*.

Lichen scrofulosorum, or, as it is more conveniently called, *Lichen*

* *Strophulus*.—This name, derived from *στροφος*, a swaddling band, was apparently first used to describe any skin eruption occurring in an infant. A popular English name is *red* or *white gum*, or *tooth-rash*. These names point to the popular explanation of all cutaneous rashes, and most other affections which occur during teething; but it is probable that originally "red gum" was only a corruption of another vernacular title—"red gown," a not inapt description of a child covered with general erythema; and this word *gown*, though in English meaningless without the prefix, is only a translation of *Strophulus*.

scrofulosus, is a somewhat rare form of eruption, which was first accurately described by Hebra. He defined it as consisting of papules arranged in groups with some amount of pigment and slight but constant desquamation, not itching, and lasting for a long time without change. It is almost always confined to the trunk, and in forty-five out of fifty of Hebra's original cases the patients had swollen lymph-glands, or chronic disease of the bones, or scrofulous ulcers, or were supposed, from a fulness of the abdomen, to be subjects of *tabes mesenterica*, the exceptions being found in children. On the other hand, in none of these cases was there evidence of phthisis. All Hebra's cases occurred in young men, the youngest patient being fifteen and the eldest twenty-five. His description has been followed by subsequent German writers, who have added little to the account which he gives. The writer saw two of his cases in Vienna, and can testify that they were not, as has been naturally supposed by some writers, cases of pityriasis scrofulosorum sive tabescentium, the xeroderma or dry rough scaly condition of the skin not uncommon in phthisis and other wasting diseases.

Hans von Hebra calls it *scrofuloderma papulosum*; Sack, *tuberculosis lichenoides*. It does not appear under any form of Hardy's *scrofulides*; but might have been included in Bazin's large group of *scrofulides bénignes*.

Kaposi has made sections of the affected integuments, and describes the sebaceous glands as blocked by epidermic plugs, and surrounded by a copious infiltration of leucocytes, so that according to this excellent observer, the disease would be a chronic inflammation of the corium surrounding the sebaceous glands. The late Dr Tilbury Fox transcribed Hebra's account without comment, only stating that the condition is "of infinitely rare occurrence in England." He afterwards published six cases in the 'Clinical Transactions,' vol. xii, with a plate. Dr Liveing has met with a few typical cases among poor out-patients, and thinks that the inconspicuous colour of the papules and the absence of itching lead to its being overlooked. Several well-marked cases have been lately shown before the Dermatological Society; one was a patient of Dr Payne's, a girl aged seven, pale, and with swollen glands. When seeing out-patients I have occasionally observed the characteristic pale papules on the back in sickly children who came for tuberculous diseases, but it is very rare among patients who come for diseases of the skin.

From English experience, Lichen scrofulosus is more common in children than in adults, and is as common in one sex as in the other.

Its locality on the back, its absence on the face and limbs, the circumscribed patches, the pale colour of the papules, and the yellowish pigmentation, together with entire absence of itching, are sufficient characters for diagnosis, and justify its recognition as a distinct variety of chronic papular dermatitis occurring in tuberculous subjects.

It has been mistaken for *tinea corporis*, papular syphilis, *keratosis pilaris*, and papular eczema. The characteristic bacilli of tubercle do not appear to have been detected in the affected skin, except in one or two doubtful cases; the infective agent is probably a tuberculous toxine.

A case of this disease has been recorded by Dr Gilchrist, of Baltimore, in a negro girl eleven years old. The distribution was on the back and slightly on the chest and abdomen; the histology was that of a circum-follicular proliferation of lymph-cells and giant-cells, but no caseous desquamation.*

* Sydenham Society's 'Selected Essays and Monographs,' 1900, p. 271.

The treatment consists in the internal administration of cod-liver oil. Locally the skin should be anointed with vaseline, cold cream, or olive oil.

LICHEN PLANUS.*—There is a form of chronic superficial papular dermatitis which is so distinct from the rest that it is well entitled to a separate name. In fact, it is the only one of the traditional species of Lichen which is a primary and characteristic disease. Of all forms of papular dermatitis it recedes furthest from typical eczema, and approaches nearest to the dry tetter or psoriasis which will be described in the next chapter. It does not appear to have attracted the attention of the older dermatologists, and is indeed a somewhat rare disease. It was first described by Hebra under the title *lichen ruber*, and shortly afterwards, from a different point of view, by Erasmus Wilson, under the more distinctive name *lichen planus*, which has been generally adopted in this country. In Germany the two forms are distinguished as *lichen ruber acuminatus* and *lichen ruber planus*. Unna recognises a third variety, *lichen ruber obtusus*.

Hebra gave an elaborate table of the differences between *lichen ruber* and *lichen scrofulosus*, psoriasis, eczema, and pityriasis rubra. Some dermatologists question whether the disease described in Vienna is really the same as *lichen planus*, but in pathology and the essential points of their natural history the affections named by Hebra and by Wilson are one, although they represent two varieties which may be recognised.

Wilson thought that the cases described by him were varied examples of the *lichen ruber* of Hebra, but Hebra himself considered the two affections to be distinct, and this was also the opinion of Fagge and of Crocker, and probably of a majority of dermatologists. The substantial identity of *L. ruber* and *L. planus* is held by Kaposi and by Unna. It is held also in America by Duhring, A. R. Robinson, and R. W. Taylor, of New York, where Hebra's form of the disease seems to be more common than Wilson's. Hans von Hebra describes two forms: one very rare, more acute, with greater formation of scales, more itching, and more generally diffused dermatitis, and also followed by more severe affection of the general health; the second common, more chronic, never spreading over the entire surface, with only slight irritation, and with no injurious effects on the health ('Kr. Veränd. d. Haut,' p. 376, and 'Brit. Journ. of Derm.,' March, 1890).

It appears to the writer that we must recognise two types of the disease: one more discrete, more widely spread over the surface, and with more marked general symptoms ("*L. acuminatus*"); the other more confluent, more strictly localised, and with less irritation and general disturbance ("*L. obtusus vel planus*"). Moreover, it is probable that most cases of *L. acuminatus* begin in the hair-sacs, while those of *L. planus* do not. That they are varieties of the same malady seems shown (1) by the fact that one passes into the other by intermediate cases and even in the same patient, so that we have seen what Hebra would have called "exquisite *Lichen ruber*" of the trunk and legs, with equally typical *Lichen planus* of the arms; (2) by the identity of the elementary lesion in appearance and histology; and (3) by the course of the disease.†

* *Synonyms*.—*Lichen ruber*—*Lichen invétéré* (Hardy), including *Lichen plan corné* (Vidal)—*Lichen ruber et planus*—[*Pityriasis rubra pilaris*]*—Lichen ruber acuminatus*.

† See an account of the discussion on the mutual relation of *Lichen planus* and *L. ruber* which took place at the Dermatological Congress at Paris in 1889, reported in the 'British Journal of Dermatology' for October of that year.

The lichen-planus type is the most characteristic, the other approaches nearer to other forms of papular dermatitis.

The earliest lesion is a rather large violet-red papule; this soon is tipped with a white scale. Others form near it, and as they increase in number come closer together, and at last coalesce; but they certainly also grow larger. When confluent, they form a raised flat nodule or plateau, with a scaly, shiny surface.

Anatomy.—No one who has seen a well-marked example of Lichen planus can doubt the accuracy of Wilson's description; the raised flat patches, their dull, glistening, striated surface, purple-red colour, slight desquamation, chronic course, and resulting pigmentation are together most characteristic. Hebra insisted upon the genuine papular origin of the affection, on the deep red colour of the papules, and their not increasing in size when once formed. Fresh papules appear, and they become confluent, so as to form the raised flat patch which struck Wilson's attention (see model 260 in the Guy's Museum). As Dr Crocker described, the papule is, as a rule, found where a sweat-duct opens; and this may be connected with the presence of a small plug of epidermic scales, which afterwards drops out and leaves a slight depression in the centre of the papule.

The cells of the rete mucosum contain granules of dark-brown pigment, the natural papillæ are enlarged, the sebaceous glands atrophied. There is after a time considerable thickening and induration of the skin, as in other forms of chronic dermatitis.

In less typical cases the papule has, at least at first, a brighter and less purplish-red tint. They are often pointed or rounded instead of flat, and long remain separate. A moniliform distribution of the papules in a chain has been described by Kaposi and two or three other observers. The late Dr Cavafy recorded a case of an annular arrangement of lichen-papules, and Marrant Baker (quoted by Crocker) and Hallopeau have seen the papules white instead of red.

Pigmentation of the surrounding skin is not remarkable in ordinary cases, but in those of long standing it becomes obvious, most intense where there is most irritation and most scratching.

Distribution.—Lichen planus occurs on the extremities or trunk, but has never been observed on the face or head. Hebra described it as sometimes affecting the palms and soles, and this statement is confirmed by Wilson and by Hutchinson, but such cases are rare. The patches are apt to be most marked in parts subject to friction, as the waist and the circle of skin pressed on by a garter.

Its favourite positions are on the limbs, especially the forearm and wrist (front as well as back), and the leg below the knee; also the thigh, particularly the skin over the internal condyle and the hollow over the great trochanter. In the lower limbs the colour is more deeply purple than elsewhere. Lichen planus in the most typical form may occupy both elbows and both hams as well as the wrists.

It is often symmetrical, but less constantly than psoriasis or eczema. In a case shown by Dr Mackenzie at the Dermatological Society of London, the papules followed the course of branches of the brachial plexus. Several other writers have recorded Lichen distributed in the course of lesser sciatic or branches of the great sciatic nerve. These cases suggest the possibility of a nervous pathology of Lichen: but after all they are exceptional, and perhaps some of them accidental, in distribution.

In cases which agree more nearly with Hebra's lichen ruber the papules are of a brighter colour, and more generally distributed over the limbs and trunk. The papules do not form raised patches, and are pointed, not flat (*Lichen acuminatus*). In extensive cases the nails may be affected, but this complication is less common than in chronic eczema.

Mucous lesions.—Lichen planus is undoubtedly often associated with the white patches on the tongue and cheeks which have been described under the varied titles of ichthyosis linguæ, psoriasis linguæ, tylosis, keratosis, and leucoplakia. The association was noticed in two of his cases by Mr Hutchinson ('Lectures on Clinical Surgery,' vol. i, pp. 211, 213), and it probably can be found if looked for in a third of the cases.

Natural history.—Hebra's patients were almost all men. In England *Lichen planus* has been more often seen in women, but in 28 consecutive cases of the writer's there were 15 men and 13 women. It seldom or never attacks children, but the writer has seen a typical case in a girl of thirteen and another in a boy of nine. Observers differ as to the severity of itching. In the cases on which the present account is founded it has once or twice been absent, sometimes troublesome, but never severe, that is not comparable to the irritation of eczema, scabies, or prurigo. In a minority of cases, however, there is the most severe and obstinate pruritus, and these are usually the ones which begin acutely and are accompanied by other nervous or even mental symptoms.

Lichen planus is, as a rule, chronic in its development and course. Hebra described lichen ruber as leading to marasmus and death. Mr Hutchinson says that the large majority of his patients believed themselves to be in their usual health when it began, but that if it persists long the general health may fail.

The cases seen by the writer were in persons in average condition, some of them in robust health. No internal organ is affected, nor are the nervous symptoms or general disturbance above noticed anything but exceptional.

Lichen planus does not tend to spontaneous recovery; it continues indefinitely, may spread extensively, and, as above stated, may in certain severe and protracted cases seriously affect the general health. After being cured it is liable to recur.

Diagnosis.—This affection is distinguished from eczema by its never forming either vesicular or raw surfaces, by its avoidance of the face and ears, its distribution, and the comparatively slight amount of itching. In some cases it undoubtedly approaches very closely to psoriasis, especially to inveterate cases of the latter disease which have become generally diffused and have lost much of their characteristic appearance. Mr Hutchinson would recognise transitional cases, and indeed proposes to name lichen planus "lichen-psoriasis." But difficult as the diagnosis occasionally is, the distribution, the character of the scales, and the persistence of the papules sufficiently distinguish lichen planus from psoriasis.

Lichen ruber acuminatus is by many authorities distinguished from what Devergie called *L. ruber pilaris*. But when the same case receives the two names from equally eminent authorities, as it has in several instances, they must be identical. Those, however, who unite them would, in order to do so, separate Lichen acuminatus further from Lichen planus than seems to be at all necessary.

Pityriasis rubra pilaris is a most unfortunate name, for the affection has no alliance with "*Pityriasis simplex*" (branny desquamation after der-

matitis), *Pityriasis rubra* (Exfoliative dermatitis), or *Pityriasis pilaris* (Keratosis pilaris). The real question is whether what French writers still call *P. rubra pilaris*, and German writers *Lichen ruber acuminatus*, is the same as Lichen ruber of Hebra and of many English and American writers.

A most important distinction is between lichen planus and syphilis, for which it is often mistaken. The colour, the frequent absence of itching, and the somewhat irregular distribution, lead to this error, which is apt to be confirmed if white patches are found on the tongue or cheeks. This leucoplakia, however, is no proof of syphilis. The colour of lichen ruber is more purple and less brown than that of a syphilide. The freedom of the face and scalp, the absolute uniformity of all the lesions, and their persistence unchanged during long periods of time, usually ensure a correct diagnosis; but some cases which conform to Hebra's type (*L. ruber*) rather than to Wilson's (*L. planus*) are so much like a secondary scaly eruption, that the present writer must confess that he has more than once mistaken the one for the other.

Treatment.—The treatment of lichen (planus or acuminatus), both in Germany and England, is chiefly by arsenic. Most writers speak of this as specific and certain in its effects, but like other certain remedies it sometimes fails, and lichen planus is certainly slower in yielding to the remedy than most cases of psoriasis. Locally, tar ointments or some of the milder preparations, which will be described under psoriasis as substitutes for tar, are important aids in treatment, and sulphur ointment has been also found useful. In obstinate cases an ointment of pyrogallic acid sometimes succeeds. Unna recommends one composed of hydr. perchlor. gr. $\frac{1}{2}$, ac. carbol. gr. xx, and ung. zinci $\bar{5}$ j. Dr Heitzmann, of New York, who tried this treatment, found a 3 per cent. solution of carbolic acid much more efficient ('Journ. Amer. Derm. Soc.,' Sept., 1889, and *ibid.*, pp. 52 and 64). To check the itching in severe cases quinine, phenacetin, chloral, and full doses of henbane are of service, and locally a hydrocyanic acid lotion.

Parakeratosis variegata.—This name was given by Unna to what was probably correctly described by Erasmus Wilson as "a reticular variety of Lichen planus." Three cases were shown by Dr Jamieson at the dermatological section of the British Medical Association at Edinburgh in 1898, which he regarded as aberrant Lichen planus, but Unna recognised as identical with those which he had described under the above title.* The condition is probably allied to, if not identical with, a case in a woman of sixty named *Erythrodermie pityriasique en plaques disséminées* by Brocq in 1897, and two similar cases recorded by Dr White, of Boston, in 1900.† The peculiarity which led to such epithets as "reticulated," "variegated," and "spotted like a leopard" is a marked dull reddish network over the body like the mottled, livid appearance of the arms and legs in very cold weather, or the hæmorrhagic stains from the reticulated cutaneous veins seen in the deadhouse in the corpse of a patient who has died of acute dyspnoea. The locality is sometimes confined to the palms and soles where there is great thickening of the epidermis; sometimes the disease spreads over the trunk as a papular eruption, very chronic, and without itching or

* One of them, however, subsequently developed tumours like granuloma fungoides, a result which had been acutely foreseen as a possible event by Dr Crocker and Mr Malcolm Morris at the meeting.

† These cases and reference to others will be found reported in an admirable monograph by Dr Colcott Fox and Dr Macleod, in the 'British Journal of Dermatology' for 1901, p. 319.

pain. Dr McLeod's observations show that the disease is not histologically identical with Lichen planus, and is not a Parakeratosis as Auspitz or even as Unna defines it. Dermatitis variegata chronica would perhaps be a better term for the present than either Unna's name or Crocker's Lichen variegatus.

PRURIGO.—Prurigo, "the disease attended with pruritus or itching," was a term formerly very loosely applied, and is still somewhat difficult to define. Willan described it as a papular eruption in which the papules are of the same colour as the skin and accompanied by itching. His "species" were *P. mitis* and *P. formicans*, which are merely more or less severe cases of the same affection, and *P. senilis*, characterised by the age of the patients and the difficulty of cure. Bateman thinks that pediculi are not unfrequently generated when prurigo senilis is present, thus putting the cause for the effect, since it is now well ascertained that most, if not all, cases of prurigo senilis are directly caused by pediculi corporis (*supra*, p. 855).

Willan and Bateman also mention *Prurigo pubis*, which they rightly ascribe to the presence of pediculi (p. 854), and *P. præputii* and *urethralis*, which are both sympathetic pruritus. Lastly, their species *Prurigo podicis* and *P. pudendi* correspond to the drier and more papular and indurated forms of the irritable local dermatitis which was described in the last chapter as eczema ani and eczema genitalium (p. 845).

Pruritus.—Prurigo, a papular inflammation of the skin, must be distinguished from pruritus or subjective sensation of itching without any local lesion. Pruritus accompanies not only prurigo but also eczema and the desquamative stage of many exanthems. It is the constant effect of pediculi and of the acarus; it may be produced by jaundice, and may be the result of the various atrophic changes which take place in the senile skin. These, which have been well described by Neumann, include the wasting and ultimate disappearance of the papillæ, and it is probable that the process gives rise to senile pruritus without pediculi.

Irritable papules may be produced by primary pruritus. This occurs in hot weather among children, most often from the irritation of sweat or of vermin (the *Lichen urticatus* of Bateman). The papules are large, flat, and discrete; but there is no pigmentation, no thickening of the skin, and the distribution is irregular. Mr Hutchinson has observed this summer pruritus affecting the face and arms of adolescents and relapsing every year (*Pr. æstivalis*). According to this author, another form of prurigo is common as a sequel of varicella. Many cases of erythema, particularly bullous forms, recur every summer.

Some persons, usually young adults, are liable in cold, frosty weather to great irritation of the skin, which becomes dry, harsh, and pale. This affects the covered parts most, and is often supposed to be due to flannel underclothing. It has been called *winter prurigo* by Hutchinson, and *pruritus hiemalis* by Duhring; but in England is more common during the east winds of spring than in midwinter. The scratching of the patient produces a crop of papules. Dr Payne has published several cases of this disorder ('Rare Diseases of the Skin,' chap. iv). He recommends unmedicated glycerine or vaseline locally, and chloral as a sedative at night.

Many cases formerly described as prurigo should be called papular erythema, or urticaria, or papular dermatitis—arising from the irritation of

bugs or pediculi (which in infants do not cause the characteristic appearance of prurigo pedicularis), or from the friction of flannel next the skin.

Prurigo.—True prurigo is quite independent of pediculi. One form is the prurigo of Hebra, a striking description of which is given in the Sydenham Society's translation of his work (vol. ii, p. 258). He admits milder cases which correspond to the prurigo mitis and formicans of Willan, but would separate them broadly from the severe form, which is congenital and incurable. The writer saw at Vienna cases of this "Hebra's prurigo," as it has been called, and ventures to think that their characters were somewhat over-described, if not exaggerated. At all events cases have been described, both in America and in England, which agree with Hebra's cases in all essential particulars, and which would make an uninterrupted series connecting the worst of those in Vienna with the slightest forms of infantile prurigo.* We may therefore fairly include these affections under a common name, using such adjectives as *mitis*, *gravis* or *agria*, *congenitalis*, *infantilis*, *inveterata*, to denote the varieties which we find in practice.

The papules of prurigo are at first scarcely distinguishable in colour, and, as Hebra says, are felt rather than seen. They are not closely set, and do not appear in patches; they produce great itching, which causes black spots and scratch-marks, as in so-called prurigo pedicularis. The skin between is more or less pigmented, and is generally covered with a fine branny desquamation. In course of time it becomes thick and indurated, and in many cases there is traumatic eczema, often pustular from infection through the nails. In severe cases, secondary indolent enlargement of the lymph-glands occurs both in the groins and the axillæ.

The distribution of prurigo is over the trunk and limbs. The face is almost always free, and also the flexures of the joints, palms, and soles. It is generally most severe on the back, chest, and abdomen, on the buttocks, the shoulders, and upper arms; and it is generally worse on the lower than on the upper extremities, and worst of all below the knee. It scarcely affects the face or scalp, nor yet the hands and feet.

Prurigo begins in early life, and either disappears during childhood, or if present in an adult, has persisted from that period. It is generally worse in winter. In one case of the writer's it began in a lad at the age of fourteen. It first appeared on his legs, and affected the whole surface except the head, palms, soles, and flexures. There were a few spots on the cheeks and neck, on the hands and feet, and on the penis; the trunk was moderately affected, the buttocks and thighs more so, and the arms and legs most of all. There were severe buboes, and he was thin and wasted. He improved greatly under treatment, but the disease returned from time to time.

In twenty consecutive cases observed by the writer, the ages of the patients when first seen were—under twelve months 2, between two and five years 8, between five and ten 1, between ten and fifteen 5, between twenty and forty-five 3. It is more common in men and boys than in females. In long-standing cases the skin is often much thickened as well as pigmented.

The *treatment* of prurigo, even in its most typical and severe form, is far from being as hopeless as Hebra supposed. Frequent warm baths and assiduous inunction, together with arsenic internally in steadily increasing doses, with cod-liver oil and good feeding, will often restore even inveterate cases to health and comfort. It is, however, almost certain to return, pro-

* See, on this point, a paper by Mr Marrant Baker, 'Internat. Med. Congr., 1881,' vol. iii, p. 177, and the discussion which followed.

bably more than once, and must be kept at bay for years before it finally disappears.

The slighter forms of true prurigo in infants and children are very much aggravated by scratching, and the first point is to prevent this by hydrocyanic lotion or other local anodyne, and by sedatives at night, as described under eczema (pp. 835, 840). In some cases quinine appears to be almost a specific, both for the irritation and the disease.

Locally ointments are often more effectual than lotions in allaying the itching. *Ung. hyd. ammoniati* or *Ung. hyd. ox. rubri* is particularly useful for circumscribed regions, as in pruritus ani. *Liq. picis carbonis* (3j—ij ad 3j) is often serviceable in addition. Of local anodynes cocain is more generally efficient than belladonna or calomel ointment. All mercurials must be reserved for local use, for if at all extensively applied salivation may be the result.

PSORIASIS

(*DRY OR SCALY TETTER*)

“Duratæque cuti squamas increscere sentit.”—OVID.

Frequency—Name—Anatomy and histology—Course—Symptoms—Distribution—Ætiology—Varieties: guttate form, inveterate form—Relation to eczema, to lichen, to pityriasis rubra, and to syphilis—Prognosis—Treatment.

EXCLUDING scabies and syphilis, by far the most common cutaneous disease is eczema; next comes acne, and then psoriasis. Like the affections hitherto described, it is a chronic superficial dermatitis, and like them has been described as a dartrous or herpetic affection. It stands, however, at the opposite extreme from typical idiopathic vesicular eczema, with which it offers points of contrast rather than of resemblance.

An old and good name for psoriasis was “dry tetter.” The Greek term signifies the condition of *psora* or itching, and has no bearing on the present signification of the term. Certain forms of psoriasis were formerly known as *lepra*, a term which from its etymology, “the scaly disease,” would be more appropriate, but the word *lepra* is definitely attached to one form of skin disease which is totally unconnected with psoriasis, the terrible disease of Leprosy. The Greek term *alphos*, referring to its white scales, was revived by Erasmus Wilson, but without general acceptance.

Anatomy.—Psoriasis is an extremely well-marked and characteristic form of disease. It begins as papules, which rapidly increase in size, and form flat patches. From the beginning white scales can be seen on the papules, and by the time they are as large as a pea the scales form conspicuous shining white spots (*Ps. guttata*). They are large, perfectly dry, strongly coherent, and not easily separable from the skin; they have also a characteristic white silvery lustre, due to the abundance of air which is included between the layers of horny epidermis. When the scales are removed, the surface on which they rest is seen to be red, shining, and dry, but dotted over with red points which occasionally bleed. The injection is not that of acute hyperæmia, and either stops at the edge of the scaly patch or only extends very slightly beyond it.

Local evolution.—From this punctiform or guttate origin, the disease spreads, partly by fresh spots appearing, but also by gradual enlargement of each lesion into a separate disc with well-defined margin (*Ps. nummulata*). These, as they increase in size, become confluent with the originally diseased surface and with each other, so as to form figure-of-eight patches.

The upper and lower extremities or the back may thus be covered; but there will always be found more or less extensive islands of healthy skin between the diseased parts, and these will have a concave, while the scaly patches have a constantly convex, outline.

As the raised, red, and scaly edge of the eruption advances, the inner parts, which were first affected, lose their scales and return, more or less incompletely, to a healthy condition; so that by this progressive spread and involution of the disease the scattered scaly patches in which it began gradually give place to extensive surfaces of almost normal appearance, bounded by sinuous lines of red and scaly skin, made up by the intersecting segments of many circles. *Psoriasis gyrata* was the technical term applied to this stage.

After psoriasis has lasted for some time, its colour begins to acquire a deeper and brownish tint. It no longer disappears completely upon pressure,—that is to say, pigmentation has been added to hyperæmia. In inveterate cases this becomes very characteristic, the colour being of a deep brown, sometimes almost mulatto tint. When the disease has been cured, when the scales are removed, the hyperæmia has subsided, and the finger cannot feel anything but healthy skin, dark pigment-blotches remain to attest the nature of the recent malady. They always disappear in time, but, especially in old persons, their disappearance is slow. It may be said that, next to syphilis, psoriasis produces pigmentation more quickly than any other form of dermatitis, and the depth of pigment may be as great as in the most chronic cases of eczema or of prurigo senilis. In this, as in other respects, psoriasis resembles lichen planus, and differs from pityriasis rubra.

Histology.—The earlier dermatologists, Gustav Simon, and even Hebra, were unable to prove what they recognised as probable, that psoriasis is essentially a form of dermatitis. As with eczema and most other cutaneous affections, the characteristic appearance is lost after death; but Neumann established the existence of abundant cellular infiltration of the papillæ of the corium, extending along the tracks of blood-vessels in its deeper layers. The papillæ are enlarged to ten or twelve times their natural size, and this papillary hypertrophy is present from the first, not only, as with eczema, in the later stages. The scales of psoriasis, like those of pityriasis rubra, consist almost entirely of keratin—unmixed with fibrin and leucocytes, as in chronic eczema and syphilis, or with sebum, as in pityriasis capitis.

Dr Liveing long ago detected exudation between the scales of psoriasis, and by careful staining leucocytes may be recognised in the deeper layers of the epidermis, so that formally psoriasis is like eczema and lichen, an irritative dermatitis. The horny transformation of the epidermic cells is less complete than in health; they are thick and soft and show a nucleus longer,—that is, the process of cornification is accelerated and imperfect.

Psoriasis was included by Auspitz with Lichen, Pityriasis rubra, and some forms of Eczema under the title *Parakeratosis cutis*; but the histological condition of imperfect corneous metamorphosis of the epithelial cells appears to be present in other conditions and not to be constant in psoriasis.

The fact of hypertrophy of the epidermis is obvious, the evidence of irritative hyperplasia with scanty exudation of leucocytes in the rete and papillary layer is clear; but the origin of the process is undiscovered. Much interest, therefore, attaches to the statement by Dr W. J. Munro, of Sydney, who, working in Sabouraud's, came to the conclusion that the initial lesion of psoriasis is the presence of minute collections of leucocytes—"dry ab-

scesses," as he calls them—in the horny layer of the epidermis ('Brit. Journ. of Derm.,' vol. xii, p. 63).

Course.—Psoriasis is never acute. Even when it develops rapidly it is unaccompanied by the ordinary symptoms of inflammation, and never causes disturbance of other organs. Often a patch on each elbow, or on the elbows and knees, may appear and remain for years before it shows signs of spreading. When it has become extremely diffused and passed through the centrifugal process above described, it will, if untreated, enter upon a very chronic and almost interminable course, the skin being habitually thick, harsh, and dry, and the general aspect resembling that of some of the forms of dry scaly chronic eczema in old persons.

Of all skin affections psoriasis is most prone to *recur*, more so even than eczema. In fact it is very rare for only a single outbreak to occur. Sometimes, when the eruption has only just disappeared under treatment, a fresh attack comes on, and the very means which will almost infallibly cure it when developed are often powerless to prevent its return.

Notwithstanding its etymology, psoriasis is not often very irritable. Sometimes there is no itching at all, and when present it is much less severe than in eczema, scabies, or prurigo. It is still more rare for the affected parts to smart or to feel hot and tender. Though pathologically it is an exudation, it is the most chronic, cold, and uninflamatory of all kinds of superficial dermatitis.

It produces no constitutional effects, and persons subject to it are entirely free from special liability to any other disease. The digestive, urinary, and other functions are carried on as usual, unaffected by the condition of the skin. There is, however, frequently, if not constantly, a moderate degree of leucocytosis.

Distribution.—Psoriasis is no less characteristic in the regions it affects than in its anatomical lesion. Its favourite spots are over the olecranon process of the ulna and over the patella, ligamentum patellæ, and tubercle of the tibia. In fact, it is remarkable how very rarely these spots are found free even in the most chronic and varied forms of the disease. Here it begins and here it almost always remains.

Psoriasis is of all diseases the most completely and constantly symmetrical; not more so, it is true, than typical forms of eczema, but its range is so much more restricted, and its varieties so unimportant, that while typical eczema does not include three fourths of the whole number of eczematous cases, we seldom meet with one of psoriasis which deviates from the characteristic type. As above stated, its favourite or almost its constant seat is upon the two elbows and the two knees; next it is common over the whole extensor surfaces of the extremities, specially the forearm, the front and outside of the thigh, and the peroneal side of the leg. Even when most extensive, it shuns the bend of the elbow and the popliteal space. It not unfrequently extends from the forearm to the back of the hand, and from the leg to the dorsum of the foot, and occasionally may cover the fingers, and even affect the nails.

Psoriasis unguium is known by the excessive and unsightly thickening of the nail, which is sometimes reeded or transversely grooved, and also by the absence of soreness and suppuration of the matrix. It sometimes occurs when the other fingers or toes are free from the disease, but almost always spots of psoriasis will be found on the elbows or knees, or the patient has previously suffered from the disease. The only other affections of the

nails which at all resemble it are eczema unguium, above described, and onychomycosis, to be mentioned under ringworm.

Psoriasis very rarely affects the palms or the soles, and never unless other parts of the body are previously the seat of the disease. What used to be described as primary psoriasis palmaris was probably either eczema squamosum or scaly syphilis. When present, the scales of palmar psoriasis are comparatively small, but the patches keep their well-marked edge. There is little or no disposition to form cracks, and the soreness and irritation which attend eczema of the palms or soles is absent.

Next to the extensor surfaces of the limbs, the trunk is the most common seat of psoriasis. The shoulders, back, and loins, the sacral and gluteal regions, are very frequently its seat, the chest and abdomen somewhat less so. Indeed, we never see psoriasis of the abdomen which does not also affect other parts of the trunk, and it is very rare to find psoriasis of the trunk when the limbs are completely free. The genital organs are occasionally the seat of psoriasis, which has usually spread thither from the abdomen or the thighs; but this is far less frequent than eczema of the same parts, and what used to be called *psoriasis scroti* is really *syphilis squamosa*.

The face and head are less frequently attacked by psoriasis than the trunk, far less frequently than the limbs; but with so common a disease cases often occur in which the red scaly patches appear on the neck, the forehead, or the scalp. The scales are usually smaller upon the face, the whole aspect less characteristic, and apt to be further confused by slight ordinary dermatitis, but the presence of unmistakable psoriasis on the limbs or trunk prevents any mistake in diagnosis. On the scalp the closeness of the hairs prevents the formation of large scales, and the sebaceous secretion gives them a greasy consistence and a yellowish tint. *Psoriasis capillitii* is a not infrequent affection, and must be carefully distinguished from eczema and impetigo capitis, from syphilodermia and from seborrhœa sicca or pityriasis capitis. It is always dry, the scales are coherent, the hair does not fall out, and it is coincident with existing or previous psoriasis of other parts. When psoriasis affects the face at all, it often has spread from the scalp.

Brocq describes a form of psoriasis, affecting the flexor aspect of the joints or the umbilicus, or the scalp, of a bright red colour with small yellowish scales (the scarlatiniform psoriasis of Bazin?). Such cases would probably come under the head of seborrhœic eczema.

The affection called *psoriasis labiorum* has been described already as a form of eczema. It is doubtful whether psoriasis ever affects the mucous membranes. *Psoriasis lingue* is *leukoplakia*, white patches on the tongue, distinct from syphilis and often antecedent to epithelial cancer; when it is coincident with an affection of the skin, that affection is usually lichen planus. The occasional co-existence of these patches with ordinary psoriasis must, however, be admitted.

Ætiology.—The cause or causes of psoriasis are absolutely unknown. By French writers it was ascribed to a dartrous diathesis, and by those who went still further into speculation a *dartrous* was distinguished from an *arthritic* psoriasis. In England also it is frequently regarded as *gouty*, while by some authors it is considered, especially in children, as a *scrofulous* disease; and there is about the same evidence for the one hypothesis as the other. Yandell supposed that much of psoriasis as well as of eczema was *malarial* in origin; and in the eighteenth century psoriasis was by

some surgeons considered undoubtedly *scorbutic*. Other writers have speculated on the possible connection of psoriasis with *leprosy*, and would have us regard it as the gradually mitigated manifestation in modern times of the scaly leprosy, "white as snow," which is described in the Old Testament.

The remarkable centrifugal progress above described naturally suggests the idea of a parasitic vegetable growth; but no fungus is present, and although it has been asserted that *bacteria* (*e. g.* Unna's *morococci*) may be occasionally found in the affected skin when the scales have been removed, their presence is neither constant nor specific. So unwelcome is it to admit a disease without a cause, that some dermatologists refuse the title to psoriasis, and regard it as a condition of health.

With respect to gout it must be remembered that the word, in Germany and even in England, is often applied upon very insufficient evidence; and sometimes does not imply the presence of urate of soda in the joints. Moreover, the diagnosis of gout is always acceptable to an Englishman of the middle class. On the other hand, it is true that few families of rank in this country are free from unmistakable gout in some of their members. But psoriasis is comparatively rare in private practice as compared with that of hospitals. Very few of those who have unmistakable podagra are liable to psoriasis; and psoriasis is as common in Scotland, Germany, and America, where gout is rare, as in England, where it is frequent. If we determine that every disease must have for cause some condition already known, it will be easy to find one in the list given above for every case of psoriasis; but such a practice hinders the progress of knowledge of the real causes of disease, interferes with rational and successful treatment, and leads to an acquiescence in unsupported statements and fallacious arguments which is fatal to medicine either as a science or an art. Psoriasis is a disease of the skin, a kind of malformation of the epidermis which is not associated with lesions of other organs, which is compatible with a condition otherwise healthy, and of which we at present do not know the origin.

Sex and age.—Psoriasis occurs equally in both sexes and at all ages above infancy; it becomes more common from the age of six or seven up to puberty, and the first attack usually falls in childhood or early adult life. It may, however, begin after fifty and even in old age. It is never congenital.

The present writer published in the 'Guy's Hospital Reports' (vols. xxv, p. 243, and xlvi, p. 419) a tabulated statement of 267 consecutive cases of psoriasis observed by him. Of these patients, 145 were men, and 122 women. Their ages varied from four to seventy-two, but in many cases the disease has begun long before the patient appeared. There were 22 patients under ten years old, 35 between ten and sixteen, 32 between sixteen and twenty, 66 between twenty and thirty, 23 between thirty and forty, 25 between forty and fifty, 23 between fifty and sixty, 22 between sixty and seventy, and 3 over seventy.

Varieties and diagnosis.—As above stated, psoriasis compared with eczema is singularly uniform in its anatomy and natural history. The description above given applies to ninety-nine out of a hundred cases, of course with individual variations, but less than those of most diseases. There is, in fact, only one variety which demands separate notice: in *children* the ordinary form is frequently seen, but more commonly the early spots of *Psoriasis guttata* do not grow into the nummular stage, and the large patches of gyrate psoriasis are decidedly rare under puberty.

This would be scarcely worth mentioning in itself, but the spots are also remarkable for having little or no red border. They produce no irritation, and—as is common with other diseases affecting children—the local distribution is less rigidly marked than in adults. The guttæ are frequently seen on the face, and they are perhaps more abundant on the trunk than on the limbs. Dr Liveing thinks, moreover, that guttate psoriasis occurs particularly in tuberculous children. It is, however, often seen in those who are robust, and certainly in most cases there is no chronic enlargement of the lymph-glands, no caries, no chronic synovitis, or evidence of tubercle.

When psoriasis has lasted for many years and has spread over the greater part of the surface, it loses much of its characteristic appearance; the scales are less abundantly formed, the margins are less definite, and the whole skin becomes thickened and indurated, so that it often requires careful investigation and a knowledge of the earlier stages of the affection to distinguish this *psoriasis inveterata* from the dry and chronic eczema described in a former chapter.

There is no question that psoriasis may pass into, or be supplanted by, the dry, scaly, and universal dermatitis to be described in the next chapter as pityriasis rubra. As is there stated, the late Dr Baxter thought that any dermatitis—eczema, psoriasis, lichen, or pemphigus—might, if sufficiently extensive, assume the characters of that remarkable disease. In a paper in the 'Guy's Hospital Reports' (vol. xxv, p. 266), reasons are given against accepting this hypothesis. The writer had a remarkable case in which a woman, who had been in St Thomas's Hospital under Dr Payne with ordinary psoriasis of the elbows and knees, and whose daughter was a patient of his own, also with psoriasis, came under his care with marked and typical pityriasis rubra. It is very probable that some at least of the cases of general psoriasis, described by Hardy as very rare, would have been recognised by Devergie as pityriasis rubra.

One may admit that eczema and psoriasis, which in so many ways are allied by points of contrast, have connecting links on the one hand with pityriasis rubra, or universal exfoliative dermatitis, and on the other with lichen planus, which, as we saw, may closely resemble psoriasis, while by its relation to other forms of lichen it has affinities with papular eczema. We draw lines as nearly as we can in accordance with the broad demarcations of pathology and natural history, but here, as in other departments of medicine, it would be pedantry to deny that there are transitional forms which it is difficult or perhaps impossible to classify.

The important question, however, of the diagnosis between psoriasis and the scaly forms of syphilis is one which rests on the absolute distinction of cause, and is, therefore, of the utmost practical importance. The locality and symmetry, the character of the scales, the colour, the presence of itching, the uniformity of the lesion, and the absence of other signs of syphilitic disease are the points to be attended to. The last, however, may deceive, for a man with psoriasis may acquire syphilis, as he may scabies; and the writer has notes of four or five cases in which true psoriasis and a secondary syphilitic eruption existed in the same patient, ran independent courses, and were cured by different treatment.

Prognosis.—Psoriasis, if left to itself, lasts for an indefinite time, though almost always getting better or worse at intervals. It never interferes with the health or affects other organs than the skin. After being cured it is of all cutaneous diseases most apt to return.

Treatment.—The external treatment of psoriasis consists in inunction of some preparation of tar. Nothing is more effectual than the *unguentum picis liquidæ* of the Pharmacopœia well rubbed in at night, and allowed to stay on while the patient sleeps in a special suit of underclothing; it may then be washed off in the morning, to be reapplied at night. When the scales are very thick and indurated, it may be well to precede this application by the use of hot baths and soft soap. When the smell and colour of tar are objected to, useful though less efficient substitutes may be found in the liquor picis carbonis, made into an ointment with lanoline (one, one and a half, or two drachms to the ounce), or in the *huile de Cade*, ung. creasoti, oleum rusci, &c. Another plan of obtaining the same result is to apply a spirituous solution of tar or the liquor picis carbonis diluted with water. Goa powder, and the chrysophanic acid or chrysarobin which it contains, are powerful cutaneous stimulants, and have been often used with success in the treatment of psoriasis. They occasion, however, with many patients considerable pain, and stain the skin and linen unpleasantly, as well as the hands of the person applying them.* In cases where tar is inadmissible, another substitute is pyrogallic acid, gr. xv—xxx to an ounce of benzoated lard or lanoline, the strength being increased with caution. For rapidity of cure, with freedom from unpleasant smell, this is an efficient application, but it stains like chrysarobin. Naphthol and thymol are more elegant and sometimes very useful.

Beside the local treatment, it is almost always desirable, after the scales have been thus removed, to put the patient upon a course of *arsenic*. It is usual to prescribe it in a bitter infusion, but it will be found to agree quite as well, and to be more constantly taken, if merely diluted with water or flavoured with syrup or peppermint. It should always be taken at or immediately after a meal. Three, four, or five drops of Fowler's solution in an ounce of water three times a day is the dose to begin with, and it may be increased to ten or beyond. If properly diluted, and taken with food, even full doses seldom cause pain, sickness, or diarrhœa. The first sign of the physiological limit being reached is usually irritation and slight injection of the conjunctiva. As soon as the patient feels his eyes begin to itch, he should be instructed to leave off his medicine for a couple of days, and then resume it in slightly smaller doses. He has reached what is for him the full therapeutical benefit.

Psoriasis may often be cured by arsenic without any external application whatever, or by local treatment, without internal medication; but in most cases the cure will be hastened by the application of tar, and will be rendered more permanent by the administration of arsenic.

In the case of anæmic persons it is desirable to give steel. We may then combine the liq. arsen. hydrochlor. with the liq. ferri perchlor. In other cases Fowler's solution acts better alone. It used to be the fashion to administer Pearson's solution (the arseniate of soda) to children, but there is little doubt that it is absorbed in exactly the same form. When arsenic

* Dr Crocker writes ('Brit. Med. Journ.,' November 19th, 1887): "The stains in linen are quite indelible without injuring the fabric, but they may be avoided by using the Auspitz method. A gutta-percha varnish, called traumaticin, is made by dissolving ʒj of pure gutta percha in ʒx of chloroform, ʒj of chrysarobin is added, making an emulsion, which is painted on with a stiff brush after removing the scales every day, until a thick coat is formed; it is then allowed to peel off and is renewed. It acts effectually and does not stain. Besnier modified this by brushing in ʒj of chrysarobin in ʒx of chloroform, and then varnishing with traumaticin. Both plans are equally good."

disagrees, it should not be hastily given up, but the dose should be diminished until unpleasant effects no longer follow; or we may sometimes prevent them by adding a few drops of laudanum or a little compound tincture of camphor to the dose. When the patient suffers from gastritis and sore eyes, four drops of Fowler's solution—three, or even two—will probably be sufficient to cure the psoriasis; when a patient can take ten without discomfort, it may be that fifteen will be needful.

Children take arsenic well and in full doses. When they are pale and thin and ill-nourished, cod-liver oil is often a useful coadjutant. In the guttate form of the disease, most common under puberty, local treatment is often scarcely required.

Purgatives and diuretics are quite unnecessary; and colchicum is not needed, as it undoubtedly is in the treatment of certain irritable and probably gouty forms of eczema.

In some cases psoriasis is very obstinate, not only returning again and again after being cured, which is common, but yielding very little to the most careful local measures and to the most persevering use of arsenic. In these cases liquor potassæ in half-drachm doses sometimes succeeds, sometimes iodide of potassium. The latter salt has been advocated by Dr Cæsar Boeck and other physicians in Norway and Denmark. Dr Haslund gives enormous doses without harm (see '*Brit. Med. Journ.*,' 1888, vol. i, p. 27). The writer has seen two or three obstinate cases (one in particular that had resisted arsenic from childhood upwards) which yielded to full doses of iodide. But we occasionally meet with cases which seem to be quite incurable. These have been treated with thyroid extract, administered externally, and apparently with some benefit. Mr Brooke, of Manchester, has shown cases of this kind; but the writer has seen more failures than successes, and the treatment is not an indifferent one.

Other drugs recommended in psoriasis are oil of turpentine, carbolic acid (one drop in a pill), sodium salicylate (recommended by Dr Crocker in early and acute cases), and antimonial wine (recommended by Mr Malcolm Morris in the same condition of the disease). Natural warm baths, indifferent, alkaline, or saline, act only by soaking off the scales, and are inferior to hot soap and water baths at home.

PITYRIASIS RUBRA

(UNIVERSAL EXFOLIATIVE DERMATITIS)

“Which, like a searching tetter uncorrected,
Left no part of his body unaffected.”

QUARLES, *Job Militant*.

Name and traditional species of pityriasis—Pityriasis rosea vel circinata.
Pityriasis rubra—Exfoliative or desquamative dermatitis—History—Accounts by
Devergie, Hebra, Wilson, Hutchinson, Baxter—Course and symptoms—
Histology—Prognosis—Diagnosis—Treatment.

THE word “Pityriasis,” meaning, as its etymology implies, a branny or furfuraceous desquamation of the skin (πίτυρα), is conveniently used to describe that condition, but no one disease is entitled to the name.

The species defined by Bateman as *Pityriasis capitis* is in most cases *Seborrhæa sicca*, an affection of the sebaceous glands of the scalp; or it may be slight local dermatitis (*Eczema capitis*), often due, as he remarks, to want of cleanliness, and removable by soap and water, but apt, if neglected, to degenerate into “Porrigo,” that is to become pustular eczema or impetigo of the scalp. Or, again, pityriasis capitis may be a slight form of psoriasis capillitii.

Pityriasis versicolor, now known as *tinea versicolor*, is caused by a parasitical fungus, and will be described in a subsequent chapter.

Pityriasis nigra, described by Willan as occurring in children born in India, was not identified by Bateman, nor probably by anyone else. A case of Alibert’s, which Devergie calls “pityriasis nigra with prurigo,” was apparently *Prurigo pedicularis* with pigmentation and leucoderma.

Bateman’s fourth species, *Pityriasis rubra*, “resembling psoriasis diffusa,” is a stage in the involution of eczema.

Pityriasis rubra pilaris is an unfortunate name for a condition affecting the hair-sacs, which is closely allied to, if not identical with, *Lichen acuminatus* (*supra*, p. 863).

Pityriasis rosea was the name given by Gibert, in 1860, to a trivial and somewhat rare affection of the skin which he regarded as erythema. It has been named by other French writers *Pityriasis rubra maculata*, or *P. marginata et circinata*. Hebra and some other authors regarded it as a form of ringworm of the body, and named it *Herpes tonsurans* (*Tinea ton-*

surans) *maculosus*, but for this opinion there is no adequate evidence. Dr Liveing calls it *Roseola circinata*.

At present its position among other forms of superficial dermatitis is doubtful, but its course and natural history is like an erythema. It has no pathological relation to Pityriasis rubra.

It begins as pale rose-coloured patches very slightly raised above the surface, which rapidly extend so as to form fresh discs, then circles, and, lastly, gyrate bands. In this respect it resembles psoriasis as well as ring-worm. As the border advances, the central part fades into a yellowish stain (*P. maculata*) and a slight branny desquamation ensues which gradually disappears.

The favourite locality of this eruption is the trunk, particularly the chest or abdomen; but it sometimes spreads to the neck and face, the buttocks, and the proximal part of the limbs. It is not symmetrical in distribution, and its course is usually rapid.

It is often seen in children. It is not contagious, nor associated with rheumatic fever or chorea, nor complicated by urticaria, nor does it seem caused by external irritants or by internal toxic agents.*

It usually passes away of itself after a few weeks, and needs little or no treatment, for the irritation is but slight.

It has a certain resemblance to *Lichen circumscriptus vel circinatus*, to *Tinea versicolor*, to *T. corporis*, and to an early syphilitic rash. From each of these it is separated by its course, the absence of fungi, and the absence of polymorphism and other characters of lues. Its locality also is quite different from Hebra's *eczema marginatum* and from *seborrhœic eczema*. The slight desquamation which follows distinguishes it from *erythema papulatum*.

PITYRIASIS RUBRA — Exfoliative dermatitis.—The term "pityriasis rubra" was unluckily applied by Devergie in 1854 to a severe and remarkable form of superficial dermatitis which certainly deserves a special name.† It is probably identical with Alibert's "*Herpes squamosus*." Wilson's proposed names of "*Pityriasis foliacea rubra*" and "*Eczema foliaceum*" (1876), or the better title, "*Exfoliative dermatitis*" (1870), have not displaced the original term. *Universal exfoliative* (or *desquamative*) *dermatitis* is perhaps the description that would most clearly express its characters until its essential origin and relation to other forms of superficial dermatitis have been ascertained.

History of the disease.—Devergie (*Traité pratique des Maladies de la Peau*, p. 263) described the disease under the title *Pityriasis rouge aiguë*, as beginning with an erythematous redness, usually on the chest or flexor surface of the limbs, and spreading rapidly, with a well-defined margin, deep colour, abundant scales, and more or less thin serous discharge. He adds that it covers the whole body, is very obstinate, lasting for months, and occasionally proves fatal by exhaustion and diarrhœa; but that, as a rule, the patients slowly recover, though relapses are frequent. Devergie admitted the difficulty of distinguishing his new disease from *eczema*, and bases the

* A microsporon has been described by Vidal, who proposed the name *Pityriasis anomœon*, but it is not constantly present, and is not accepted as specific by Besnier and Doyon.

† The case for which the name pityriasis rubra was first proposed (by Cazenave) seems to have been *Tinea versicolor*, with more irritation than usual. Besnier uses the term *Erythrodermie exfoliante*, surely a needless variation.

diagnosis on the following points:—It is of a deep red colour, it has sharply marked borders, it may affect the whole surface; the skin, and even the subcutaneous fascia, are thickened; it is less itching, more burning than eczema; its secretion is thin, and does not stiffen linen; the scales are abundant, readily detached; and from the first no red moist points (*état ponctué*) are seen when the scales are removed.

In the 'Glasgow Medical Journal' for January, 1858, p. 421, Dr McGhie recorded a case of "pityriasis rubra acuta," which he rightly regarded as one of Devergie's disease. This seems to be the first published in this country, and preceded Hebra's cases. The same patient's condition was described by Professor Gairdner ('British Medical Journal,' March 13th, 1875, p. 359) seventeen years later. Among the earlier cases may be mentioned one by Dr Wilks in the 'Guy's Hospital Reports' for 1861, which he called "general dermatitis;" the eruption was universal, red, and dry, with abundant desquamation. Another was recorded by the late Dr Hillier ('Handbook of Skin Diseases,' p. 101) in 1864, and another by Dr Fagge in the 'Guy's Hospital Reports' for 1876, vol. xiii.

Hebra had independently observed cases which were certainly the same disease as the pityriasis rubra of Devergie, and described them under the same title in 1860, but very properly protested against the adjective acute.

Some authors have regarded pityriasis rubra as a form of Eczema, and one of Sir E. Wilson's titles was Eczema exfoliativum. Dr Liveing agrees with Wilson and Fagge in looking on it as only a peculiar form of eczema. Mr Hutchinson ('Lectures on Clinical Surgery,' part i) would separate pityriasis rubra from eczema, and regard it as the type of a group of affections which differ in anatomy, but agree in being universal, in resisting treatment, and in often proving fatal. This would include *Pemphigus foliaceus* with certain cases of psoriasis and lichen.

The late Dr Baxter had previously published a valuable paper on this disease under the title of "General exfoliative dermatitis" ('British Medical Journal,' July 19th, 1879). He considered the affection as the result of a universal inflammation, and as arising by the general diffusion of either eczema, psoriasis, lichen, or pemphigus. The objections to this view are that eczema, and less often psoriasis, may be nearly universal, and for long periods together without losing its characteristic features, and without endangering the health. The same appears to be true of *Lichen planus*, if we accept Hebra's descriptions of universal chronic *Lichen ruber*; for this he carefully distinguishes from pityriasis rubra. *Pemphigus foliaceus*, though general, is seldom universal, and differs markedly, as will be seen hereafter, from pityriasis rubra. That the whole skin may be occupied by a scaly eruption without interference with health is proved by many cases of ichthyosis.

Auspitz, who is followed by Hans von Hebra, separates pityriasis rubra from the inflammatory diseases, and places it among affections of the epidermis (keratonoses) as *keratolysis*, a somewhat arbitrary decision.

What is unquestionable is that though sometimes appearing on a healthy skin and rapidly spreading over the whole surface, pityriasis rubra is not always primary. It frequently develops out of dry scaly psoriasis, and occasionally out of moist weeping eczema, and in each case gradually loses its original anatomy and distribution, and assumes the characters of primary *P. rubra*.

Origin, course, and characters.—However it begins, pityriasis rubra

rapidly spreads over the trunk and limbs, but in an irregular fashion, unlike the gradual and methodical extension of eczema or psoriasis.

Finally, it affects the whole of the cutaneous surface, including the scalp, the palms, and the soles. The skin is of a full deep red colour, not thickened and indurated as in chronic eczema (the exact reverse of Devergie's statement), covered with profuse and abundant scales, which are large, thin, and easily detached, unlike those of psoriasis, or of syphilis, or the branny desquamation which follows eczema and the exanthems. They are apt to form successive undulating ridges, which Wilson compared to those of the "ribbed sea sand;" and they are so abundant and so loosely attached that the patient's bed is filled with them by the peck.

In most cases the surface is absolutely dry; occasionally there may be a slight inflammatory exudation, especially in the flexures, where the inflamed skin is apt to crack. This exudation has not the stiffening property which marks that of eczema, and it is clearly an accidental, not an essential character of the disorder.

There is more or less pyrexia and general disturbance of health, especially at the onset. If, as is most frequently the case, the disease becomes chronic and inveterate, albuminuria is occasionally observed, and the appetite and health begin to fail. The irritation varies in different cases; sometimes it is very slight, but more often it is considerable, and occasionally almost as intense as in eczema, so as seriously to interfere with sleep.

In a certain number of cases there is Bright's disease present, usually in the most chronic form, and this makes the prognosis graver. But the combination is not so frequent as to throw much light on the pathology of exfoliative dermatitis.

Histology.—In a case of a year's standing examined after death by Hans von Hebra, the whole of the cutis was filled with leucocytes; in another case, which had lasted several years, all signs of active inflammation had disappeared; the Malpighian layer was thin and its cells shrunken, the papillæ atrophied, and the deep layer of the cutis transformed into thick bundles of elastic fibres with abundant pigment; the glands had also suffered atrophy. In chronic cases the hair may be lost.

Dr Crocker describes and figures a section of skin taken from a patient suffering from *Pityriasis rubra*. Beside the desquamation of the horny epidermis, there was enlargement of the papillæ, and inflammation of the papillary layer of the cutis, with abundant exudation of leucocytes. The Malpighian layer of the epidermis and the deep layer of the derma were almost unaffected.

Etiology.—General exfoliative dermatitis is common to both sexes and to all ages. Though more frequent in the latter periods of life, it is not unknown in children.

A certain number of cases, as above stated, begin in eczema or psoriasis, but the majority have no such origin. Its true causes are entirely unknown. See Dr Walter Smith's paper ('Brit. Journ. of Derm.,' December, 1898).

In thirty-eight cases collected by the writer, twenty-one occurred in males and seventeen in females. The ages ranged from early childhood to old age. There were five children under 10, seventeen patients between 13 and 50, and fifteen above 50 years old.

Ritter's epidemic cases of dermatitis in infants, which he described in

1879 as *Dermatitis exfoliativa neonatorum*, are in all probability quite distinct from the disease under consideration.

Attempts have been made to connect exfoliative dermatitis with tuberculosis and also with rheumatic fever, but any such connection is probably one of mere coincidence.

Diagnosis.—Pityriasis rubra is distinguished from *eczema* by its abundant scales, by the absence of moisture, by the skin not being thickened, and by its not showing predilection for the ears, face, and flexures of joints; from *psoriasis* by the large, thin, loose scales, and by its not specially affecting the elbows and knees; from *pemphigus foliaceus* by the scales not being preceded by bullæ; from all these forms of superficial dermatitis by its uniform course, by its being strictly universal in distribution, and by the severe symptoms which usually accompany it.

Prognosis.—This is much graver than that of more frequent forms of dermatitis—eczema, psoriasis, lichen, prurigo, or pemphigus; for not only is it difficult to cure, but it sometimes ends in death, especially in elderly people. The presence of albumen is a bad sign, though not a fatal one. Emaciation is still more serious, depending, as it usually does, on loss of appetite or sleeplessness or diarrhœa. Yet the disease is not, as Hebra supposed, incurable, nor is it by any means constantly fatal. Since his book was written cases of recovery have occurred at Vienna and elsewhere.

In forty cases collected by the writer, from various sources ('Guy's Hospital Reports,' series 3, vol. xxv), recovery ensued in fifteen, improvement in several more, and death in only eight. In these cases the fatal event was caused by bedsores and exhaustion, by lobular pneumonia, by acute pneumonia, or by bronchitis. In other instances marasmus ensues, and diarrhœa ends the disease. *P. rubra* often persists for an indefinite period almost uninfluenced by treatment.

Treatment.—Locally the best applications are those which have been recommended in the drier forms of eczema—weak carbolic oil, or olive oil unmedicated, lanoline, lead and zinc ointment, or liquor carbonis picis with vaseline (3j ad 3j) freely and frequently applied. Tepid baths are not counter-indicated, and usually give relief, but if too warm they lead to irritation afterwards, and the effect on the pulse must be carefully watched. Arsenic has not the power it possesses over psoriasis and chronic eczema, but it is often useful and sometimes remarkably so; it is best given in small doses combined with steel. Quinine or bark and mineral acids are often useful. Milk and farinaceous diet appears to suit best, and cod-liver oil should be taken if it does not interfere with other food. Good red wine, or sometimes porter, is in certain cases decidedly beneficial.

In one obstinate case the patient, an otherwise healthy old gentleman, completely recovered after six weeks' sojourn at Strathpeffer, in Ross-shire. In another well-marked and very severe case, dry, scaly, universal, and pruriginous, in a clergyman about sixty, long perseverance with soothing ointments, and increasing doses of arsenic internally, restored him, after several months, to almost complete freedom from the disease.

PEMPHIGUS*

"A most instant tetter barked about,
Most lazar-like, with vile and loathsome crust
All my smooth body."—*Hamlet*.

Names and definition—Anatomy—Histology—Local distribution—Age and sex—Diagnosis—Ætiology—Prognosis—Pemphigus malignus—Acute form of Pemphigus—P. foliaceus—P. serpiginosus—P. vegetans—Hutchinson's cases—Cheiropompholyx—Frequency of pemphigus—Treatment.

Epidermolysis bullosa.

Dermatitis herpetiformis of Duhring—Hydroa aestivalis—Impetigo herpetiformis.

WE now come to a form of superficial dermatitis which is decidedly rare compared with eczema or psoriasis. Although not less remarkable than these in its anatomical characters, its course and natural history are far less characteristic, its causes more obscure, and its true pathology equally unknown. It has been called by two names, *pemphigus* and *pompholyx*;† but of these terms, which, like "lepra" and "psoriasis," were made separate genera by Willan and his disciples, there is no need to retain more than one.

The name *pomphus* seems to have been first medically applied to what we now call a wheal; *pemphix* meant a bulla, and the word *pemphigus* was originally coined to a supposed "febris bullosa" of doubtful nature.

It was by Willan associated with erysipelas, a striking example of the ill result of following an anatomical, or any exclusive, basis of classification for such complex conditions as diseases. He had previously united it with vesicular diseases, but distinguished the two orders in consequence of the criticism of Tilesius, of Leipsic ("über die flechtenartigen Ausschläge," in Martin's 'Paradoxien,' 1801). Bateman practically admitted only one bullous disease, a chronic superficial dermatitis, characterised by blebs.

Until more is known of the pathology of bullous eruptions the recognition of pemphigus as distinct from the rest must be regarded as only provisional.

Anatomy.—The bullæ of pemphigus may begin with a scarcely demonstrable papular stage; but the first lesion seen is a small transparent vesicle which rapidly increases to the size of a pea or larger. These bullæ are sometimes seated on perfectly natural skin; sometimes they are surrounded

* *Syn.*—Bladder-tetter—Pompholyx—Pemphigus chronicus—P. vulgaris.—*Fr.* Pemphigus.—*Germ.* Blasenausschlag.

† Πομφόλυξ and Πέμφιξ both mean a bubble or blister. The former is almost synonymous with φύσαλις, and is applied by Hippocrates to the froth which forms on urine ('Aphor.,' vii, § 34).

by a rose-coloured injected ring, but this is narrow, and they are never found upon an actively inflamed or swollen surface. They usually burst when not bigger than a pea or a marble, but, on the other hand, will sometimes grow to the size of a billiard ball. They are usually tense and hemispherical, occasionally oval. There may be either a single bleb or several of various sizes, irregularly scattered over the same region, and in such groups the intervening skin is often injected. Each bulla, however, forms separately, and it is very rare for two to run together. The contained liquid is transparent, and gives the bulla a pearly appearance. When removed by pricking the bleb, it is thin, colourless or yellow, usually not coagulating, but becoming opalescent or turbid on heating, and showing a few leucocytes under the microscope. After a time, however, it often becomes thick from increase of leucocytes, and before the bulla bursts it may be opaque and yellow—in fact, almost purulent. The contents do not, however, acquire the thick creamy character of pure pus, and always begin as serous and not purulent exudation. Threads of fibrin also appear, not unfrequently before the rupture of the vesicle. Still more common is an admixture of blood, which gives a pinkish aspect to the bulla. After it has burst fresh secretion soon ceases, the ruptured cuticle is either torn off or adheres to the exudation, and the lymph, whether serous, puriform, or coagulated, dries up into a thin yellow crust, which may be more or less stained by hæmoglobin. This soon falls off and leaves a smooth, healthy surface, with scarcely any desquamation; but some passive injection remains, and with this may be mingled more or less pigmentation, so that the circular patches, of sizes varying from a sixpence to a florin, remain for some time as characteristic evidence of pemphigus.

Histology.—The inflammatory exudation of a bulla produced by such an irritant as cantharides takes place in the deepest part of the Malpighian layer of the epidermis. The cells of this layer are first drawn out into bands by the accumulating serum, so that in the early stage each vesicle consists of a series of loculi, as in the case of a burn, first described by Biesiadecki; this stage is long and well marked in the case of traumatic bullæ and in the vesicles of smallpox.

The bullæ of pemphigus, however, as shown by Leloir and Auspitz, consist of an exudation of serum between the horny layer of epidermis and the deeper Malpighian layer, or between the granular layer and those beneath it, without the cells themselves being much affected. There is no vacuolation of cells; the cavity is at no stage multilocular, and the formation of the bleb is far more rapid than that of an inflammatory vesicle or pustule of half its size.

No scars are left after pemphigus, but there is often some degree of pigmentation, and in long-standing cases this may become deep and extensive.

Distribution.—Pemphigus differs from eczema and from psoriasis in being *unsymmetrical*, and having no definite local predilection. The bullæ appear sometimes singly (*P. solitarius*), or succeed one another indefinitely upon distant parts of the body—*pompholyx diutina*, a needless term and ambiguous. More often two or three up to half a dozen form an irregular patch; and isolated bullæ, or one or two other patches follow on other parts of the surface. Occasionally the trunk and limbs are so covered that scarcely any region can be said to be entirely free, yet even then the lesions show no preference for one part over another.

There is scarcely any part of the surface on which pemphigus may not

be seen. On the trunk and limbs it is most frequent; the abdomen and thighs, the genital organs, the ears, the hands and the feet, even the palms and soles and the matrix of the nails may occasionally be the seat of pemphigus; the hairy scalp is least frequently affected.

Bullæ have been observed in the mouth and on the conjunctiva, and in one case of the writer's the latter complication was present.* Oculists meet with such cases confined to the eye, and when adhesion of the two layers of conjunctiva takes place, the result is the "essential" shrinking of the conjunctiva described by von Græfe (C. J. Symonds, 'Clin. Trans.,' 1890).

Age and sex.—Pemphigus, though belonging to the rarer diseases of the skin, may be seen in patients of almost any age. It is commonest in children, decidedly infrequent in adults, but may sometimes be observed in elderly patients, when it is apt to assume its more severe characters.

Occasionally pemphigus occurs in local epidemics among infants living in unfavourable conditions. This has been described as *Pemphigus neonatorum*, and is distinct from the bullous eruption of hereditary syphilis.

Among 38 consecutive cases of the writer's, 10 occurred in males, and 28 in females; 7 between one and five years of age, 15 between six and ten years, 11 between nineteen and fifty, and one in an old woman of sixty-eight.

Diagnosis.—The bullæ of this disease are so characteristic that it cannot be overlooked, and cannot be mistaken for eczema, lichen, psoriasis, or any other of the forms of superficial dermatitis already described. But all bullous eruptions are not pemphigus.

1. Blisters may be produced designedly or accidentally by local irritants, especially by scalding water, or by cantharides. The traumatic bullæ which follow extreme heat are of two kinds—true inflammatory products containing serum or pus, and bladders filled with gas, which have been formed by the lymph of the living skin being turned into vapour and expanded by heat. The latter condition was long ago described by Hilton as the result of burns and scalds; it is of rare occurrence. Its purely physical nature he proved by the fact, which the writer has himself verified, that it is possible to produce it in the skin after death. If a hot iron be held close to the surface, the cuticle rises in a blister like that produced by the sun on a painted board, and on pricking it, no liquid is found within. This is strikingly seen in a negro's skin, when the white cuticle is raised from the dark rete mucosum beneath.

Factitious inflammatory bullæ are usually seen on the arms, and in doubtful cases the glistening scales of the elytra of the Spanish fly may often be distinguished by a lens.

2. Scabies is sometimes accompanied by bullæ, especially in children. An example of this was figured by the writer in the face of a child whose appearance closely simulated that of pemphigus ('Guy's Hospital Reports,' 1877, pl. i), and another was recorded by Dr Fagge (*ibid.*, 1870, p. 333).

3. Syphilitic eruptions in the later stage of the disease are often bullous. Usually the exudation becomes purulent, and the resulting crusts are massive, dark from blood-pigment, and more or less conical, forming the condition described as "rupia," and leaving a superficial ulcer when they fall off, with considerable pigmentation and final cicatrization. In cases of congenital syphilis, however, bullæ exactly like those of pemphigus may be observed. Besides other signs of inherited lues, the appearance of the bullæ on the palms and soles is a character which is diagnostic.

* See Mr Brailey's account of this case ('Guy's Hospital Reports,' 1891, xlviii, p. 165).

4. Iodide of potassium occasionally produces blebs, along with other lesions, which have been mistaken for those of pemphigus.

5. More difficult of distinction from true pemphigus are the bullæ of certain forms of erythema, to be described in the following chapter as herpes iris and erythema bullosum, and also the chronic bullous affection called pemphigus pruriginosus in pregnant women (p. 897). Their locality and symmetry, their multiformity, and their more acute or subacute course, are the chief marks which distinguish these erythematous bullæ from true pemphigus.

6. Bullæ are undoubtedly caused by certain forms of peripheral neuritis and also by lesions of the cord. Such are the occasional results of myelitis, and such is the pathology of the bullæ of leprosy and the vesicles of zona, and also of many cases of injury to the spinal centres or their nerves recorded by Weir Mitchell, by Hutchinson, Bowlby, and other writers (*cf.* vol. i, p. 574). But such secondary cutaneous lesions do not seem to be clinically related to pemphigus, or to bullous erythema, or to zona.

7. The distinction between pemphigus and Dermatitis herpetiformis will be considered separately, p. 888.

Ætiology.—The cause of pemphigus is absolutely unknown, although, as in other cases, teething, gastric irritation, excess in diet, irritability of the system, mental affections, anxiety, fatigue, amenorrhœa, exposure to cold, and residence in damp situations have been confidently stated as each a cause of the disease. According to Alibert, the “lymphatic temperament” predisposes to pemphigus, which perhaps meant what is true, that it is more common in children than in adults.

It is now well established that pemphigus is never contagious. Hebra recorded one remarkable case of heredity.

Some cases appear by their distribution to depend on peripheral neuritis, and such tropho-neurotic cases would connect pemphigus with zona (so Schwimmer, of Buda-Pesth, and some other authorities). No doubt some forms of bullous dermatitis are angioneurotic in pathology, but these are not pemphigus.

In other cases bacteria, most often diplococci, have been found in the contents of the bullæ; but they have often been sought for in vain, and there is no reason to believe that they are specific.

Prognosis.—In children pemphigus is rarely fatal (excluding so-called *syphilitic pemphigus*), and under suitable internal treatment it is in most cases quickly curable. But in old persons it is apt to spread very widely; sleeplessness and loss of appetite follow, and death may result. This is most to be feared when there is chronic renal disease present, but a fatal result may occur independently of this complication.

Such cases have been made a distinct variety, *pemphigus malignus vel cachecticus*. The bullæ are very numerous, are never tightly filled with serum, but look flaccid and rupture early. There is little effort at healing, and extensive raw patches cause much pain and distress, combined sometimes with more or less itching. The exudation is frequently hæmorrhagic and sometimes fibrinous, and the mucous membranes are apt to be affected. As in other severe and extensive forms of dermatitis, there is sometimes albumen in the urine, independently of previous Bright’s disease.

These serious cases, though rare except in old persons, may occur at any age; in children, bullæ after bursting are sometimes succeeded by gangrene, and this *Pemphigus gangrænosus* has also been separately described.

It has no doubt been frequently confused with what used to be called "*rupia escharotica*," that is to say with a bullous syphilitic eruption. But there is no question that true gangrenous pemphigus does occur, alone or after varicella. In a little boy aged four, who died of gangrenous pemphigus after chicken-pox in Guy's Hospital in 1882, we found *post mortem* all the viscera perfectly normal, and there was no reason to suppose the presence of congenital lues.

Acute pemphigus.—Hebra discussed the existence of acute or febrile pemphigus (*febris pemphigodes*), and, like Bateman before him, concluded that when urticaria and herpes iris, erysipelas, and rupia, with other forms of syphilis are excluded, there is no such disease. The existence of such cases, however, is now well established. Dr Southey recorded a case ('Clin. Soc. Trans.,' viii, 1875), Dr Payne another, with a table of temperature ('St Thos. Hosp. Rep.,' vol. xii), and Sir D. Duckworth a third ('St Barth. Hosp. Rep.,' vol. xx). The last occurred in a man of fifty-four, suffering from Bright's disease, and he died on the ninth day; but the event was probably not due to the eruption on the skin, and if not cut short by death this might have proved chronic. Moreover, it is possible that this, as well as other cases, might be interpreted as bullous erythema, though it would doubtless be difficult to maintain the distinction in every instance. Some authors distinguish the fatal cases as malignant, but this seems needless. The acute cases, whether they run a favourable course or not, are very rare.

Mr Pernet recorded eight cases of a febrile bullous disease occurring in butchers, which he regarded as acute pemphigus. In some of these cases Dr Bullock found a diplococcus, probably identical with that described by Demme ('Brit. Journ. Derm.,' May, 1896). See also Dr Whipple's two cases of Acute Pemphigus, published in the 'Lancet' for 1896 (vol. i, p. 1219, and Dr Payne's, *ibid.*, 1893, vol. ii, p. 421).

Pemphigus foliaceus.—Cazenave described under this title a remarkable form of cutaneous disease which has since been recognised by Hebra and later dermatologists. It is rare, and the writer has only seen two well-marked cases, one at Vienna, and the other in Guy's Hospital, which was modelled for the museum ('Catalogue,' p. 94).

The patients are usually adult women. The blebs appear at first like those of ordinary pemphigus, but they never become tense and pearly in appearance. They rupture early, and form thin, dirty-white laminæ, which continue to exude a scanty secretion. The aspect of the affected skin has been likened to that of flaky pie-crust, to birch-bark, and to dead leaves—whence the specific name.

The distribution of this form of pemphigus is remarkable, in being more or less universal. On this ground and that of its malignancy the late Dr Baxter associated it with pityriasis rubra. Cases have been described as originating in a condition like Dühring's Dermatitis herpetiformis. The mucous membrane of the mouth and fauces is affected, and the hair falls off.

The course of *P. foliaceus* is very slow, and there is no disposition to recovery. Indeed, it is doubtful whether any genuine case of pemphigus foliaceus has ended favourably. Drugs have little or no influence upon it, and after a protracted illness the patients die emaciated, or are carried off by some intercurrent disease.

Serpiginous pemphigus is a rare form, which arises only in the most chronic cases. The bullæ, which are small, are seen on the red advancing border of a considerable space of skin, formerly the seat of others that have disappeared. When first seen in this latter stage it might well puzzle an observer. A well-marked case of pemphigus serpiginosus, which began as an ordinary case, recurred, and each time was cured by arsenic. It furnished two of Mr Towne's models in the museum of Guy's Hospital ('Catalogue,' p. 92).

Pemphigus vegetans.—Neumann described under this title a bullous disorder in which the bursting of the bullæ is followed by the appearance of granulation masses (*i. e.* ulcers covered with frambœsiform papillary growths). The cases were not benefited by arsenic, and ended fatally. Eleven cases have now been recorded in Germany, one by Dr Crocker ('Med.-Ch. Tr.' March 12th, 1889), one by Dr Mapother, of Dublin (*ibid.*), one by Dr R. Hyde from America ('Journ. Cutan. and Gen.-ur. Dis.,' Nov., 1891), and another by Mr T. P. Lowe, of Bath ('Lancet,' 1891, vol. i, p. 1046), and Dr Stopford Taylor ('Journ. of Derm.,' 1894, p. 178). It is called *Erythema bullosum vegetans* by Unna. The only case of this remarkable form of disease which the writer has seen was in a patient of Mr Hutchinson's, who has referred to the case in his 'Archives' (vol. vi, p. 199). Like *P. foliaceus*, this form differs from ordinary pemphigus in affecting the mucous membranes, in resisting arsenic, and in a fatal event.

Hutchinson's bullous disease of hands and feet.—A curious form of bullous eruption, which may be provisionally called *pemphigus vegetans mitis*, was shown by Mr Hutchinson at the Pathological Society, as "hand-foot-and-mouth disease." Beside the bullæ on the trunk and limbs, there was severe inflammation of the hands with loss of nails, and also inflamed mucous membrane of the mouth and tongue. A case was exhibited at the Dermatological Society in 1885, which was recognised by Mr Hutchinson as of the same character. There was unmistakable pemphigus here, and both loss of nails and sore mouth in pemphigus have been described by Hebra, so that no doubt he would have included under that title the curious cases described. Moreover, the same combination was described by Rayer as complicating pemphigus.

Pemphigus neonatorum has been described as affecting cachectic infants, and as sometimes complicated by purpura or by gangrene. They are distinct from the bullous form of congenital syphilis, and are probably the result of septic infection.

Cheiopompholyx.—This affection also was described by Mr Hutchinson. It is chiefly confined to the hands and feet, is symmetrical, affects the nails, is recurrent, and the bullæ are small without dermatitis around them. Dr Robinson, of New York, Dr Liveing, and other writers have described similar cases. It affects the palm and sides of the fingers, as well as the dorsum of the hand. Before rupture the small bullæ, or large vesicles under the thick skin of the fingers, are described as like sago grains. This disease is probably distinct from the affection of the sweat-glands described by the late Dr Fox as dysidrosis (*cf. supra*, p. 858). It more nearly resembles erythema bullosum.

Frequency of pemphigus generally.—Hebra, writing of twenty years' experience in the General Hospital of Vienna, as well as in his large private practice, could reckon only about 200 cases. He estimated that, excluding infants, one case of pemphigus occurred in 10,000 cases of illness generally. We must remember that the hospital statistics apply to all medical (as well as surgical) diseases, whereas his own practice was exclusively dermatological. He found in thirty years' statistics at the General Hospital that there were ten cases of pemphigus in men for rather more than three in women, excluding pemphigus foliaceus. In a report to the American Dermatological Association in 1881, only twenty cases occur in 11,000; Erasmus Wilson reported only nineteen cases among 10,000 private patients; and Kaposi, in 1890, had met with only 210 cases among more than 40,000 cases.

Treatment.—In Joseph Frank's work on diseases of the skin he stated that the best treatment of pemphigus is to leave it alone. Hebra proved the uselessness of diuretics and purgatives, tonics and quinine, mineral acids and Carlsbad waters. Formerly English physicians recommended venesection or leeches with antiphlogistic regimen, but with a caution to pursue the plan guardedly, which probably meant not to pursue it at all. They also recommended acids and bark. In the first edition of Wilson's work (p. 142) he writes: "When there is reason to believe that the eruption is an effort on the part of nature to determine to the surface a morbid disposition, I should strongly recommend the employment of mustard baths to the entire surface of the skin, or a stimulating liniment of some kind, such as that of croton oil, in the proportion of a drachm to an ounce of olive oil, to be well rubbed into the sound parts of the skin." Hardy says: "Le traitement général du pemphigus est encore à trouver." German authorities speak doubtfully of the prognosis in this disease, and depend chiefly upon local applications for its treatment. The late Dr Tilbury Fox recommended chlorate of potash, good food, and above all quinine, which he preferred to arsenic.

At the present time, however, most English physicians are agreed that *arsenic* is as much a specific remedy for pemphigus as for psoriasis. No doubt it occasionally fails, even in ordinary cases; but this can also be said of mercury in syphilis, and no one pretends that arsenic will cure gangrenous pemphigus, or pemphigus foliaceus, or bad cases of extensive pemphigus in aged patients; but in nine tenths of the cases of pemphigus occurring in children and young adults the statement holds good that arsenic may be esteemed a sure remedy, even in severe cases. The drug should be administered on the same principles and with the same determination as recommended in psoriasis.

Occasionally, however, we meet with cases in which, after perseverance with varied doses and varied forms of administration, we are obliged to abandon arsenic, not because of its disagreeing (that can always be met by diminishing the dose), but because the disease is unchecked. The best remedy then is tincture of steel. Sometimes quinine or cod-liver oil succeeds when other drugs have failed. Cod-liver oil is useful in the case of children, and full diet is always necessary. Alcohol in some form is called for in the more severe cases, rum and milk, or mist. sp. vini gallici, or porter where nutrition is most at fault, and champagne or port wine where

stimulation is needed. Neisser recommends in such cases subcutaneous injection of strychnia.

In true gangrenous pemphigus of children, excluding syphilis, brandy and strong broths, or raw meat, with chlorate of potash internally, is the best treatment, and commonly proves successful. But even in infants one-minim drops of Fowler's solution should be administered.

In bad cases, especially in aged patients, attended with restlessness and distress, *opium* is a most valuable remedy, but unfortunately its use is forbidden or much circumscribed by the not infrequent presence of albuminuria. When this is absent it has a most valuable effect. In *P. vegetans* and "hand and mouth" cases, opium is particularly indicated.

So far as is known, neither arsenic nor any other drug is of service in cases of pemphigus foliaceus.

Locally, whatever application is most soothing is best: either zinc ointment, or oxide of zinc in powder, or what is often more pleasant and effectual, oxide of zinc with finely powdered chalk or gypsum suspended in water, and applied with a large soft brush. In very extensive and severe cases continuous baths have proved useful.

Epidermolysis bullosa.—Under this title have been described several cases of bullous dermatitis, which differ in certain respects from Pemphigus. Some of the cases have been described as Congenital Pemphigus, and Dr W. Beatty, of Dublin, has recorded three cases in a father and his two children.*

The characters are that the bullæ are called out by friction or injury, that they often occur soon after birth, that they are usually hæmorrhagic, that they affect the nails and the conjunctiva, causing more or less permanent results. There is sometimes great irritation, like *Pemphigus pruriginosus*. Arsenic is of no service. Three cases have been published by Payne,† two by Wickham Legg;‡ and others by Köbner (who gave the name "Dermatolysis bullosa hereditaria"), Brocq, Lesser, Duhring (as congenital chronic pemphigus with atrophy), and Colcott Fox.

Dermatitis herpetiformis.§—Dr Duhring, of Philadelphia, proposed in 1884 to unite certain bullous and vesicular forms of dermatitis under this title, including most cases of what Bazin had called Hydroa, as well as those which Dr Tilbury Fox, in a posthumous paper (published in the American 'Archives of Dermatology' for 1880), had recognised under that name. This would have been only a change from a meaningless but distinctive term to a new one; but there was a more important change involved when Dr Duhring included also Herpes bullosus, Pemphigus pruriginosus, Herpes gestationis, Impetigo herpetiformis, and other ill-defined species of Pemphigus or Herpes.

* See his article in the 'Brit. Journ. of Derm.' for August, 1897, with a full record of cases, and Dr Colcott Fox's case in the September number following. Also Dr Payne's earlier account published in the 'Lancet' in August, 1893, p. 425, under the title "Congenital Traumatic Bullous Disease;" Goldschneider drew attention to the condition in 1882 as "Hereditäre Neigung zur Blasenbildung;" and the cases of congenital ulceration with pemphigus in children recorded by Tilbury Fox in the 'Lancet,' in 1879, no doubt belong to the same group.

† 'St Thomas's Hosp. Rep.,' 1882 and 1886.

‡ 'St Bartholomew's Hosp. Rep.,' 1883.

§ *Syn.*—*Dermatitis polymorpha pruriginosa* (Brocq); *Hydroa herpetiformis* (Crocker).

The tendency of recent writers has been to restrict the meaning of Pemphigus, as understood by Hebra, to a primary bullous eruption with a chronic or subacute course, absence of severe irritation, and irregular distribution. After excluding traumatic and artificial bullæ, caused accidentally or intentionally by scalding water or cantharides, and the occasional blebs seen in the course of scabies or as the result of congenital or of acquired syphilis, and toxic eruptions with bullæ, there remain a considerable number of cases which resemble pemphigus in the presence of bullæ only. Many, perhaps most of these, find their place as bullous erythema, for to this group they seem to be most nearly related by their acute or subacute course, their predilection for the back of the forearms and wrists, the shins and the insteps, their recurrence, their mildness, the absence of severe itching, and their occurrence along with urticaria and papular forms of erythema. This form of bullous dermatitis will, therefore, find its place in the following chapter.

Duhring's cases are marked by an acute onset, but a chronic and recurrent, or relapsing, course; by the presence of large red patches with raised margins and depressed centres, of vesicles, and even pustules, beside bullæ; by an extensive distribution, chiefly on the trunk, but often affecting the limbs; and, above all, by severe itching.

The lesions are described as multiform, macular, papular, vesicular, or bullous, but apt to occur in groups of vesicles on an injected disc, like Herpes as defined by Willan and Bateman. The parts affected seem to be chiefly the arms, the hands, the buttocks and thighs, but also the back, abdomen, genitals, and trunk generally, the only part exempt being the face. The course is acute or subacute with recurrence, but often chronic and persisting for years. The symptoms are burning at first, and afterwards pruritus. The mucous membrane of the mouth and nose may also be invaded.

The result is usually favourable, but not uniformly so; and the treatment is chiefly what used to be called expectant, though some cases are reported as cured and others as aggravated by arsenic. It does not appear to be ever fatal, as Pemphigus undoubtedly is in a considerable minority of cases, but it is not very amenable to treatment, and very prone to relapse. Belladonna, quinine, ol. morrhuæ, alkalies, colchicum, and mineral waters have each been recommended as more or less useful.

Locally tarry preparations, like oleum cadini (juniper oil) and liquor picis carbonis, ichthyol, phenol, and thymol, have been found useful. Duhring himself found sulphur ointment of great value in vesicular, pustular, or bullous cases. Hyde and Montgomery conclude by saying that it is not certain that the disease is ever completely relieved, though temporary recovery from repeated attacks is common.

It seems clear to the writer that a certain number of the cases described as *D. herpetiformis* would have been called Pemphigus by Hebra, and, indeed, they are called so still in Vienna. Others he believes belong to the group of bullous and vesicular Erythema, under which he includes in the following chapter most of so-called Hydroa and Herpes.

There appear to remain certain cases which are neither chronic and purely bullous, like pemphigus, nor yet acute, benign, and either self-limited or recurrent within moderate periods, like erythema. These cases are marked by great irritation, long duration, and resistance to treatment, by pigmentation of the skin, and by the vesicles appearing in groups.

Whether the presence of leucocytes with eosinophil reaction, both in the blood and in the vesicles and bullæ, is a constant condition, and whether it is distinctive, remains to be seen. The question of a nervous pathology of these cases also, though constantly affirmed,* has not yet been proved. The likeness to Zona is more than balanced by the fact of that disease being unilateral, not irregular or symmetrical in distribution; and the participation of vaso-motor and trophic nerves is too generally true of all exudations to make it distinctive of any one in particular.

Hydroa æstivalis is the name given to a rare affection of the face in children, which occurs on the face and hands from exposure to the sun and wind. It is the recurrent summer eruption of Hutchinson, the *Hydroa vacciniformis* of Bazin, the *Hydroa puerorum* of Unna. It runs an acute course, is not irritable, and leaves scars, but has been accepted as an aberrant form of *Dermatitis herpetiformis*. The remarkable cases observed by Dr MacCall Anderson ('Brit. Journ. of Derm.,' Jan., 1898) are certainly very peculiar, and approach Raynaud's disease.

Impetigo herpetiformis.—This name was given by Hebra ('Wiener Med. Wochenschrift,' 1872) to a small number of cases, remarkable for their occurrence in women, and frequently in pregnant women, for the small pustules spreading over the whole body, and affecting the mucous membranes also, and lastly for their almost uniform fatality. A few other similar cases have been recorded since the first thirteen were described by Kaposi in 1887. Auspitz and others believe them to be of septic origin. In some respects they approach herpes gestationis, in others they resemble Mr Pernet's cases of acute pemphigus above mentioned. Duhring at first considered them to be a pustular form of his *Dermatitis herpetiformis*; but at the Congress at Paris in 1889 he ejected them from this harbour of refuge. Recent cases observed at Strassburg, reported by Maret (1887) and by Gunsett (1901), show that the condition may appear in men as well as in women, and that the prognosis is not so bad as Hebra supposed.

* Thus, for instance, Dr Jamieson, of Edinburgh, quoted as "much the best definition" Unna's, that *Dermatitis herpetiformis* of Duhring is a chronic neurosis of the skin, associated with some yet unexplained blood changes, and not markedly interfering with the general health. This causes a more or less universal eruption, coupled with burning and itching sensations, and regularly recurring for an indefinite period after intervals of complete or comparative immunity. The type is erythemato-bullous—which, however, may undergo considerable modification.

ERYTHEMA

AND ITS ALLIES

“Τὸν ὑπὸ βλεφάροις
φοίνικ’, ἐρύθημα προσώπου.”—EURIPIDES.

Definition of the group—Its characters—the anatomical lesions—course—locality—symptoms—etiology—Symptomatic and traumatic erythema—Varieties: (1) Erythema multiforme—(2) E. bullosum—Herpes iris—Herpes gestationis—Hydroa—(3) E. nodosum—(4) Urticaria—Urticaria pigmentosa—Treatment of erythematous affections.

Erythematous and other rashes produced by drugs—Copaiba—Bromides and iodides—Belladonna—Opium—Quinine—Salicylates—Arsenic—Mercury—Antitoxic Sera.

WE have hitherto considered diseases of the skin which, though differing from one another in many particulars, are all examples of superficial dermatitis; they never leave scars, they are chronic in course, they are very apt to return, and are accompanied with more or less decided irritation. Eczema, Psoriasis, and Lichen planus are the most important members of this group, the Tetters of English, the Dartres of French popular pathology.

Pemphigus forms a natural link between the true tetters—eczema, lichen, and psoriasis, on the one hand, and the erythematous affections on the other. Indeed, while Hardy classes it decidedly with the *dartres*, Auspitz and Hans v. Hebra place it in close relation to *Erythema multiforme*.

We now pass to disorders which form another natural group, though the line is perhaps more difficult to draw.

They also are *superficial inflammations* of the skin, and therefore leave no scars, but they are *acute or subacute*, never chronic, in their course. Moreover, their anatomical lesions are usually slight and evanescent; a rose rash is the most frequent; papules, wheals, or bullæ are often met with; but vesicles rarely, primary pustules never, nor yet permanent scales. Perhaps their most characteristic anatomical feature is the presence of inflammatory *œdema*, and this is often accompanied by slight *hæmorrhage*. The sensations accompanying them are usually *smarting* rather than itching. They have no relation to external cutaneous irritants, but may be traced to gastric or other *internal* causes, to specific febrile diseases, *i. e.* to organic toxines, to certain kinds of food, or to certain drugs. If we call much of Eczema, scabies, and prurigo in part *traumatic* in origin, we may call the affections described in the present chapter *toxic*.

Eczema, lichen, and psoriasis, even when their onset is acute, run an indefinite course afterwards; while the present group has an acute or sub-acute, self-limited course, often recurrent but never chronic.

ERYTHEMA is the name which has been given to some affections belonging to this group, and it may be conveniently extended to the whole.*

Willan classed erythema with roseola, urticaria, scarlatina, rubeola, and purpura under the title "exanthemata," the general character of the order being hyperæmia of the skin without further lesion. Subsequent writers have called a mere hyperæmia "rose-rash" or roseola; while the word erythema, or *erythema exudativum*, as Hebra called it, has been confined to a rose-rash with palpable inflammatory exudation, diffuse or forming pimples. Hebra included the so-called tubercular and nodose species of Willan's genus erythema under the general title *polymorphe erytheme*, but further distinguished between *E. nodosum* and *E. multifforme*.

But in truth we have no need of a special title for mere hyperæmia, that is, dilatation of the blood-vessels without inflammatory exudation, such as follows division of a vaso-motor trunk in an animal. A transitory blush is a physiological, not a morbid, phenomenon.† Clinically, a persistent, "active," or arterial hyperæmia is always part of inflammation. Even the erythematous eruption of scarlatina, of measles, or of enteric fever can be proved by its course and sequelæ to be in each case true dermatitis. Bate-man himself remarked that the efflorescence to which Willan appropriated the title of roseola is of little importance practically, and quoted the dictum of Fuller in his 'Exanthematologia' that it is "rather a ludicrous spectacle than an ill symptom."

We must, however, recognise two meanings of the word "erythema," just as we were obliged to recognise two of the word "eczema." We saw that eczema is, as Hebra proved, a common superficial dermatitis, which has reached the stage of visible and usually serous exudation; but we saw also that the disease is in many cases not called forth by ordinary irritants, and not limited by their action.

In the same way "erythema" may be defined, and has been used by Hebra and most authors to denote the slightest form of dermatitis, in which the classical signs of redness, heat, and pain are accompanied by little or no perceptible swelling. The irritation of a mustard plaster, for instance, will in most persons produce such a typical "erythema;" the scorching of the sun does the same; and if the skin be more than usually delicate, and the mustard or sun more than usually strong, what was an "erythema" becomes an "eczema."

Traumatic dermatitis of slight degree is still often called erythema. Thus *intertrigo*, mentioned above under eczema (p. 767), was classed under erythema by Willan, Hebra, and Neumann. Erythema leve, the inflammatory blush which sometimes appears in Bright's disease, is another example of this use of the term.

It would, however, conduce to clearness of language, and to what can

* This word, denoting "redness" of the skin, is applied in classical Greek, either as *ἐρύθημα προσώπου* (see motto at head of chapter) or alone, to a blush, and by Thucydides to the redness of the eyes seen in those suffering from the plague at Athens. It was also used by medical writers as almost, if not quite, synonymous with *ἐρυσιπέλας*.

† Even when arterial hyperæmia is produced artificially by dividing the cervical sympathetic in a rabbit, the condition may be prolonged for many months, as the writer proved ('Journal of Physiology,' 1884), without there being inflammation, *i. e.* exudation of serum and leucocytes.

hardly exist without it, clearness of thought, if the term "superficial traumatic dermatitis" were used for both stages of the inflammation; or we might speak of the earlier as an erythematous and of the latter as an eczematous or weeping dermatitis.

Apart from this use of "erythematous" as indicative of a slight degree of inflammation with hyperæmia, we are only following Hebra's initiative by taking the best marked forms of disease to which the name Erythema is attached, and grouping with them under the same title others which have the same pathology and clinical features, so as to form a natural group.

Although often strictly "erythematous" in the usual sense of the word, they sometimes exhibit other lesions, to which Hebra's adjective "multi-form" applies.

1. Their most characteristic *anatomical lesion* is a rose-rash, resembling the first degree of traumatic dermatitis, that is to say hyperæmia of the surface; sometimes with obvious diffused *œdema*, sometimes with circumscribed *œdema*, forming *wheals*, and sometimes with *papules*—which are distinguished from those of eczema by not developing into vesicles, from those of lichen by their brighter colour and transitory duration, from those of impetigo and prurigo by never becoming pustular, and from those of psoriasis by never becoming scaly. In certain comparatively rare forms of erythema separate *bullæ* are formed which may simulate those of pemphigus. The rash is usually followed by a slight branny desquamation. It will be seen that, after all, the multiformity of the lesions of erythema is less than that of the lesions of eczema.

2. Whatever the form of the lesion, the exudation is of a watery rather than a corpuscular nature, so that *œdema*, diffused or circumscribed, is its characteristic, in contradistinction to the sero-purulent or purulent vesicles and pustules of eczema and scabies. Moreover, along with the hyperæmia and *œdema*, there is very apt to be a certain amount of escape of erythrocytes as well as leucocytes, an event which never occurs in eczema except as the result of direct injury, as by scratching. The result of this hæmorrhage is sometimes so marked as to give the title "purpura" to the eruption. Willan and Bateman rightly included *purpura urticans* with erythema among the exanthemata, although other kinds of purpura are altogether distinct from any form of dermatitis, and are only parts of a general hæmorrhagic condition. The result of the hæmorrhage is to leave a bruise-like pigmentation behind, which when present is very characteristic of true erythema.

In these characters, and in the fact of the occasional occurrence of *bullæ*, the erythematous group bears a closer relation to pemphigus than to any other of the chronic forms of dermatitis belonging to the so-called darts group, but if one attempts to include pemphigus as an erythema its course and treatment forbid the conjunction. Auspitz, however, and Hans von Hebra, whose classification differs widely from that of his father, unites under the title "Angioneurotic affections of the skin," the erythematous rashes of infectious diseases, the rashes produced by drugs and poisons, the primary erythema, and lastly pemphigus and acne rosacea.

3. The erythematous rashes do not spread. They appear simultaneously at different spots, and fresh patches appear which may occasionally unite, but we do not see the affected part of skin gradually enlarge its borders—the characteristic serpiginous progress of eczema and psoriasis.

4. The *course* of erythema is acute (or subacute), that is to say it begins

quickly, sometimes with slight febrile symptoms, and does not last indefinitely. Even when its course appears to be comparatively chronic, it will be found that the protracted disease is really made up of a series of outbreaks, which may sometimes run into one another, but always preserve a recurrent or intermittent character. No true erythematous disease ever acquires the chronic, stable, and inveterate stamp of eczema, lichen planus, psoriasis, pityriasis rubra, or pemphigus.

Erythema is, however, often recurrent, and, when frequently so, may produce chronic induration and hypertrophy. Of this, Gutta rosea is a capital instance.

5. The *distribution* of erythema is much less definitely marked than that of psoriasis or of eczema. On the whole it is symmetrical, sometimes accurately and exclusively so, but there are frequent exceptions to the rule.

The favourite localities are, first, the extensor surface of the forearms and legs, especially the back of the hand, wrist, and ulnar side of the forearm, the dorsum of the foot, and tibial side of the shin;* secondly, the forehead, nose, cheeks, and neck; thirdly, the chest and abdomen. The back of the trunk, the buttocks, thighs, and upper arms are much less frequently affected; while the scalp, the flexures of the joints, the palms, and the soles are scarcely ever the seat of true erythema.

6. As a rule, smarting and tingling are the *symptoms* which accompany erythematous eruptions, while severe pain and itching are both rare. Local tenderness is more marked than in eczema. Sometimes, however, and especially when wheals are present, the irritation is considerable, though it does not equal that of chronic eczema, scabies, or prurigo.

Of the *ætiology* of erythema we know little. The lesion can be produced by moderate irritation—the diffuse forms by heat or friction, those with wheals by the lash of a whip, or by the poison of the stinging-nettle, the hairs of certain caterpillars, and the thread-cells of anthozoa and hydroids. But in the non-traumatic, idiopathic, or “true” cases of erythema, the eruption can, in striking contrast to those of eczema and psoriasis, be in most cases traced to some *internal* disorder. In other words, erythema is usually symptomatic. The most striking instance of this is the erythematous rash produced by copaiba and by certain articles of food. Many other cases are dependent on dyspepsia; others, again, on rheumatic fever, particularly *Er. nodosum*, urticaria, and the hæmorrhagic form of erythema known as *Peliosis rheumatica*.†

As symptomatic, or secondary to a general toxic condition, we may group this rheumatic erythema with the early rash of syphilis, the exanthems of scarlatina and of measles, and the occasional prodromic roseola of smallpox and cholera, all of which belong to the erythematous type.

Generally, therefore, we may say that erythema and its allies are due to internal causes and, as a rule, to poisons.

Erythema occurs most commonly in children and young adults; it is comparatively rare after forty. Among persons past their prime it is less uncommon in women than in men. In these as in other points, we observe a resemblance to pemphigus and also to rheumatism.

* Although the true homology of the tibia is undoubtedly with the radius and not with the ulna, yet the tibial aspect of the shin, from its having no underlying muscles, agrees pathologically with the skin covering the subcutaneous surface of the ulna, just as pathologically the patella answers to the olecranon, and the second metacarpo-phalangeal to the first metatarso-phalangeal joint.

† ‘Guy’s Hosp. Rep.’ 3rd series, vol. xix, p. 325, in 1881; *ibid.*, vol. xxv, p. 208.

The following are some of the lesions which have been called erythema, but not in the sense above defined.

Erythema intertrigo of Willan is a form of traumatic dermatitis, and has been already described (p. 823).

Erythema induratum of Bazin is a chronic tuberculous inflammation, often ending in ulceration.

Erythema elevatum diutinum was a name imposed by Dr Crocker on a case of persistent raised nodules in a child, which more or less resembles cases recorded by Dr Judson Bury and Mr Hutchinson. Whether the nodules were rheumatic or not appears to be doubtful.

Roseola furfuracea (*roseola maculata et circinata*) is a slight inflammation approaching some forms of erythema, which has been described above as *Pityriasis rosea* (p. 876).

Pernio.—The chronic congestive disorders, of which chilblain is the type, are chronic in course and venous or cyanotic in aspect and pathology. They have no claim to be ranked under erythema, as above defined, and will find their place in a subsequent chapter, along with *Gutta rosea*.

Exanthems.—The erythemata which are symptomatic of measles, scarlatina, enterica, rubeola, as well as choleraic roseola, and that which sometimes precedes the characteristic rash of smallpox,—all these have been described in the first volume of this work.

Medicinal erythema.—The rashes which follow the administration of drugs will for convenience be considered together at the end of this chapter.

There remains a group of dermatoses which agree in the general characters of anatomy, course, and natural history described above, but they are not symptomatic like exanthems, nor due to what French writers call "intoxication" with drugs or poisons. These we may style idiopathic, primary, or essential erythema. Their common characters have been already sufficiently expounded. It remains to point out the principal varieties which they present. We include under the general head *Erythema* not only Hebra's multiform and nodose kinds, but Herpes (in part), *Urticaria*, and some cases of what Bazin called *Hydroa*, and *Duhring dermatitis herpetiformis*.

1. *Erythema multiforme*.—Simple or ordinary erythema, *erythema papulatum*, *erythema exudativum*.

The commonest kind of erythema is that which consists in general hyperæmia with œdema of the skin, a diffuse dermatitis which may either spread over a large surface with indefinite edges, or, as is more frequently and characteristically the case, occur in circumscribed patches.

On careful examination, small papules may be often distinguished scarcely rising above the level of the skin, as in the eruption of measles; sometimes these are well marked enough to deserve the title *erythema papulatum*, but this is comparatively rare, and most lesions of the skin which receive this name are probably either traumatic dermatitis or an early stage of lichen and eczema. Large, firm, and persistent papules such as occur in *prurigo* are never seen in true erythema.

The inflamed patches have usually a very short duration; they may disappear in a few hours (*Erythema fugax* of Willan) and be succeeded by others, but if they persist for a day or two they may form rings which have been specially described as *Erythema annulatum* (*E. circinatum* of Willan),

or *Roseola annulata*. When closely set, several of these rings unite, and a sinuous reddish band is produced which has been named *Erythema marginatum* or *E. gyratum*. Finally, the redness fades, the œdema subsides, and may leave no trace behind. If there is desquamation it is very slight and furfuraceous; more frequently a slight amount of pigment marks the seat of the eruption.

The favourite localities are the back of the wrists and forearms, and the legs; less often the face and neck; sometimes the trunk is affected, but very rarely the thick parts of the skin or those covered by hair.

Erythema annulatum is sometimes observed in association with rheumatic fever, either in the course of an attack or preceding it. It is more common in children and young adults than in older persons.

The inflammation of the skin called *Erythema leve* is a common dermatitis which is apt to appear upon the tense skin of dropsical parts, and may go on to deep dermatitis and sloughing. It is most often seen in cases of renal dropsy. It was formerly not uncommon as the result of acupuncture, and simulates traumatic erysipelas.

2. *Vesicular and bullous erythema*.—The exudation of erythema, instead of being a somewhat deep diffused œdema, sometimes appears in superficial collections of serum. These when small are called vesicular erythema or “herpes;” when large, “erythema bullosum.”

Herpes,* the common Latin term for an eruption of the trunk, in contradistinction to *porrigo* or an eruption of the head, was limited by Willan to vesicular eruptions which he distinguished from the vesicles of smallpox and chicken-pox, from sudamina, and from the inflammatory vesicles of eczema. His species of herpes were as follows:

Herpes zoster, or *Zona*, an eruption erythematous, it is true, in its anatomy and course, is so dependent on nervous disorder that it is rightly separated from all other vesicular eruptions, and will be described in a future chapter among cutaneous disorders of nervous origin.

Herpes circinatus, which we shall afterwards describe as the form assumed by ringworm when it affects the body, is a parasitic disorder, and is now classed with the other *Tineæ*.

Willan and Bateman's remaining species are *Herpes phlyctænodes*, of uncertain seat, called *H. labialis* or *H. preputialis* when affecting the lips or the foreskin respectively, and *H. iris* when found on the back of the hands or the instep. These from their course and natural history may be well included in the general group of erythemata. This was, indeed, to some extent admitted by Hebra, and even by Rayer before him.

Herpes labialis or *facialis*—or Herpes without addition—consists of a little group of vesicles upon a red patch of skin which appears almost suddenly, most often upon the upper lip, frequently on the alæ of the nose, sometimes on the cheeks or chin. The writer once saw symptomatic herpes or vesicular erythema cover one ear of a boy ill with lobar pneumonia, and run a typically acute course. Sometimes the vesicles appear on the buccal mucous membrane. In a few days the clear, pearly contents become somewhat turbid and puriform, and dry up into a thin brownish crust which falls off and leaves no trace behind. The time it lasts is twelve or fourteen

* The word ἑρπης is derived from ἑρπεῖν. “Herpes dicitur eo quod videtur ἑρπεῖν, quod est serpere per summam cutem, modo hanc ejus partem modo proximam occupans.” From the same creeping progress the disease was, according to Bateman, called *formica*.

days. The vesicles differ from those of eczema by their large size, their longer persistence, by their not running one into another so as to form a weeping surface, by their acute course, and by the sharply limited edge of the patch. They also are unattended with itching or pain, and never consist of pure pus, like the eruption of impetigo or scabies. Moreover, they are always symptomatic of some internal disorder, most characteristically of acute lobar pneumonia. Many persons are liable to such patches of herpes, either on the lips, or less frequently on other parts of the face, when they are attacked by acute catarrh. Broncho-pneumonia and Pleurisy are not infrequently accompanied by herpes, Phthisis and Enteric fever very rarely. Sometimes the eruption appears to follow a rigor even when this symptom does not prove the precursor of pneumonia or catarrh.*

This curious eruption has clearly little or no connection with eczema and its allies; nor can we link it with zona, for it frequently recurs, it is not unilateral, it does not follow the course of a nerve, and is unattended with pain. Its superficial character, sudden onset, and rapid course agree with the erythematous group as here defined, and the fact that it is symptomatic of internal disturbance and usually of irritation of a mucous tract completes the analogy.

Herpes preputialis, when no longer left as Willan placed it, and as Hebra was content to leave it, among vesicular inflammations, is difficult to classify. Hardy was even driven to the untenable assertion that it is nothing but local vesicular eczema. The rapidity of its onset and course, its superficial extent, the red patches on which the vesicles are seated, the absence of notable irritation or pain, all point to a pathological connection with true erythema, while the anatomical lesion and its occurrence at the orifice of a mucous tract bring it into close relationship with herpes labialis. The chief difference is that, occurring as it usually does on the inner side of the prepuce or on the glans, the vesicles are broken almost as soon as they form, and superficial ulcers take the place of scabs. The condition is exactly like that of a vesicular eruption on the tongue. Like herpes labialis, it often recurs in the same patient; like it, also, it is often symptomatic of inflammation or stricture of the urethra, although it does not seem to be produced by cystitis, and certainly does not follow inflammation of the kidney as labial herpes does inflammation of the lung. It is said to be particularly frequent in persons who have suffered from syphilis. The chief practical importance of preputial herpes is its diagnosis from a soft chancre.

Herpes iris is a rare and remarkable form of eruption, well described by Willan, which is unmistakably erythematous in its nature, and is better named *Erythema iris*, or *Iris*. It occurs sometimes as a single, sometimes as two or more rose-coloured patches with all the characters of erythema, almost always upon the back of the hand, the wrist, or extensor aspect of the forearm, more rarely on the corresponding part of the foot, the instep, and ankle, and exceptionally on the face. It rapidly becomes annular, but before the ring is faded the patch of erythema reappears in the middle, and may thus be surrounded with one or (from a repetition of the process) by two or even three concentric rings. The surrounding ring may exhibit similar vesicles, or they may be more or less abortive, so that one might often question whether, if we adopt the anatomical nomenclature, we should describe the lesion as erythematous or bullous or vesicular. In its most strik-

* See an interesting autobiographical account of a case of the kind by Mr C. J. Symonds in the 'Clinical Transactions' for 1884, p. 60.

ing form, with a single large tense bleb, like one of pemphigus, surrounded by vesicular circles, the whole patch as large as a crown-piece, it is one of the most remarkable of eruptions. The inflammation is very superficial, produces little pain or irritation, and after forming thin scabs passes off after a few days, leaving more or less pigmentation, yet not a trace of scar behind. Partly the resulting pigment, and partly the rosy red of the rings, the pearly grey of the vesicles, and the more or less yellowish contents of the older bullæ seem to have combined with the bow-like form to give the title iris. The course of this curious disease, its superficial character, and its locality, all make it unmistakably erythema, as also the fact that it occurs almost exclusively in young persons; but it appears to be symptomatic of nothing.

Erythema bullosum.—Iris is not the only form of erythema with blebs.* It is not very uncommon to see rose-red patches on the forearms or back of the hand or the fingers of young people, which break into bladders as big as a split pea, or larger still, filled with serum. The same condition may be seen on the legs, and less frequently still on the face or trunk. Its short duration distinguishes it from Pemphigus, and it is often associated with more ordinary forms of Erythema or with Urticaria.

Erythema gestationis bullosum.†—There is a remarkable and rare affection which has been described as a species of pemphigus, of herpes, or of "Hydroa," or has been included under the title Dermatitis herpetiformis. Its pathological alliance appears to be with the form of erythema which depends on ovarian irritation, but the existence of bullæ makes it liable to be confounded with pemphigus.

It occurs in women during pregnancy. The bullæ, vesicles, and vesiculopustules appear in abundant crops over the trunk, and often on the face and limbs also. Pruritus is marked, more so than in the preceding kinds of erythema. There is more or less constitutional disturbance, and sometimes the temperature runs high. The clinical aspect is therefore serious, and occasionally alarming; but the result appears to be always favourable. The disease is cured by delivery. It is quite distinct from *Impetigo herpetiformis* (p. 890).

The writer has seen one instance—and others are on record—in which this remarkable form of bullous erythema appeared again and again in successive pregnancies.

Cases of "herpes gestationis" have been carefully described since Chausit and Hardy, by Dr Liveing, Dr Bulkley, and other observers in this country, on the Continent, and in America. The general features are very uniform, and there is no doubt of the reality and distinctness of the disease; but its true pathology and the means of prevention or treatment are still obscure.

Hydroa.—The group of eruptions named hydroa by Bazin is not a natural one, either clinically or pathologically. Of the three species described by him, the first, or "vesicular hydroa," would clearly seem by its localisation on the back of the hands and wrists, and on the front of the knees, as well as by its acute but sometimes recurrent course, to be erythema. Other cases are identical with the curious affection long known as herpes iris, which is itself a form of erythema (p. 897). "Bullous

* Dr Duffin ('Pathological Transactions,' 1875), Dr Crocker and Dr Frederick Taylor ('Clinical Society's Transactions,' Feb. 25th, 1881), and the writer reported two typical cases in the 'Guy's Hospital Reports' for 1880 (3rd series, vol. xxv, p. 211).

† *Synonyms*.—Hydroa (in part)—Pemphigus uterinus, hystericus, v. pruriginosus—Herpes gestationis—Dermatitis herpetiformis (in part).

hydroa" seems to be nearly identical with *pemphigus pruriginosus*, or herpes gestationis (p. 898). Some cases, again, which have been described as hydroa, have turned out to be iodide rashes.

Several good dermatologists now define Hydroa as identical with Duh-ring's Herpetiformis dermatitis (p. 888).*

Diagnosis.—When vesicles and bullæ appear in the course of an erythematous rash, we must first make sure that they are not due to local irritants, whether accidentally or designedly applied; next we must separate them from the bullous and pustular eruptions produced by iodide of potassium, from the bullous form of syphiloderma, from symptomatic Herpes, and from that which follows the course of cutaneous nerves and will be afterwards described as Zona; and, lastly, we must diagnose them as well as may be from the bullæ of typical Pemphigus, although in many cases the line is here difficult to draw.

3. *Erythema nodosum.*—This curious affection was well described by Willan, and subsequent authors have added little to his account. It occurs "in large oval patches, the long diameter of which is parallel with the tibia, slowly rise into hard and painful protuberances, and as regularly soften and subside in the course of nine or ten days, the red colour turning bluish on the eighth or ninth day, as if the leg had been bruised." In this form of erythema the anatomical lesion is especially characterised by œdema; the spots do not itch, but are somewhat painful and very tender, more so than in any other of the erythematous group. There is almost always not only deep venous congestion of the typical erythematous rose tint, exaggerated by its position on the legs, but there is almost always more or less indication of actual hæmorrhage. Probably the pigmentation, which it is apt to leave behind, is deepened by chemical transformations of the effused hæmoglobin such as we see in a bruise.

The locality of erythema nodosum is, as Willan says, most frequently over the tibia, but it is not confined to this part, for it may be seen on the ankle or the calf, and it is not uncommon over the corresponding surface of the ulna. It is usually symmetrical, and may affect the whole extensor surface of both forearms and both legs. It is very seldom seen elsewhere.

It has a slower course than most kinds of erythema, but like them is prone to recur. Willan and Bateman, and also Green in his 'Practical Compendium,' state that erythema nodosum only affects women; but Plumbe in 1824 ('Practical Treatise on Diseases of the Skin') notes its occurrence in children, and it is not unfrequently seen in boys under or about the age of puberty who are also liable to hysteria, chorea, and other female disorders.

It occurs very frequently in those who have suffered from *rheumatic fever*. Dr Stephen Mackenzie brought before the Clinical Society in April, 1866 (vol. xix, p. 215), more than 100 cases of erythema nodosum collected from the four largest hospitals in London. Ninety patients were females, and 18 males. Only 25 were over thirty years of age, 30 were between twenty and thirty, 39 between ten and twenty, and 14 were children under ten. In 17 cases there was also past or present rheumatism (acute in 13,

* On the history of Hydroa, see an elaborate paper by the late Dr Tilbury Fox in the 'Philadelphia Archives of Dermatology' for 1880, p. 16, and one by Dr Crocker in the 'Lancet' for May 22nd, 1886.

The intended meaning of the word Hydroa is uncertain. Dr Crocker gives its derivation from ὕδωρ; but surely it is only a mistake for Hidroa (ἵδρωα), the regular term in Greek medicine for what the Latins called *sudamina*.

subacute in 4), beside about as many more in which the existence of true rheumatism was asserted or probable. There was a cardiac murmur in 13 cases, in only two of which was there history of rheumatic affection of the joints.*

The course, the lesion, the œdema, the hæmorrhage, the locality, the subjects of this affection, are all typically erythematous, and so is its relation to rheumatism.

4. *Urticaria*.†—Willan rightly placed urticaria in close relation to erythema. Almost all subsequent writers have followed this indication; and if convenience did not forbid innovations, it might be called “erythema pomphosum,” for the characteristic lesions are *pomphi*—wheals, *i. e.* raised flat white patches, sometimes surrounded by an erythematous blush. Their histology is that of acute inflammatory œdema of the cutis, which fills the lymph-spaces and expels blood from the venules. The effusion takes place very rapidly, and may be called forth either by a mechanical or by a chemical irritant, as in the wheals produced by the nettle (*Urtica urens*), from which the disease receives its name. In persons liable to the affection it can be produced by the finger only drawn across the skin, so that it is possible to write characters in raised wheals. This last has been defined as “factitious” urticaria. The anatomical lesion has therefore its counterpart in the traumatic wheals produced in any skin by the sting of the nettle or the lash of a whip, and produced by much slighter irritation in susceptible subjects.

Beside the well-marked oval or linear wheals of ordinary urticaria, we sometimes see the lesion in the form of small round patches, or as large white plateaux, formed by the coalescence of several smaller ones (“giant urticaria,” *U. tuberosa*). Both these forms are sometimes produced by nettles. We may also include as essentially of the same nature the large, flat, white papules, which are obviously distinct from those of ordinary eczema, and which have been described as *strophulus albidus*, and also not unfrequently under the name of *infantile prurigo* and *lichen urticatus*. These papules are distinguished by rising rapidly, and by following, not causing, pruritus; for they are the result and not the occasion of the patient’s scratching. They are most often seen in infants, but may be observed along with more obvious wheals in ordinary cases of adult urticaria. In quite exceptional cases it is said that the wheals last, so as to produce a chronic condition (*U. perstans*). Sometimes, but also as an exception, bullæ are described as mingling with the wheals—a fresh sign of affinity between urticaria and erythema.

The *distribution* of nettle-rash is less definite than that of other forms of erythema, and, indeed, of most other cutaneous affections. We do not observe any predilection for the erythematous regions, the extensor surface of the forearms and legs. It is quite as common on the back and trunk generally as on the limbs; the only parts it avoids are the scalp, face, palms, and soles. It is not symmetrical.

* In this view of its pathology Strümpell and Boeck agree with Mackenzie and the present writer, although most Continental and American dermatologists are opposed to it.

See Barlow, ‘Brit. Med. Journ.,’ Sept. 15th, 1883, p. 511; also ‘Guy’s Hosp. Rep.,’ 1881, p. 209. Dr Cæsar Boeck has published a monograph on this point; also Dr Mackenzie’s paper on the same subject, read before the Dermatological Congress of London in 1896, p. 602, and the discussion which followed.

† *Synonyms*.—Nettle-rash—Cnidosis: including lichen urticatus, purpura urticans, and much of strophulus.—*Fr.* Urticaire.—*Germ.* Nesselsucht.

The mucous membrane of the mouth is occasionally affected, and something like wheals have been observed by the laryngoscope in the trachea; whether it affects the stomach or bowels is uncertain.

Urticaria is abrupt in origin and acute or subacute in *course*, but is often recurrent and obstinate in successive attacks.

Of all forms of erythema, urticaria is the most irritable, the severity of the itching being comparable to that of eczema, scabies, or prurigo. There is no pain or smarting, and no general symptoms except from the restlessness and sleeplessness which it occasions, especially in children. It is most frequent in them or in young adults, but is not confined to any age. According to Dr Liveing it sometimes alternates with neuralgia of the same parts.

The *ætiology* of urticaria is uncertain. As above explained, it is often purely secondary to some local irritant, as pediculi, or complicates a previously existing malady, as prurigo; and it is probably always aggravated by the patient's scratching. It is often of toxic origin; animal, as in the case of Medusæ and the larva of moths, or vegetable, as in that of stinging-nettles: or the poison may, instead of being absorbed from without like a subcutaneous injection of morphia, be swallowed first; as with belladonna and strawberries, or whelks and crabs, or copaiba.

Its close alliance with erythema is shown not only by sometimes alternating with it, but also by its following precisely the same kind of gastric disturbance, both in the most marked forms, which are the direct result of drugs or of poisons, or of some particular article of food, and in the less evident cases associated with ordinary dyspepsia.* When there is no internal irritant, and no gastric disturbance, urticaria in women can often be traced to ovarian disorders, breaking out at each menstrual period, and subsiding when dysmenorrhœa is cured. Like erythema, again, it is, according to the writer's experience, a not infrequent complication of rheumatic fever.

All forms of erythema are liable to be complicated with hæmorrhage. This is a rare complication in urticaria (when it constitutes the *Purpura urticans* of Willan), though common in erythema nodosum, when it produces the subsequent bruise-like pigmentation, and also in the forms of erythema which occur in the course of rheumatic fever.

This last condition seems to have been first observed by Schönlein, who named it *Peliosis rheumatica*. The erythematous patches appear acutely, with fever and synovitis. They are most often seen on the back of the hands and feet, the forearms and shins, but may also affect the thighs, hips, and trunk; they are not often symmetrical. Either from the beginning, or soon after their appearance, the redness is found no longer to fade on pressure; hæmorrhage has taken place. Successive crops of these papules or patches may occur, each lasting about a week, and disappearing with only a slight macule to mark its place.

There is no need of a special name for this disorder. It is a true erythema, whether occurring in the course of rheumatic fever or in persons

* Dr Osler has drawn attention to an article by Schliesinger in the 'Münchener med. Wochenblatt,' Aug., 1899, on *Hydrops hypostrophos* or Angioneurotic œdema (both, one may remark, unnecessary synonyms of what is sufficiently plain as acute circumscribed œdema), and has published an interesting series of cases in which recurrent urticaria, with often concomitant purpura, local acute œdema, rheumatism, and cardiac disease were associated with internal crises of gastralgia, colic, hæmatemesis or hæmaturia, and synovitis with effusion. The relation of acute œdema of the lips, tongue, or fauces to urticaria has been admitted by several observers, but the internal crises are perhaps more doubtful pathology ('Brit. Journ. of Derm.' 1900 v. 227).

who have already suffered from that disease, and we have seen that both this connection with rheumatism and the liability to hæmorrhage are characteristic of the whole group of erythemata.

Urticaria pigmentosa.*—A similar and rare form of skin disease belonging to the erythematous type, but recurrent and as persistent as erythema nodosum, was first described by Mr Edward Nettleship, and again by Morratt Baker and Tilbury Fox, in the Clinical Society's 'Transactions,' vols. viii and x. Two cases, in a child of two years old and in a woman of thirty-two, were described by the writer in the twenty-fifth volume of the 'Guy's Hospital Reports,' 3rd series, pp. 212, 213.

It has received several names, among others the uncouth and misleading term Xanthelasmaidea; but Dr Sangster's proposed title, urticaria pigmentosa, is now generally accepted.

It is an erythematous eruption with occasional wheals and considerable yellowish pigmentation, lasting for an indefinite period, though its chronic course is probably always made up of more or less distinct subacute attacks. It affects the back and trunk generally, rather than the limbs, and the face very little. The characteristic buff colour always follows the rose-rash.

An excellent account of this affection is given by Dr Colcott Fox in the 'Med.-Chir. Trans.' for 1883, where nineteen recorded cases are tabulated, to which Crocker has since added one ('Clin. Trans.,' vol. xviii, p. 12).†

Dr Paul Raymond has written a valuable monograph on this curious malady, in which he has collected sixteen cases observed in England, two in America, eight in Germany, and four in France. All these cases occurred in children under two years old, often within a fortnight after birth. The majority of the infants were boys.

In an exceptional case recorded by Lewinski it was still present in a lad of eighteen. In one case under the writer's care a condition which might be called *E. perstans*, had been present for four years on the trunk, and was followed by decided pigmentation.

Microscopical examination of the affected skin by Dr C. Fox has shown that the lesion is truly a wheal, the tissue of the corium being opened out by œdema. Pick had found minute hæmorrhages in another case.

The affection recurs again and again, but as the child grows older gradually ceases, and the pigmentation still more gradually fades.

Statistics.—In 102 consecutive cases of the writer's, there were 38 of *Erythema multiforme* (marginate, circinate, annular, gyrate, papular, and roseolar), 17 of *E. bullosum*, including one of *Herpes gestationis*, two of *H. iris*, 13 of *E. nodosum*, 18 of Urticaria, and 14 of multiform erythema and urticaria both present in the same subject. Several cases of erythema and of urticaria occurred during, or after, rheumatic fever, and four cases of urticaria came immediately after the patient had eaten shell-fish.

There was in each group (except Urticaria, where the numbers were almost equal) a decided preponderance of female patients. In addition to 4 infants under two years old (one with *Urticaria pigmentosa*), there were 33 patients between four and sixteen, 48 between seventeen and thirty-five, and 14 above thirty-five.

* *Synonyms*.—Urticaria perstans pigmentosa—Xanthelasmaidea—Erythema tuberculatum—Permanent erythema.

† See also Dr Cavafy's article in Heath's 'Dictionary of Surgery,' and Dr Stellwagon's three cases in children ('Journ. Cut. and Gen.-ur. Dis.,' December, 1896).

Treatment of erythematous affections.—The various erythematous diseases which we have grouped together in this chapter are seen in their true relationship from a practical point of view. They are none of them contagious, they are none of them attended with serious consequences, they are mostly indicative of some other primary disorder, and they are rather to be palliated by local applications or indirectly cured by treating their internal cause when discovered, than met by a specific plan of treatment. In particular it may be said that they are either unaffected or aggravated by arsenic, and this is one of the most important points which separate them from pemphigus.

In many kinds of erythema, especially symptomatic herpes and iris, no treatment is needful.

The local treatment of the other erythemata consists in the astringent and sedative applications described at p. 835; although the surface is dry, it is found by experience that lotions in most cases answer better than ointments. Goulard's wash (Liq. plumb. subacet. dil.) evaporating lotions of spirit and water, eau de Cologne, hydrocyanic acid well diluted, or solution of borax are the best local applications. When urticaria is severe and these means fail, chloral hydrate may be used locally in solution or chloroform and ung. cetacei (℥x—xx ad ʒj). Warm baths should be avoided, as also excessive heat and perspiration; tepid water is better than either cold or hot. The patient should be urged to stoical abstinence from scratching; tepid bathing or continued steady pressure will be found to relieve the intolerable irritation of urticaria without aggravating it afterwards as scratching always does.

For the painful swellings of erythema nodosum, strong lead lotion gives most relief, or lead and opium. Collodion painted over and allowed to dry is often useful, or alum, tannic acid, or other astringent remedies may be used with advantage, or the affected part of the leg may be painted with a solution of nitrate of silver. Ointment containing boric acid or lead may be applied to the herpetic and bullous forms of erythema.

In the internal treatment our first care should be to relieve the gastric disorder which often accompanies common congestive or papular erythema—most often by discovering certain articles of food to which it is due. Salt fish, pickles, preserved fruit and “crystallised” sweetmeats, pork, sour or otherwise inferior wine, malt liquor, stone fruit, and even strawberries—any one of these may in certain persons excite erythema or urticaria. Those first on the list should be strictly forbidden. Of all kinds of food, lobsters and crabs, and by a curious coincidence mussels and other molluscs united with them under the title of “shell-fish,” are the most frequent causes of erythema or urticaria, and they are the most severe in their effects. Oysters are harmless. Many drugs have a similar result, as we shall presently see.

If the eruption continues after its supposed cause is removed, or if we are unable to discover any cause of disorder, such remedies as bicarbonate of soda or Liquor bismuthi or a few drops of liquor potassæ in peppermint- or cinnamon-water should be prescribed. Where there is evidence of gastritis, bismuth is the most valuable remedy, given either in powder or thus:—℞ Bism. subnitr., sodæ bicarb., pulv. tragac. co., āā gr. x, aq. chloroform. sive menthæ pip. ʒj; M.; to which ten or twelve drops of solution of morphia may be added if the pain is severe. With flatulent disorder, thymol, creosote, or carbolic acid (phenol) in the form of pills is often the

most effectual mode of treatment. In the more atonic forms pepsin given after meals is found practically useful, notwithstanding our physiological doubts; and occasionally dilute mineral acids with *nux vomica* or bitter infusion will be more valuable than anything else. Gentle saline laxatives taken before breakfast in a large draught of warm water are almost always indicated; and, for women especially, a pill containing aloes or rhubarb, taken before a late dinner or on going to bed, is a useful adjunct. In many patients occasional doses of blue pill are of unmistakable value.

In cases of erythema nodosum and in other forms of erythema which follow rheumatic fever, and occur in pale young women or lads, the preparations of steel are strongly indicated. When there is constipation, a good formula is three or four grains of sulphate of iron, half a drachm of sulphate of magnesia, and five drops of dilute sulphuric acid, in peppermint water or calumba. When this is not the case, the tincture of steel is a most valuable remedy. In some cases sulphate of iron with carbonate of potash and extract of Barbadoes aloes forms a good Martial pill. In whichever form iron is found to agree best, it is important to increase the dose until a decided effect is obtained.

In the more severe forms of recurrent bullous Erythema, quinine in full doses is of great value, particularly when there is much itching. Opium usually increases rather than relieves the irritation, but it has been given in full doses with advantage in the more severe cases.

ERUPTIONS PRODUCED BY DRUGS.—Since the most frequent and characteristic effects of drugs upon the skin are erythematous eruptions, it will be convenient to consider this group of dermatoses here. Drugs and poisons act much in the same way upon the skin as do irritant or poisonous articles of food, so that in origin as well as appearance the rash caused is an erythema.

The most striking and frequent of these eruptions is perhaps that produced by *copaiba*. This has sometimes been confounded with an early syphilide. It usually takes the form of a papular erythema, often combined with urticaria and not unfrequently with slight subcutaneous hæmorrhage. The occurrence of bullæ or vesicles is mentioned by trustworthy observers. In some cases there is no itching, which makes the diagnosis from syphilis the more difficult. The rash is generally distributed over the whole surface of the body, and does not spare even the face, as most other erythemata do. Occasionally it simulates purpura. Some writers have suggested that it is not the *copaiba* but the urethral inflammation for which *copaiba* is commonly given, which produces the rash. There can, however, be no doubt of the existence of a true *copaiba* rash. It is not uncommon from the exhibition of the oleo-resin, but is rarely observed in persons who are taking the valuable diuretic, *mistura copaibæ resinæ*.

Cubebs is generally said to produce a similar eruption, but some of the reported cases appear to have been due to accidental mixture with *copaiba*. So, at least, Dr Bulkley believes. The writer has never seen a *cubebs* rash.

Somewhat similar rashes have been observed in patients taking *turpentine*, *cannabis indica*,* and some other drugs, and have been described as purpura, urticaria, herpes, or erythema *a medicamentis*. It is possible that some at least of the eruptions ascribed to salicylic acid were really peliosis rheumatica.

* In a case reported by Dr. J. N. Hyde in the 'New York Medical Record' for May 11th, 1878.

Bromide of potassium comes, perhaps, next in frequency to *copaiba* as a rash-producing drug. The lesion here simulates very closely that which will be described in the next chapter as *acne*, but the diagnosis is generally clear from its not being confined to the very characteristic localities of true *acne*, or to the equally characteristic age which is specially liable to that disease. Occasionally the bromide eruption is more severe, and produces pustules and crusts. The late Dr Carrington reported a remarkable case of solid tumours in an infant, apparently the result of the exhibition of bromide ('Clin. Trans.,' vol. xviii, p. 28, with plate); and also Dr Lees ('Path. Trans.,' vol. xxiii, p. 247, with plate).

Less frequent, but much more varied, more severe, and more misleading is the eruption produced by *iodide of potassium*. This is, perhaps, most frequently a papular erythema, widely or irregularly distributed on the trunk, limbs, and face, free from itching, and usually unfelt by the patient. Sometimes, however, there is considerable erythematous dermatitis between the papules. A follicular inflammation undistinguishable from that described above as bromide-*acne* is a less frequent effect of the iodide salts. More often the rash which was at first papular becomes vesicular, bullous, or pustular. In these cases the inflammation is often very severe, and the constitutional disturbance considerable. The bullæ have been, there is no question, often confounded with pustular syphilis and so-called "hydroa;" indeed, until one has seen several cases, it is difficult to believe that so severe a dermatitis can be due to a drug which in most cases has no effect whatever upon the skin. The eruption may simulate scabies or eczema, but the absence of definite localisation, of chronicity, of the secretion of eczema or of the cuniculi of scabies, should make the diagnosis not difficult. Along with the pustules there may arise what the older dermatologists would have called a tubercular disease of the skin—raised fleshy nodules simulating papillary growths, condylomata, mucous patches, and the later forms of syphiloderma. They may resemble *rupia* or *lupus*, or even malignant disease. Inasmuch as these severe effects are apt to follow the large doses of iodide of potassium given in the later stages of syphilis, the difficulty of discriminating them is naturally increased.

In a patient under the writer's care suffering from an ordinary pustular syphilide, some of the lesions on the face and the back of the hand became so swollen, hypertrophied, and covered with profuse granulations, that both cheeks were deformed, the eyes almost occluded, and one hand was covered with exuberant granulations, which, when seen alone, suggested to different observers *lupus hypertrophicus* or *epithelioma*. There was, however, no doubt of the nature of the case. The diagnosis was confirmed by the patient's recovery as soon as the drug was discontinued. The chief point which guided one aright in this case was that, notwithstanding his frightful appearance, the patient was eating and sleeping well, so that it was with great difficulty he was persuaded to come into hospital.

Another form of iodide rash is punctiform, and resembles scarlatina rather than measles, of which the patches and rose tint are more nearly simulated by the *copaiba* rash. This iodide eruption is often purpuric, particularly when it affects the legs (Dr Duffey, 'Dublin Journal of Medical Science,' vol. lxi, April, 1880).

More often pustules may appear, and when deep and occupying a hair-sac, cause crops of boils. The presence of iodine and bromine has been actually demonstrated in the pustules by Adamkiewicz and Guttmann.*

* 'Virchow's Archiv,' 1878, vol. lxxiv; 'Charité-Annalen,' vol. iii, p. 381, 1878.

The iodide produces its effects on the skin much more rapidly than the bromide. In both cases there appears to be a true excretion of the drug through the sebaceous glands. For an account of the histology see a paper by Dr Thin ('Med.-Chir. Trans.,' vol. lxii, p. 189).

Weeping dermatitis, curious wart-like nodules, and other peculiar eruptions have been described as the result of bromide of potassium by Voisin and Veiel, quoted by Behrend ('Berlin. klin. Wochenschrift,' vols. xvi and xxii, pp. 626 and 714, 1879). Two cases of severe iodide eruption were figured by the late Dr Tilbury Fox ('Clin. Trans.,' vol. xi, 1877).

Various measures have been adopted to prevent these unpleasant effects. The addition of carbonate of potash or aromatic spirits of ammonia is sometimes sufficient. Moderate doses of arsenic have been recommended, but they often fail. Changing the potash to the soda salt of iodine is sometimes followed by the disappearance of the rash; but persevering with the drug in even larger doses is often followed by the disappearance of the unpleasant effect it had produced.

Belladonna in full doses often causes a bright-red and almost universal erythematous rash. It may be recognised by its association with dilated pupils and a dry throat, together with the characteristic delirium if the dose has been large. In one case under Mr Hilton it was caused by the mere application of a large belladonna plaster in a woman who must, one supposes, have been more than commonly susceptible. Children, who bear as large doses of this drug as adults, are also liable to these symptoms of intoxication. A few years ago more than a dozen children were admitted in Guy's Hospital with all the symptoms of belladonna poisoning. They had broken into a drug warehouse on a Sunday and had eaten some of the contents. The rash in most of them was remarkably like that of scarlatina.

Opium and *morphia* sometimes produce considerable pruritus, and this may lead to erythema or urticaria by the scratching which results; moreover, *morphia* has occasionally produced a morbilliform rash. Similar rashes have been observed as the result of *hyoscyamus* or *stramonium*.

Chloral hydrate.—Mr Hutchinson has figured an erythematous rash of the hands due to chloral in his 'Archives of Surgery,' vol. i, pl. v (cf. 'Clin. Trans.,' xiii, p. 121). In one instance the writer observed a severe general acute dermatitis follow the exhibition of *chloralamide* (*ibid.*, vol. xxiii, p. 137, with plate).

Quinine.—There can be no doubt that quinine may produce a general acute erythema, which was first described by Skinner, Fleming, and other English authors, and has since been observed abroad. Its symptoms closely resemble scarlatina. It begins in the face, spreads rapidly over the whole trunk, and is accompanied by fever, the temperature sometimes reaching 103.5° Fahr. It is certainly a very rare effect of so popular a medicine, and its occurrence may be regarded as due to an idiosyncrasy. In one case of Köbner's the eruption followed the exhibition of quinine three times in the same patient. A more severe local erythema of the face, sometimes vesicular, has been observed as the result of quinine by Hebra, von Heusinger, and some other physicians. (See a case in a child reported by Dr H. Hagan, of Atlanta, in the 'New York Medical Journal,' 1891.)

Dr Morrow, who has collected sixty cases of quinine eruptions, found that in thirty-eight the rash was erythematous and resembled that of scarlatina, in twelve it resembled urticaria, in two it was vesicular, and in five hæmorrhagic ('New York Medical Journal,' March, 1880). One case was

reported by Dr Fagge ('Medical Times,' February 29th, 1868); another by Dr Farquharson ('Brit. Med. Journ.,' February 15th and 22nd, 1879).

Antipyrin (more properly *phenazone*) has occasionally produced an extensive erythematous rash, as recorded by Brocq, Darier, and others.

Eruptions from *salicylic acid* * have been reported. Since erythema and urticaria are common in rheumatism, for which salicylic acid is usually given, and since adulterations with carbolic acid and consequent gastric disturbances are not unknown, some of these cases may admit of doubt.

The internal use of phenol (*carbolic acid*) itself, of *tar*, *turpentine*, and *petroleum* has caused erythematous rashes, but the cases are rare.

Arsenic is said to produce in some persons an acute vesicular eruption, which has been styled herpes, and in others urticaria. This occurrence is, however, very rare even when large doses of the drug are given. Zonaf has sometimes appeared during a course of arsenic, too frequently to be considered a mere coincidence.

Arsenic occasionally causes pigmentation of the skin after long administration; and another effect is hypertrophy of the horny epidermis, discrete on the palms and soles, or diffuse on the elbows and knees.

Mercury was one of the first drugs to be regarded as the cause of a cutaneous rash. Early in the last century Alley† described what he called Hydrargyria, before the first description of a copaiba nettlerash by Montègre in 1814. Alley's cases were mostly vesicular, and chiefly affected the abdomen, thighs, and scrotum, but sometimes they assumed a more severe pustular or bullous form, with severe pain and lymphatic inflammation. We may doubt whether local inunction of the drug, or the effects of syphilis itself, or a mere coincident attack of eczema, may not explain these cases. Hebra, with a scepticism justified by his enormous experience, denied that any eruption is due to the internal use of mercury. On this point see Judassohn's Essay, 1900.

The earlier bibliography of the whole subject is given at the end of a paper by Van Harlingen in the 'Archives of Dermatology,' Philadelphia, October, 1880. See also Köbner's article (Internat. Med. Congr., 1892); Morrow's 'Drug Eruptions' (Syd. Soc., 143), 1887; the article in 'Allbutt's System,' vol. viii, by Mr Hutchinson, jun.; the chapter on "Dermatitis Medicamentorum" in Crocker's 'Diseases of the Skin,' and that under the same title in Hyde and Montgomery's sixth edition, 1901, p. 207.

Among the antitoxic remedies now used in the treatment of more than one specific disease, diphtherial antitoxin is remarkable for frequently producing not only multiple synovitis, but also an erythematous eruption which lasts a short time, and then disappears without leaving traces. These effects are probably due to a mixed infection, and have of late been diminished in frequency and severity by improved methods of preparing the serum. A similar rash, more irritable and severe, sometimes follows the use of antistreptococcic serum, and even, it is said, of serum from healthy horses.

* Cavafy, 'Clinical Society's Transactions,' vol. x, 1877, p. 88.

† Hutchinson, 'London Hosp. Reports,' vol. v; Hans von Hebra, 'Die Krankhaften Veränderungen der Haut,' p. 204. He is convinced that the relation is accidental.

‡ 'Observations on Hydrargyria,' Dublin, 1804; London, 1810.

DISORDERS OF THE SEBACEOUS GLANDS, THE HAIR-SACS, AND THE SWEAT-GLANDS

"If Rosalinda is unfortunate in her Mole, Nigranilla is as unhappy in a Pimple."

ADDISON.

ACNE—*Nomenclature—Anatomy and course of local lesions—Distribution—Age and sex—Symptoms—Ætiology—Treatment—Acne tarsi—Acneiform eruptions produced by tar—by bromide—Acne varioliformis.*

Comedones without inflammation (acné cornée)—Miliū—Seborrhœa oleosa—Seborrhœa sicca—Seborrhœa corporis—Seborrhœic eczema—Lichen circumscriptus—Xerodermia—Steatoma meliceris and sebaceous cysts.

SYCOSIS—*Name—Anatomy and course—Locality—Diagnosis—Distribution—Treatment—Parasitic sycosis—Sycosis capillitii frambœsiformis.*

FURUNCULI—*Pathology—Anatomy—Course and distribution—Age—Contagion—Treatment—Carbuncle.*

AFFECTIONS OF THE SWEAT-GLANDS—*Anidrosis—Hyperidrosis—Bromidrosis—or fœtid sweat—Chromidrosis—Hæmatidrosis—Sudamina.*

IN the long series of inflammatory diseases of the skin we find certain affections which may be arranged, on clinical as well as anatomical grounds, in a third large group. The first we considered was that of the chronic forms of dermatitis which in various degrees resemble the common superficial inflammations produced by irritants. Traumatic eczema, idiopathic, symmetrical, weeping eczema, lichen planus, pityriasis rubra, and psoriasis—these form a natural group, of which pemphigus is an outlying member. The erythematous group of affections treated in the preceding chapter, to which pemphigus may be considered as the link, forms a second family, as natural though not so extensive.

The present chapter deals with inflammatory processes which do not affect the skin generally, but only the hair-sacs and cutaneous glands. They have also, as we shall see, peculiarities of distribution and of natural history which are no less characteristic than their anatomy.

ACNE.*—This disorder is referred to by Cicero, Martial, and other classical writers, but as a blemish rather than a disease.†

* *Synonyms.*—Acne vulgaris—Acne disseminata.— *Germ.* Finnnenausschlag.

The derivation of the word is unknown, but is commonly supposed to be a corruption of ἀκμή, and to refer to its occurrence in the prime of life. Its proper Latin name was *varus*, and it was called ἰσθός by the Greeks, and also ἀκμᾶι.

† "Pæne ineptiæ sunt curare varos et lenticulas et ephelidas; sed eripi tamen feminis cura cultus sui non potest" (Celsus, 'De Med.,' lib. vi, cap. v).

The sebaceous glands become occluded, either by their secretion being too thick or by want of cleanliness in removing the accidental obstructions from dirt. The first effect is to produce a number of small, firm, and somewhat pointed papules (*comedones*), each produced by accumulated sebum and marked by a black head, which is nothing but the dirt obstructing the orifice of the gland. This condition, which has been named *acne punctata*, may continue for an indefinite time, but sooner or later some of the papules show signs of purulent infection, and in most cases this very speedily supervenes in each obstructed gland. The papule becomes red, swollen, and before long yellow from suppuration having taken place. This pustular form or pustular stage of acne is no less characteristic. When the surrounding inflammatory œdema is considerable the deformity is of course increased. At last the minute abscess bursts, and the inflammation slowly subsides. When slight, no trace remains, but a second inflammatory process with the same course and termination often follows. When severe, a minute white scar is left behind, the gland is destroyed and incapable of renewed action. If the hair-sac into which the sebaceous gland opens is deep, the inflammation is the more acute, and the pain and swelling approach those of a boil. The face or shoulders may be seen covered with acne spots in all the above stages, so that the pain and irritation become great and the deformity distressing. The chronic form used to be called *acne indurata*.

Histology.—A section of an acne pustule shows not only the papillæ but the deeper layer of the cutis œdematous and filled with leucocytes, and the small blood-vessels dilated. In the pustular stage the leucocytes increase in number and assume the character of pus-corpuscles; the acini and duct of the gland are filled with pus, often mingled with blood-discs. The process in the larger acne-pustules is found to affect the hair-sac into which the sebaceous gland opens, so that the hair itself is uprooted and the entire follicle destroyed. When the destruction of the papillæ has taken place—in other words, when the inflammation has become “deep” instead of superficial—a scar always results after the acne is cured.

Dr Living has found that in a sebaceous sac which is the seat of a comedo many minute abortive hairs may often be found, the growth of which may perhaps be the immediate cause of obstruction.

It may here be observed that the microscopic parasitic mite known as *Demodex folliculorum* is frequent in healthy sebaceous glands and never causes acne.

No doubt the pustular stage of acne depends on the presence of pyogenic microbes, which have gained entrance into the sebaceous sac and caused a pustule, or which may penetrate deeply into the hair-follicle and cause a furuncle. But according to Dr Gilchrist's observations, while *Staphylococcus pyogenes albus* is frequently present, there is also a special acne-bacillus (or possibly *Actinomyces*), which causes the specific infection of acne, and this he has identified by cultivation as well as microscopically.*

Distribution.—Acne is confined, almost without exception, to the face, shoulders, and chest. It usually begins about the forehead, the cheeks, the alæ of the nose, and the chin; but pimples may cover the whole of the face, and the intervening skin be occupied by an erythematous dermatitis.

* Reprint from the “Welch Memorial Essays” in the ninth volume of the ‘Johns Hopkins Reports,’ 1899.

Comedones, as the black-tipped early lesions of acne are called, are also to be generally seen on the auricle, but here they rarely suppurate. On the *back* the pustular and indurated form is more common, perhaps because it is more apt to be neglected; there it is that we see the most extensive cicatrices, and there we find acne lingering in men of thirty and upwards, who have long ago got rid of it from their faces. The lesion may extend from the back of the neck and the scapular and interscapular regions down to the waist, but very seldom lower, nor does it pass round the flanks towards the chest and abdomen. A few scattered papules may be sometimes found over the deltoid and upper arm or on the buttocks. The skin over the *sternum* is the least frequently affected of the three acneic regions. The lesions are precisely the same, and never extend to the abdomen, the axillæ, or the front of the neck.

Occasionally isolated comedones or follicular pustules may be found elsewhere, most often on the outer side of the thigh and peroneal surface of the leg, in men with coarse hairy skins and large follicles. They are, however, not more common in the subjects of acne than in other persons.

Age and sex.—This singular follicular inflammation is in its origin and greatest extent confined to the age of puberty and early adult life, although acne when thus begun may continue up to thirty or even later.

Comedones may be occasionally seen in children; but though numerous and apparently characteristic, they do not suppurate, and they are found upon the forehead and even on the scalp, without the characteristic distribution of true acne.

Recurrent erythema, and especially that form which will be described as gutta rosea, may lead to pustular inflammation of the hair-sacs, so that the latter affection has been commonly described as *acne rosacea*; but the distribution, the origin, and the whole natural history of the two diseases are different. In fact, the more closely the subject is studied, the more decisively does true acne separate itself from all other affections.

The disease most commonly begins in lads of about sixteen, that is to say, when the period of puberty has already begun. It is not common for it to make its first appearance after the beard has begun to grow, but it may begin at from seventeen or eighteen up to one or two and twenty. It is very slow in its progress, and the worst cases are usually those of a year's standing or more. When once thoroughly established, the morbid process continues until the beard has fully grown; in most cases it then begins to subside, and seldom continues after the age of thirty. When acne occurs in a patient above this age, it is usually confined to the back and has been preceded by ordinary acne of the face. This, as well as the occasional occurrence of severe acne of the shoulders with only slight affection of the face, is probably sufficiently explained by the greater attention given to a visible eruption, and the less efficient treatment of all affections which cannot easily be reached.

Although the evolution of acne is, as we have seen, so closely connected with that of the beard at puberty, yet the disease is far from being confined to the male sex. Indeed, Erasmus Wilson in the first edition of his treatise supposed that acne occurs more frequently in the female than in the male; and Dr Bulkley reports the same from New York, where he found, in nearly a thousand cases of acne, 319 occurring in men to 654 in women. This is not the writer's experience, but acne is common enough in young women

about the same time as in lads, or perhaps a little later. Perhaps there are more male sufferers, but more female patients.

The affection is, as a rule, more diffused in the case of women, the papules more numerous, not so large, and with more erythema between. It is also more often confined to the face; and it is certainly much more rare to see the worst forms of acne indurata, and the disfigurement which follows, in women than in men. This is due no doubt to women taking more pains to follow out treatment, while some youths take no care of their faces.

While acne is somewhat later in its appearance in women, it is decidedly slower in its disappearance, so that acne may be more often seen about the age of thirty in women than in men, and it is chiefly in women that a lingering acne is overtaken by an early gutta rosea, a combination which has no doubt helped in confusing the two disorders.

Course and symptoms.—Acne is always a chronic affection, lasting, if left to itself, for years, but liable to occasional exacerbations. These often coincide with ovarian disorders in women; in men they are less marked, but sometimes appear to be connected with gastric disturbance, especially with the more acute forms of indigestion, such as in some people result from eating pork or salmon or preserved viands, whether salt, like herrings, or sweet, like crystallised fruits.

There is but little local irritation, and the other organs are completely unaffected; indeed but for the disfigurement, few patients with acne would apply to the physician.

Ætiology.—The immediate cause of acne is the obstruction and inflammation of the sebaceous glands, and in the severer cases of the hair-sacs also; and the characteristic pustular stage is due to infection by pyogenic microbes as above stated. But when we ask why this obstruction occurs, the answer is extremely difficult. To say that the presence of acne indicates a disordered state of the cutaneous nerves, which interferes with the vascular action of the skin; or that it depends on torpidity of the capillary circulation; to say with Bielt that it is the result of keeping the head bowed down, as in many sedentary occupations, or of drinking cold water when heated, or of smoking tobacco; or with Alibert, that it is caused by spending nights in gambling and living in anxiety—none of these explanations help us.

It may be asserted that acne has not the connection with cold which chilblains have, nor depends on local irritation as eczema solare, nor on gout or tubercle, nor on the ingestion of cold water, or hot water, or alcohol, or any kind of food, nor on any diathesis or disposition to anything but acne.

It is obvious if we consider its natural history, that acne has to do with the great change which passes over the organism at the time of puberty; first and principally with the growth of the beard, yet not as a mere mechanical result, for in the great majority of men the beard appears without acne,—men may have acne long before they develop a beard, and women have it also. Acne, moreover, affects the skin of the shoulders, which is unchanged at this period, as well as that of the face and chest, where hair grows; and it does not affect the hair of the pubes.

It is stated by Rigler ('Die Türkei und deren Bewohner,' Wien, 1862), quoted by Hebra, that acne, though common in the Levant, is extremely rare in eunuchs. There is no reason to adopt the suggestion of Rayer, followed by many French writers, that acne is connected with vicious habits.

The old adage of Plenck, "*Matrimonium varos curat*," is well exchanged for Hebra's dictum, "*Tempus varos curat*." It is not continence nor vice, nor celibacy nor marriage, nor even the growth of a beard, which are the causes of acne; it depends upon the general changes which occur in the passage from childhood to adult life. The glandular apparatus of the skin is then apt to be disordered, most apt on the region where the beard is developing, and loses this aptitude when complete development is once attained. Acne never attacks a scalp or beard covered with hair nor yet the palms or soles or other part devoid of hair. But when the hairs are small and weak (*lanugo*) the opening of the sac and its sebaceous glands is small and insufficient for the large and strong growth of adolescence. With regard to acne in women we can only say (as conversely of hysteria in men) that though they have no beards their fathers had—that is that secondary sexual characters are more or less transmissible to both sexes.

Acne attacks those in good health and those in ill-health, the blonde and so-called lymphatic, as well as the dark and atrabilious; it is said to be seldom seen with red hair, and to be less common in Ireland than in England.*

Dr Erasmus Darwin, who properly distinguished acne from gutta rosea (in his '*Zoonomia*'), nevertheless named the former affection *gutta rosea hereditaria*, "because it seems to be hereditary, or at least has no apparent cause." There is, however, no proof that acne is an hereditary disease, although it is not infrequently seen in brothers and sisters, and although the disposition to its development at puberty would, we might expect, be transmitted more or less completely, in the same way as the early growth of a beard, its weakness or abundance, and the early or late supervention of baldness.

Prognosis.—Few cases of acne cannot be decidedly relieved by careful treatment, and in many the face can be restored to its natural appearance; but success depends not only upon the physician's adapting his treatment to the wants of each case, but also upon the perseverance with which the patient will follow it out. In many cases irremediable mischief has already been done when the patient comes before us. "*Tempus varos curat*," though generally true, proves often tedious in performance, and when such a cure is complete the disfigurement it leaves is often considerable.

Treatment.—In the early stage of acne, when comedones are present with little or no inflammation, the principle of treatment is to set free the obstructed ducts, to keep them clear by extreme care, and to stimulate the local circulation. The plan found most successful is the following:—On going to bed the face should be first steamed over a basin of boiling water. It should then be thoroughly washed with a piece of flannel and yellow soap, and dried with a rough towel. On careful scrutiny in the glass the patient will then find that the acne punctata has lost a good many of the black points; but he should go over the whole of the face, and wherever a pimple shows by the slightest point of yellow that suppuration has begun, it should be emptied—not by squeezing with the fingers, but by pressing over it the end of a key or a short glass tube of suitable diameter. When this has been effectually done, the face should be again washed and a lotion applied which should be allowed to dry. This drying lotion may be of sulphur

* According to Bazin acne is of scrofulous origin; and even Hardy, while denying this, thinks that acne has a preference for lymphatic subjects, although "on peut avoir un tempérament lymphatique sans être atteint de scrofule."

suspended in liquor calcis, alum water or lead lotion, or a dilute solution of corrosive sublimate (gr. $\frac{1}{2}$ with tr. benz. co. ʒss in ʒj of mist. amygd.). The old cosmetic known as *lac virginum* was of somewhat the same composition, as was also the famous Gowland's lotion, which is said by Bateman to have contained oxymuriate of mercury in an emulsion of bitter almonds.* The sulphur is the more stimulant; the mercurial wash when too strong is apt to cause a feeling of constriction and tension of the skin.

Next morning any fresh pimples which have ripened should be emptied and the face again washed with soap and water, and a little dilute mercurial ointment (ung. hydr. ox. rubri with two parts of benzoated lard) applied to each. With many slight cases thorough washing and the application of white precipitate ointment is sufficient.

The same plan of treatment answers, even if many pustules are present, supposing that there is not much inflammation around them; but the more pustules there are, the less vigorous should be the friction used, and the more important it is to apply some ointment containing mercury to the pustules. Dilute citrine ointment is often well borne; in other cases the unguentum metallorum (p. 826, *note*) suits better.

When the inflammation, judged of by the erythema between the papules, by the amount of swelling, or by the presence of true furunculi, is severe, we must begin with other measures. Steaming is still useful and generally proves soothing, but friction must be much more sparingly used, and sometimes omitted altogether. Instead of stimulating lotion or ointment, Goulard wash must be used at bedtime; or a drying lotion of oxide of zinc suspended in water, or the almond wash may be used alone; during the day lead ointment or zinc or a combination of the two must be applied, but for women and others who are not obliged to be out of doors, the frequent application of lead lotion is better than ointment.

It is in these cases only that diet needs regulation by abstinence from stimulants, spices, and the other viands which we found tend to excite erythema of the face (p. 903). It may also be desirable for the patient to take a little carbonate of soda or bismuth with a saline laxative. By these means the erythematous inflammation will soon be subdued, and it is then desirable to return as quickly as may be to the more stimulant treatment. The sulphur lozenges of the Pharmacopœia are excellent laxatives, and perhaps have a specific effect when excreted by the skin.

In inveterate cases the stronger mercurial ointments are indicated. When, as occasionally happens, furunculi are present, they must be treated with carbolic oil to prevent purulent infection.

Acne of the shoulders, though often severe, is naturally less troublesome than that of the face; the skin is also less susceptible to irritation, and almost always bears rougher treatment with advantage. The individual attention to the several papules, which is so important in the case of the face, is difficult to carry out here, and we must depend more upon the use of mercurial ointments and on friction with rough towels or flesh gloves.

Acne tarsi.—Anatomically allied to acne is the inflammation which not unfrequently, especially in children, affects the large and specially modified sebaceous glands that serve to lubricate the eyelashes. These Meibomian glands are apt to become the seat of acute and painful suppuration (*hordeum*, styte). Chronic inflammation, with a gummy secretion which sticks

* "Merely Gowland," said Sir Walter Elliot, "I should recommend Gowland, the constant use of Gowland, during the spring months" ('Persuasion,' vol. ii, chap. 4).

the eyelids together, may either occur independently or as a complication of ophthalmia. It is generally cured by the application of unguentum hydrarg. ammon. or yellow oxide of mercury ointment.

Tar and bromide acne.—Inflammation of the sebaceous glands, papular or pustular, is occasionally called forth by external irritants, and especially by tar. This so-called tar-acne differs, however, in its distribution and natural history from the true disease, and needs no treatment but the removal of the exciting cause.

Bromide-acne is the name given to a somewhat similar follicular inflammation caused by the internal use of the bromides in certain patients.

*Acne varioliformis.**—This name was given by Bazin to what will be afterwards described as *molluscum contagiosum*. It is unfortunate that Hardy accepted such a confusion of nomenclature and of pathology.

The name has, however, been since applied to a rare and curious affection which consists in large pustules somewhat resembling those of the more severe kinds of acne, and situated chiefly upon the forehead, the temples, and the sides of the cheeks. After they have burst and healed, a deep scar is left, sometimes pitted but not pigmented, resembling that which follows the most severe kinds of acne, and situated chiefly upon the forehead, the temples, and the sides of the cheeks. It is in these scars that the resemblance of the affection to variola chiefly consists, for the distribution, the course, the absence of a vesicular stage, and the unimpaired health of the patient could never allow of its confusion with variola. In a patient of the writer's, a man of about forty, the affection encroached upon the scalp, and also spread to a considerable part of the chest, shoulders, and back. In this, as in the other cases, this curious affection was quite distinct from true acne. It is not preceded by comedones, and it seems doubtful whether the pustules are really seated in the sebaceous glands. The distribution and the severity of the eruption also distinguish it from acne, and it is unconnected with the period of puberty. It is more difficult to distinguish it from a pustular syphilide, and some cases which have been described as acne varioliformis were probably syphilitic. In one patient, in whom there was neither history nor proof of venereal disease, the pustules, which had lasted for a long time, disappeared under iodide of potassium. Dr Liveing, however, has seen instances of acne frontalis which resisted antisiphilitic treatment, and yielded to large doses of arsenic. We must wait for further observations before the true nature of this affection can be decided.

It will be convenient to refer briefly in this place to other affections of the sebaceous glands, less important than acne.

Comedones are generally the first stage of that disease, and then occur in the persons and under the circumstances above described; but beside the accidental comedo, or even pustule, which may be produced here and there by obstruction of a duct in any part of the body, and which no more make acne than one swallow makes a summer—there are occasionally to be seen large numbers of comedones in children affecting the forehead, scalp, and other parts, not undergoing inflammation, and without the locality or other characters distinctive of acne.

* Neumann, following Hebra, calls it *acne frontalis*, and describes it as a variety of true acne. Dr Bulkley, of New York, names it *acne atrophica*, after Chausit's *acné atrophique*, or lupoid acne. See a case figured by Dr S. Mackenzie in the 'Clin. Trans.' for 1884, p. 227, and remarks by Dr Mackey in the 'Lancet' (Jan. 22nd, 1876).

Acné cornée is the name given by French writers to this remarkable and rare condition, which does not deserve the name of acne, first, because it is not inflammatory; and secondly, because it has not the natural history of the disease of that name. It consists in the presence of a multitude of *comedones*, which remain as passive papules, hard pointed and black tipped. They occur in children before the age of acne, and upon the scalp and other parts unaffected by acne. We have had several cases of this singular condition, which requires to be distinguished from "lichen pilaris." Once it occurred on the forehead and scalp of two brothers, once in a brother and sister between seven and nine years old, once on the temples of a boy of eight or nine suffering from pleurisy, and once on the lumbar region of a girl aged thirteen, under treatment for erythema multiforme, but without a trace of acne, or even comedones on the face, chest, or back (see 'Guy's Hospital Reports,' 3rd series, vol. xiii, p. 213). Some of the cases described under this name seem to come very near to *Keratosis pilaris*.

Milium.*—A commoner condition is passive obstruction of a sebaceous gland with complete occlusion of the orifice. A minute white or yellowish papule is thus formed without the pointed top or the black mark of a comedo. It has been called milium from its size. It never inflames, and is of no practical importance; it occurs most often on the thin skin of the eyelids or the genitals, but may be found on any part of the skin or trunk. Sometimes it grows larger than a pin's head; and when this occurs it usually affects only a single gland. Its contents then not unfrequently become liquid, and it forms a small cyst, such as may be occasionally seen on the eyelids, and have been noticed by Mr Hutchinson to occur in association with xanthelasma and with sick headaches. In young children it is not uncommon and corresponds to Willan's *Strophulus albidus*.

On dissection the acini of the gland are found filled with a dark refracting substance, which yields on analysis cholesterin, olein, palmitin, and stearin. A good drawing is given by Neumann (fig. 10).

Seborrhœa.—The functional disorders of the sebaceous glands may lead to too abundant liquid secretion, or to too solid scaly products, or by suppression to abnormal dryness and harshness of the surface. The first of these conditions has been named *seborrhœa oleosa* or *steatorrhœa*. It is physiological at a certain period of life, when it forms the *vernix caseosa* of newborn infants. It is not uncommon about the face, and especially on the *alæ* of the nose. It also occurs on the genitals, where a local *vernix caseosa* may lead to pruritus and inflammation: cleanliness and a little lead lotion or ointment is sufficient treatment.

Seborrhœa sicca—an "oxymoron" in terms—depends upon the more solid fats, stearin and palmitin, being secreted in greater abundance than the liquid olein. The secretion forms little yellowish scales, added to by the natural desquamation, and frequently by the local irritation of a slight dermatitis which increases the desquamation. The condition is most common on the scalp, where it constitutes what is known as *pityriasis capillitii*, dandriff, or scurf. In most cases this is rightly termed *seborrhœa sicca capitis*; but, although it begins as a sebaceous affection, in cases which have lasted long one finds that the scales consist in large part of

* *Syn.*—Grutum—Acne albidæ. So-called colloid-milium is a different disease, and will be described with other neoplasms hereafter.

epidermic cells, and there is often beside local irritation, injection, and other signs of dermatitis. That this is secondary is shown by its not spreading beyond the scalp, and by its being unconnected with eczema or psoriasis of the scalp. Beside the irritation of this common disorder, it undoubtedly leads to the hair becoming thin and weak, and in many cases produces early baldness (*Alopecia pityrodes*).

In cases of *Seborrhœa capitis* also micrococci are always to be found, and the best treatment consists in the use of parasitocides particularly. (See an excellent lecture by Dr Payne in the 'Clinical Journal' for Dec., 1893, pp. 105, 121.) Mild mercurial ointments or sulphur or carbolic oil are useful, also Borax in solution. Dr Payne recommends 15 gr. of sulphur, and 15 gr. of carbolic acid in an ounce of vaseline.

Seborrhœa corporis is the condition present in a peculiar and characteristic disorder, to which the name *Lichen circumscriptus** was given by Willan and Bateman. It is a papular dermatitis in circumscribed more or less annular patches, with a yellowish tint and a greasy surface. It occurs on the trunk, usually between the shoulders, but may spread over great part of the back, or may affect the chest or abdomen; and it may spread from the shoulders to the arms and from the abdomen to the thighs, always avoiding the flexor aspects. The papules are small, red, and arranged in patches with somewhat well-defined margins. It is not very irritable, and rarely, if ever, ends in ordinary moist eczema.

Sometimes it occupies the space between the shoulders and the sternum only, forming a figure like a compound-leaf (*seborrhœa petaloides*).

Many cases which have been described under this head are probably nothing but papular dermatitis, or secondary local eczema, depending upon the irritation of decomposing sweat. The locality between the scapulæ and on the front of the chest is just where sweat accumulates; the eruption is most common in summer, and in persons who sweat freely; moreover with the papules true vesicular sudamina may sometimes be detected. But apart from this, it must be admitted that there is a distinct circumscribed papular dermatitis, which, from the shape of its patches and from their spreading at the edge while the centre returns to its natural condition, reminds one of spots of tinea. Parasitic fungi are apt to occur in the locality and under the conditions named, as in tinea versicolor, but the affection under consideration is certainly distinct from tinea versicolor. It is allied to, if not identical with, dry seborrhœic eczema of the arms (p. 826).

Dr Payne has pointed out that it is usually associated with wearing thick woollen vests, often night as well as day; hence we call it a flannel rash.

The arææ of the circles present a yellowish tint (*Eczema flavum*), and are sometimes covered by branny desquamation. When several circles combine they form irregular lines, and the eruption thus formed was called *Lichen gyratus* by Biëtt and Cazenave. Two of Mr Towne's models in the Guy's Hospital Museum (Nos. 267, 268) show this appearance perfectly.

This affection may be frequently seen in young men or young women, never in children or persons over fifty. The patients are usually cleanly, and, in fact, appear almost as often in private practice as at the hospital. Since the eruption generally does not itch much, and is most marked where it is not seen, one often comes upon it accidentally when examining the

* *Synonyms*.—*Lichen annulatus* (Wilson)—*L. marginatus* (Liveing)—*L. circinatus*—*Lichen acnéique*—*Eczema flavum*—*Seborrhœa* of the trunk (Duhring)—*Seborrhœic eczema* (in part) of Unna—*Circinaria*.

chest. In every case the patient has been wearing a thick woollen jersey next the skin, and often the same has been worn at night.

Duhring and other American dermatologists regard Willan's Lichen circumscriptus as the result of irritation of the sebaceous glands, and call it Seborrhœa corporis; and seborrhœa capitis is present in most cases that the writer has seen. On this point Dr Payne remarks "(1) that this affection is certainly often associated with seborrhœa of the scalp; (2) that minute examination undoubtedly shows that the starting-point of each so-called papule is a sebaceous gland; (3) but that it is not accurately described as merely seborrhœa or excessive secretion. The bright red colour of the papules or margins of the patches, which strikes every observer, depends not only on hyperæmia, but on dilatation and elongation of the capillary vessels, which project above the skin level, as in psoriasis. Hence it is that slight scratching causes hæmorrhage. This is something more than over-secretion. A few cases, in which the eruption existed on the limbs as well as on the body, I believe to belong to a different disease, apparently identical with *Pityriasis rosea* of Gibert."

The aspect of *L. circumscriptus* is like that caused by a parasite; and Unna has described a diplococcus as characteristic.

After shifting the underclothing, free use of soap and water with liq. picis carbonis as a lotion (1 in 10), or an ointment (3ij—3j), is speedily effectual in curing the disorder.

Xerodermia.—Diminution or absence of sebaceous secretion leads to the skin being dry, harsh, and apt to crack. The sweat-glands may be active and abundant; but sweat is apt to exert an irritant effect upon the skin undefended by its natural oily secretion. This condition is usually congenital, and was rightly described under the name of "xerodermia" by Wilson as the slightest degree of ichthyosis. It will be again referred to under that head.

A similar state of skin is, however, not unfrequently observed in children who are thin and ill-nourished, and in patients of any age suffering from prolonged wasting diseases, especially phthisis. The diminution of subcutaneous fat is accompanied with diminished supply of oily material to the sebaceous glands, and the skin becomes dry, pale, rough, scaly, and dirty. This condition, which is of only symptomatic interest, has been described as *asteatosis* and as *pityriasis tabescentium*.

The only treatment indicated is to supply the deficient oily material by inunction with olive or cod-liver oil.

STEATOMA.—When the orifice of a sebaceous gland is obstructed and an accumulation of the secretion takes place, it does not always inflame; the secretion may go on until a large cystic tumour is formed.

The orifice of the duct can still usually be found, and sometimes by mere pressure the contents can still be evacuated. They consist of inspissated sebum without, on the one hand, the pus which mingles with the secretion in inflamed acne, and without, on the other hand, the remarkably modified epithelial cells which are characteristic of molluscum. When the watery parts are absorbed, the sebaceous secretion consists of the ordinary animal fats, palmitin, stearin, and olein, some butyric and caproic acids, either free or united with glycerine to form neutral fats, a small amount of albumen, or rather globulin, with a larger proportion of

casein, epidermic cells, the flat tabular crystals of cholesterin, and earthy salts. Occasionally the fatty material appears to be absorbed as well as the water, and there remain behind only calcareous masses like those found in a diseased aorta, or in the apex of the lung in a case of obsolete phthisis. These cutaneous calculi are, however, of very rare occurrence. The yellow, somewhat granular, half liquid, and half solid mass has been compared with porridge, with putty, and with mortar. Although the word *atheroma* (from *ἀθήρη*, oatmeal) is now generally applied to the very similar products of chronic inflammation of the arteries, yet "atheromatous tumour" is still used by some writers as synonymous with what is otherwise called *Steatoma meliceris*, or, perhaps, better, a sebaceous cyst.

These tumours, called, when they attain a large size, *wens*, most frequently occur upon the scalp, where they are often multiple, and may grow to the size of a fist, or even bigger. They may also be seen upon the eyebrows, face, and neck, less frequently on the trunk, and most rarely upon the limbs. They are quite distinct from *Molluscum contagiosum*, to which they have a certain likeness when they occur on the face.

Sometimes a sebaceous cyst is pedunculated instead of sessile, and has then been dignified by the name *acrochordon*.

Cysts with similar contents, but of very different origin, and, probably (some of them certainly), congenital, occur in mucous membranes and in deeper parts of the body, especially about the root of the tongue and hyoid bone; and such *cholesteatomata* have also been described in the brain and in the bones. Many of them are true dermoid cysts, and may be compared with those of the ovary, which contain not only sebaceous matter, but hairs and sebaceous glands. Others have their origin in the epithelium and glands of persistent parts of the primitive bronchial arches.

A steatoma is easily recognised by its smooth rounded surface, the presence of a dimpled occluded orifice, the absence of pain, and its situation.

Sebaceous cysts are perfectly innocent, but occasionally require removal from their inconvenience or unsightliness. The plan usually adopted is to incise the tumour and tear or dissect out the secreting cyst wall, or, if this be difficult, to rub the interior with caustic.

ADENOMA SEBACEUM.—Rayer figured this curious and rare form of new growth under the title "Vegetations vasculaires;" and a case was described by Addison and Gull under the name "Vitiligoidea" in the 'Guy's Hospital Reports for 1851 (first case, p. 267). It was admirably modelled by Mr Towne (Guy's Hosp. Museum, No. 262), and shows scattered pale, shining papules, with minute veins giving them a faint rose-red tint, the face of a young woman of twenty-four. Dr Fagge saw that it was not Xanthelasma, nor the papular affection associated with diabetes which was then included under the same name, and labelled it "Lichen" with a doubt whether it was not rather a form of acne. Several similar cases have since been recorded in England and abroad, and it is clear that the "papules" are really minute neoplasms arising in the sebaceous glands of the face, innocent, and glandular in structure. (See Dr Pringle's article, 'Brit. Journ. Derm.,' 1890, with plate.)

There are five models of this rare malady at St Louis, and it was first correctly described by Dr Balzor as *Adénomes sebacés* (1885). It is probably congenital, but apt to become much more marked about puberty. It is said to be less rare in women than in men, and in persons of feeble intel-

lect than in others. It is almost confined to the face, where it may simulate gutta rosea.

In elderly patients an adenoma of a sebaceous gland has sometimes developed into a malignant epithelioma.

SYCOSIS.*—Closely allied to acne is a disease which consists in pustular inflammation of the large hair-sacs of the beard. Anatomically it is difficult to draw a broad line between them, for although we speak of acne as inflammation of the sebaceous glands, yet since all these glands open into a hair-sac, obstruction of their duct and obstruction of the corresponding hair-sac are almost about the same. The anatomical difference lies in this, that whereas on the general surface of the body the hairs are small with shallow sacs, and the sebaceous glands large, in a man's beard the hair-sacs are large and deep, and the glands comparatively small. Moreover, there is no doubt that in acne it is the gland which is first obstructed so as to form a comedo and which afterwards inflames, while in sycosis it is the hair-sac which is the primary seat of disturbance.

It was Bateman who first accurately defined the characters of sycosis in the modern sense of the word. He placed it under Willan's order *Tubercula* on account of the hard swelling which often surrounds the pustules.

Anatomy.—Hebra expressed the opinion, since supported by Liveing and other observers, that the immediate cause of a sycosis pustule is the presence of two or more hairs growing together in the same follicle. Wertheim, in 1861, read a paper before the K. k. Ges. der Aerzte of Vienna, in which he referred the origin of sycosis not to the growth of more than a single hair in the follicle, but to its being abnormally thick.

Whatever the determining cause, streptococci find their way into the acinus of a hair-sac, and the hair bulb becomes loosened, but the shaft still blocks the sac. The drop of pus first formed is pent up and produces pain and fresh inflammation. By the time that the hair is at last detached, a small but deep cutaneous abscess has formed, and considerable congestion and œdema around it has produced what Willan called an inflammatory tumour or nodule. When the pus at last finds exit, it dries into a scab; and this is rendered much more adherent than that of impetigo by the numerous hairs which tether it to the skin. Fresh accumulations of pus take place beneath it, and thus in severe cases of sycosis a most repulsive and "malignant" aspect may be produced.

Excluding parasitic sycosis, which is of course contagious, Hebra and most German writers maintain that inflammation of the hair-sacs of the beard is non-contagious; but this seems to be very doubtful. As we now know, pus is itself an extremely contagious product from the staphylococci or other pyogenic organisms it contains. We see that it is so in cases of contagious impetigo and of furunculi, and probably all cases of sycosis are

* *Synonyms.*—*Mentagra*—*Varus mentagra*—*Acne menti vel barbæ*—Barber's itch.—*Fr.* *Impétigo sycosiforme* (Devergie), *Adénotrichie* (Hardy).—*Germ.* *Bartfinnen*.

The name *sycosis* was applied to this affection by the Greeks, from its supposed likeness in the worst cases to the inside of a ripe fig, the red pulp answering to the inflamed and swollen skin, the seeds to the little pustules. "*Est etiam ulcus quod a fici similitudine σῦκωσις, a Græcis nominatur*" ('Celsus,' lib. vi, cap. 3).

The terms *mentagra* and *lichen menti* of Pliny and Martial, like the sycosis of the Greeks, were often applied to syphilitic affections of the lips and other parts, probably to what we now call mucous patches and condylomata; but the Latin *ficus*, like the Greek *σῦκον*, was certainly applied not only to condyloma ani, but also to hæmorrhoids.

more or less capable of spreading by inoculation in the same person, or even under favourable conditions, as in a barber's shop, to another person.

Not only ordinary ringworm of the beard, but pustular sycosis can in many cases be proved due originally to infection with *Trichophyton tonsurans*, though pyogenic cocci are present also.

Course.—If left to itself sycosis is a most obstinate disease. The hair-sacs are successively destroyed, and cicatrices result which are sometimes deep and obvious. When at last the disease has worn itself out, the greater part of the beard is often permanently destroyed and the face disfigured by scars. The affected part is usually very tender while the process goes on, though, except when touched, there is rather tension and heat than severe local pain.

Distribution.—As explained above, the peculiar kind of inflammation described can only occur in large and deep hair-sacs like those of the beard. The disease usually begins upon the chin, frequently on the upper lip, on the cheeks, or under the jaw. It may, however, occasionally be observed in the eyebrows and on the pubis, and still more rarely similar pustules have been seen on the chest, thighs, and other hairy parts.

True sycosis is confined, if not absolutely, with the rarest exceptions, to the chin, lips, and cheeks of male adults. It is rare to see it in a soft beard which has never been shaved.

Diagnosis.—Sycosis must be distinguished from eczema of the face, which sometimes invades the cheeks and lips. It is possible for the dermatitis thus produced to penetrate to the deep hair-sacs, and then a condition ensues which must be termed true secondary sycosis. But this is certainly very seldom the case; the superficial dermatitis as a rule preserves its superficial character, the pustules and crusts are those of impetigo, and when removed leave the surface but little affected; in these cases treatment by ung. hydr. ammon. is rapidly successful; no scars are left behind, and no hairs are destroyed.

Treatment.—In cases of true sycosis which affects the hair-sacs, mercurial ointments must be combined with epilation. It is not, however, necessary, in most cases to remove all the diseased hairs, and certainly not healthy ones. It is enough if those which are already loosened are extracted, so that the rule is for the patient to pluck out all hairs which will come easily and without pain, that is, those which are already detached from their sacs. The first step in severe cases is to steam the face, and, if necessary, to soften the crusts with poulticing and sweet oil; then to remove the loose hairs with broad-pointed forceps. The beard should be cut short, but not shaved. If there is much local pain and swelling, the inflammation should first be subdued by lead lotions, lead and zinc ointments, or other soothing and astringent applications. When this is accomplished the treatment above advised should be begun and followed out day by day. In most cases the result is successful. When cure has resulted it is generally better for the patient not to shave for several months, but to allow the beard to grow naturally.

Not infrequently, however, sycosis proves very obstinate in spite of all care and diligence. The presence of a parasitic cause should in such cases be carefully looked for. In the most obstinate cases complete epilation on Plumbe's and Hebra's plan is no doubt the only effectual treatment, but it should be carried out piecemeal, and with the help of previous application of potash soap, and other remedies which soften and loosen the hair.

During epilation dilute red oxide, or better, perhaps, the yellow oxide of mercury ointment, should be rubbed into the surface.

Parasitic sycosis.—Since Gruby, in 1847 ('Gazette Médicale,' No. 37), published an account of a cryptogamic plant which he discovered in cases of sycosis, French writers have generally described sycosis as a parasitic affection. Bazin named it Teigne Mentagre. Gruby's name for the fungus was *Microsporon metagrophytes*, but Bazin and Rubet proved that it is identical with *Trichophyton tonsurans*, the name given to that of common ringworm in 1846 by the Swedish writer Malmsten. So also Köbner in 1864. Hardy followed Bazin, and diverged from Cazenave and from Bielt, who had placed sycosis among pustular dermatoses, for he practically maintained that all sycosis is parasitic. In his recent work, however, while regarding "true sycosis" as a teigne sycosique, he admits non-parasitic sycosis as an impetiginous inflammation of the hair-sacs.

Hebra, after reviewing the statements of previous authors, affirms that in more than 300 cases of sycosis or "follicular inflammation of the beard," he has never seen a single case accompanied with parasitic fungi.

There can, however, be no doubt of the existence of what the French writers call parasitic sycosis. The writer saw many cases at St Louis, and others, though much more rarely, in London. He never saw a case in Vienna,* and those who have studied dermatology in several schools will probably agree that this is one of the instances in which we must admit local differences in the frequency of diseases. Parasitic sycosis certainly does occur, and will be found if looked for in London, but it is far less common here than in Paris; and it must be added that the presence of the fungus is often only to be ascertained after prolonged and repeated search. When detected, however, the case acquires at once a new character, and for practical as well as scientific reasons it is desirable to separate "parasitic sycosis" from the non-parasitic disease.

Sycosis capillitii.†—This title of Willan's should perhaps be given to five remarkable cases of sycosis or pustular eczema of the hair-sacs observed by Hebra on the occiput and nape of the neck.

This very rare condition was named by Kaposi *dermatitis papillomatosa capillitii*. Hans von Hebra ('Archiv für Derm. und Syph.,' 1869, p. 382) prefers the name suggested by his father, of *Sycosis frambæiformis*. He describes it as beginning in very small, somewhat red papules, each traversed by a hair, which grow together and form hard tumours resembling raspberries, and at last end in a long, tough, cheloid-looking band. The disease occurs on the nape of the neck where the hair is growing; the skin around is eczematous and red, and the place painful. The course is very slow, and as new papules arise they fill with thin pus. When raised flat papules have been formed, hairs are seen pushing out in bundles. This, with the hardness of the growth, its extreme slowness of development, and its locality, are the characteristic points. It may, however, occasionally

* At present (1891) paralytic sycosis is said not to be very rare in Vienna, and usually of the *Megalosporon* variety.

† *Synonyms*.—*Sycosis frambæiformis* (Hebra)—*Dermatitis papillomatosa capillitii* (Kaposi)—*Acne keloid*. The sycosis capillitii of Willan, p. 66 of his 'Atlas,' is not unlike the curious affection described on p. 845 as *Acne varioliformis*. The *Pian ruboide* of Alibert (pl. 35) may have been the same disease, unless we suppose with Bateman that these were mismanaged porrigo favosa, or with Hebra that they were undiagnosed syphilis.

occur on the scalp. This same affection has been described by Dr Sangster as "a papillary tumour of the scalp" (Internat. Med. Congr., 1881), by Dr Vérité as *Acné keloïdique* (Académie de Médecine, 9 Mai, 1882), and by Mr Marrant Baker, with a figure, under the same title ('Pathological Transactions,' 1882).

The treatment of this remarkable disease consists in destruction with caustics or removal of the tumours while still small with a sharp spoon, by galvano-caustic, or by other means. When the disease has already gone far excision is the only remedy.

Histological sections show that there is true enlargement of the papillæ, very scanty exudation of leucocytes, and gradual formation of parallel and interlacing bundles of fibrous tissue, among which the sebaceous and sweat-glands are squeezed and atrophied.

FURUNCULI—Boils.—Recognising the complete impossibility of any complete and satisfactory classification of skin affections which can set forth all their complicated mutual relations, it seems convenient to associate the troublesome and painful affection of boils with acne and sycosis; for although we cannot prove that the seat of suppuration is always in a hair-sac, yet in many cases this is certainly true, and it is probably so in all. The depth of the inflammation, its pustular character, and the scars which it leaves behind, are points in which it is closely related to the affections described in this chapter, and particularly to the deep and painful suppuration which affects the glands of the vibrissæ of the nostrils and the ears.

The characteristic pathological feature of furunculus is that the inflammation leads to the death of a minute portion of the deeper layer of the cutis. This slough or core of necrosed connective tissue is passed out by a process of liquefaction and suppuration, and the abscess which is formed then slowly heals. In its early stage the disease appears as a pimple, distinguished by its excessive pain, a pain which resembles that felt from the plucking out of a hair, and no doubt depends on special sensibility of a hair-sac, as distinguished from the cutis generally and the sebaceous glands. The papule speedily shows a yellow spot at its pointed summit, and this little pustule is never preceded by even a transient vesicle. Meantime, a bright, intensely injected halo appears around the pustule, and considerable inflammatory oedema swells the whole skin into a conical elevation; the pain increases and becomes throbbing in character, while the dull constant aching and sense of tension is varied from time to time by sharp stabbing pains. When the abscess has ripened and is lanced, or bursts of itself, the "core" becomes visible, and is sometimes not expelled for a day or even longer. This stage is accompanied by a characteristic sharp pricking pain, which rapidly subsides when the core is got rid of; a small scab forms, the redness and oedema disappear, and soon nothing but a minute scar remains.

Unfortunately, however, it is seldom that this process is confined to a single furuncle. Most often a second and a third appear before the first is completely healed, or a whole crop may spring up almost simultaneously. A succession of painful abscesses may thus be established and last for weeks or even months, until the patient's health seriously suffers from the pain and the discharge. Sleeplessness, loss of appetite, and much depression, both physical and mental, may be the result. When a crop of boils thus appears, they are generally found to vary in size and severity, from those

which are so large and deep as to challenge the name of carbuncle, to small superficial pustules which formerly would have been called ecthyma.

Distribution.—There is scarcely any part of the surface which may not be the seat of a furunculus, but the affection has nevertheless a decided predilection for the back of the trunk, from the hair at the nape of the neck to the fold of the nates. The thick cutis, thin epidermis, and small but numerous hairs of the dorsal region appear to furnish the most favourable conditions for this kind of inflammation. The back of the neck, especially at the edge of the scalp, is perhaps the most frequent seat of all; the buttocks come next to the nape of the neck in liability to boils. Moreover, the friction of the collar of the dress in the latter situation, and that occasioned by riding or by rowing in the former, aggravate the misery of the complaint and probably keep it up.

Boils are far from unfrequent in the coarse skin of the outer part of the thighs, which resembles that of the dorsal and scapular regions anatomically. They may also appear, though less frequently, on the leg below the knee, on the upper and forearm, on the wrist, and on the back of the hand. The chest and abdomen, and even the face, are not exempt, but boils very seldom occur on the scalp, and never on the palms, or the soles of the feet. They are rare on the male genital organs, but not unfrequently occur in the neighbourhood of the anus, in the perinæum, and on the vulva.

Ætiology.—There is no ground for supposing that boils are the result of indigestion, or of overwork, or of exhaustion from any cause; when numerous and long continued they produce, but are not the product of, anæmia. Nor, on the other hand, do they come from plethora and over-richness any more than from "poverty of the blood." In the practice of the water-cure it is customary to wrap patients who suffer from dyspepsia, paralysis, and most other chronic diseases in wet sheets, which are then surrounded with blankets; free perspiration is thus produced, and in many cases it is offensive in odour and accompanied by a copious crop of boils and pustules. This is supposed to indicate the efficiency of the plan in bringing out the poison from the system, but in reality it only means stimulation of the sudoriparous glands, possibly vicarious excretion of urinary or fæcal products, and certainly traumatic inflammation of the skin.

The true cause of furunculi is the access of pyogenic microbes to the deep hair-sacs, and their appearing in crops is due to the contagion of their pus.

Age.—Boils are far the most frequent during youth. They are rare in infants, and not common in early childhood, but schoolboys are very liable to them, especially to the most characteristic form of successive crops on the neck and shoulders or on the nates. After thirty this painful affection becomes decidedly rare, and we seldom find boils in an elderly man. Women during the whole of life seem less liable than men, though some of the most severe cases occur in young women.

Furuncles are contagious from one patient to another; and almost an epidemic may sometimes run through a school; and still more frequently they spread from place to place on the same patient.

Treatment.—The fact of their pathology furnishes an important indication for treatment. While the first furuncle is developing, no doubt a poultice gives great relief from its warmth and the relaxation of tissue it produces, but by making the skin sodden and softening the epidermis it renders the access of the pyogenic microbes more easy. Hence the constant

application of poultices over large surfaces affected with boils very much tends to spread and continue the disease. A much better plan is to dispense as much as possible with poultices, using water dressing instead, and to apply to the skin immediately around the boil Goulard wash, or a somewhat stronger lead lotion. Tannic acid may be used with the same object; or, as each boil appears, a circle may be drawn around it with tincture of iodine or dilute solution of silver. Sometimes collodion, especially the flexible collodion, applied in the same way, seems to act best both as an astringent and a protective. Meantime the pustules should be covered with lint soaked in carbolic oil (one in ten) and the same antiseptic dressing should be continued after the pustule has burst. It was formerly the practice to open each boil successively with the lancet; but though the process is undoubtedly thus hastened, the pain and the dread of the pain are so severely felt, especially in young people, and when a sensitive part of the body is affected, that at least in such cases the furunculi may be left to ripen and burst of themselves. A recently introduced method is to inject the boil with a needle containing solution of corrosive sublimate. This also is not free from pain; but is less formidable, and if skilfully performed, effectual.

There is no evidence of special advantage in the purges and alteratives which are the traditional treatment for boils. Where only one or two exist, no internal treatment is necessary; but where the crops are numerous and successive, the treatment by local astringents and antiseptics should be combined with the internal administration of wine or porter with the meals, and of either *nux vomica* with mineral acids or tincture of steel in full doses. Small doses of calcic sulphide are frequently prescribed for boils as well as for other chronic suppurative affections, and apparently with benefit. Thin, delicate boys will often be much benefited by cod-liver oil, either alone or, if anæmia indicates it, in combination with steel. There is reason to believe that a stay at the seaside is particularly useful during convalescence.

CARBUNCLE.—This term, as the diminutive of *carbo*, is the Latin translation of *ἀνθραξ*, a coal, and was applied to any red, angry, inflamed pustule. The word anthrax has in recent times been restricted to the disease known as splenic fever, accompanied with a characteristic boil or carbuncle of the skin derived by contagion from cattle and associated with the presence of a specific bacillus, which has been fully described in the first volume (p. 415).

A carbuncle is pathologically identical with a boil, differing only in its severity and extent, but its natural history is sufficiently different to justify the old distinction being retained.

Anatomically, a carbuncle is the inflammation which accompanies a large and deep cutaneous and subcutaneous slough. It differs from the larger and deeper boils only by the affected tissue being so extensive that not a single opening forms, but several, giving a characteristic perforated aspect to the broad summit of the tumour. If left to itself this gradually opens by ulceration, and a deep and wide aperture is formed, through which the slough is at length extruded, often with considerable hæmorrhage. The surrounding redness is commonly deeper and more lurid in hue than that of a boil; the œdema also is more extensive.

A carbuncle almost always occurs singly. Its most frequent seat is the nape of the neck and the shoulders; it may occur on any part of the trunk, but it is rarely seen on the limbs or the buttocks. Occasionally it appears

on the face, and is then severe and often dangerous. Carbuncles are more common in the old than in the young, and frequently occur in diabetes (vol. i, p. 446). The modern treatment of early and complete excision has greatly diminished the danger of the disease.

Darier's disease.—Two cases of this rare condition—independently described by Dr White, of Boston, in 1889, as *Keratosis follicularis*, and since identified with an earlier case called *Acne sebacea cornea* by Erasmus Wilson—were described in Paris by Darier in 1889 ('*Annales de Derm. et de Syph.*,' vol. x) under the title *Psorosperme folliculaire végétante*. The lesion appears to begin in the hair-sacs and sebaceous glands, particularly on the face, back, axillæ, and groins. It consists of a horny plug occupying the orifice of the hair-sac, and leaving, when removed, a funnel-shaped pit in a reddish papule. Sebaceous matter can be squeezed out of the little tumour, sometimes mingled with pus. As the disease spreads, the elementary papules coalesce, the horny plugs increase (to a length of three or four centimetres in one case), and gradually a mass of raw granulation tissue is formed, which exudes foetid secretion. This stage is more marked in the inguinal and anal regions, or in the axillæ. A constant histological condition appears to be the presence in the epidermic cells of the sebaceous duct of corpuscles which resemble psorosperms or coccidia (p. 476) in appearance and reactions to stains. They are no doubt the same as the bodies described in Paget's disease and in carcinoma, and also in molluscum contagiosum. Darier's view of these structures is supported by Brocq, by Ruffer, and by Plimmer; but Boeck and Unna, with the majority of pathologists, are unconvinced.

The disease has repeatedly begun in children, and sometimes in two of the same family. Its progress is slow but ingravescens, and treatment has at present been little more than palliative (see Dr Pringle's article in '*Allbutt's System*,' vol. viii, p. 879).

AFFECTIONS OF THE SWEAT-GLANDS.—The sudoriparous or coil-glands are less liable to disease than the sebaceous, and their affections are less important from a local, though more so from a symptomatic, point of view.*

Anidrosis, or deficiency of sweat, is seen as a concomitant of many forms of pyrexia, and usually accompanies erythematous and roseolous eruptions. In lichen planus, psoriasis, and pityriasis rubra, little or no sweat is secreted, and probably the same is true of eczema madidans. Ichthyosis, including "xerodermia" (dry skin), is also marked by absence of sweat. That the function of this secretion is only supplementary to that of the kidneys and lungs as an excretion of water, and that its chief purpose is not excretory but regulative of temperature, is shown by the fact that patients with universal ichthyosis or pityriasis rubra show no symptoms of blood-poisoning from retained excreta.

Hyperidrosis.—General or profuse secretion of sweat takes place under two conditions. First, along with hyperæmia; this occurs in health during

* It appears to the writer that Unna has made good his contention that the coiled glands secrete oily products as well as water. In fact, he candidly admits that this was the statement of Krause and Köliker, Henle, Ludwig, and Ranvier ('*International Congress of 1881, Section of Dermatology*,' vol. iii, p. 187; and also '*Brit. Journ. of Derm.*,' 1894, p. 265). There is, moreover, a close relation between the coiled acini of the sweat-glands and the "fat-column" which, as shown by Dr Warren of Boston, communicates with the subjacent adipose tissue.

the natural sweating and warmth of skin induced by active exercise, and pathologically in rheumatism and the sweating stage of ague. Secondly, profuse cold perspirations take place with an anæmic state of the skin, as in the cold perspirations of terror, the night sweats of phthisis, and the cold perspiration which sometimes marks the approach of death. Modern physiology teaches that the vascular supply and the epithelial activity of sweat-glands, as of other secreting organs, are governed by distinct nerves.

Local hyperidrosis, when it affects the hands and feet, is sometimes the source of considerable annoyance. Astringents are often useful, particularly tannin and alum. In a troublesome case, profuse perspiration of the palms of the hands in a young lady was cured, after other treatment had failed, by the local application of belladonna. Internally the same drug is indicated by our knowledge of the physiological action of atropine upon the submaxillary gland.

Bromidrosis and *osmidrosis* are names given to foetid perspiration, which is usually also excessive. This most frequently affects the feet, and may become a source of the utmost discomfort. The persons it affects are almost always young adults, and women more frequently than men. A horrible stench results from decomposition of the fatty matter which mingles with the sweat, particularly the fatty acids which belong to the formic acid series—butyric, caproic, and caprylic. The chief seat of evil-smelling sweat beside the feet is the axilla. Dr Thin figured a bacterium to which this decomposition may be due ('Proc. Royal Soc.,' 1880). This is, however, probably identical with *B. epidermidis capsulatus*, which is almost constantly found in this part of the skin.

The treatment of this distressing affection is often extremely difficult. The first step is to check the secretion by astringents, and to prevent its soaking into the clothing by absorbent powders, such as lycopodium; the next is, by frequent change of linen, to remove the products of excretion as rapidly as possible. Antiseptics like thymol and salicylic acid may be usefully applied, and the latter preparation, especially in the form of colloidion or a salicylic plaster, has the further advantage of softening the accumulation of macerated cuticle. With the same object Hebra used to envelop the foot and toes in strips of diachylon plaster, and many can testify to the efficiency of this treatment, the details of which will be found in the English edition of his work (vol. i., p. 89). Dr Thin recommends a saturated solution of boracic acid ('Brit. Med. Journ.,' Sept. 18th, 1880); and a 5 per cent. solution of chromic acid painted on the feet has been found useful in Germany.

Chromidrosis is the name given to the occasional secretion of coloured sweat. The sweat of the axillæ in some persons contains enough pigment to stain their linen of a reddish tint. The writer has met with one well-marked case, and Hoffman has recorded another in the 'Wiener med. Wochenschrift' for 1873, No. 13. Sometimes, however, a bluish pigment stains the sweat on the face or elsewhere. Cases of supposed chromidrosis occurring in young women should be watched. In most cases the apparently dark sweat is an *arte factum*. But although most supposed cases have proved to be factitious, there is no doubt that true chromidrosis does occasionally occur; and in some cases it has been proved to depend on indican being excreted in the sweat, and turning to blue indigo when oxidised by exposure to the air. Dr Foot published a case in the 'Dublin Quarterly Journal' for August, 1869, and collected no less than thirty-seven

others. Another source of colour is the production of blue or greenish fungi in decomposing sweat. When coloured sweat affects the eyebrows it is usually of black colour, looking almost like soot. A remarkable case of red-coloured sweat was reported by Dr Wilks in the 'Guy's Hospital Reports' for 1872. In this case a chemical analysis by Dr Thomas Stevenson proved the presence of iron but the absence of hæmoglobin.

Uridrosis, or the excretion of urea in the sweat, probably only occurs as a morbid phenomenon. The observations of Funke on the normal excretion of urea through the skin have not been confirmed, but in Bright's disease urea has been visibly discharged in the sweat (p. 474).

Hæmatidrosis (or *hæmidrosis*), or bloody sweat, is an extremely rare but undoubted morbid condition. It does not appear to accompany purpura or other diseases in which one would anticipate such hæmorrhage from changes in the blood or capillaries; and in some of the very few authentic cases it appeared during apparent health, as in that of a friend of Hebra, who observed the exudation of blood-stained sweat upon his hand while sitting at table. A similar condition has been observed in the lower animals.

Dysidrosis was the name given by the late Dr Tilbury Fox to a curious affection of the skin of the hands ('Path. Trans.,' 1878), described as *chiro-pompholyx* by Mr Hutchinson ('Lancet,' 1876). It consists in large vesicles without any surrounding inflammation, occurring in groups upon the palm and back of the hand and the fingers, especially near the web. These vesicles have been compared to sago grains, though they sometimes reach a much larger size. Rasori, who published a case in the 'Transactions' of the International Medical Congress for 1881 (vol. iii, p. 146), calls it *hydro-adenitis diffusa*. Hans v. Hebra records a case in his 'Kr. Veränderungen der Haut,' p. 426. It affected the palms and soles of a woman forty-two years old, and some of the larger bullæ were surrounded with a red halo. Mr Hutchinson has observed relapses of this singular affection on several occasions. Whether it depends, as Dr Fox supposed, on obstruction of the duct and accumulation of its contents is doubtful. Possibly bullous erythema and true retention-blebs of sweat have been confounded.

Sudamina.—However this may be, there is a well-marked cutaneous affection which undoubtedly depends upon accumulation of sweat in little vesicles under the cuticle, and has been known for centuries as *ἰδρωμα*, *sudamina*, or *miliaria*. It is often seen during the profuse sweating of rheumatism. The orifice of the duct becomes obstructed, and the horny cuticle is raised as a thin transparent layer enclosing a drop of transparent fluid (*miliaria crystallina*). This ruptures before it exceeds the size of a pin's head, but sometimes the contents become turbid and alkaline from slight inflammation. On the chest and back these sudamina are most common; they never occur on the face, and on the thick skin of the palm attain larger dimensions, so as to form the "sago-grain" vesicles of dysidrosis.

The profuse sweat which causes sudamina also produces local irritation. This seldom goes beyond the stage of papules or erythematous redness except where it is aggravated by friction. This *dermatitis a sudore* affects the vertebral groove from between the shoulders to the sacrum, and the front of the chest. The more severe results in the axillæ, between the toes, and between the nates, are known as *intertrigo*.

RINGWORM AND ITS ALLIES, WITH OTHER AFFECTIONS OF THE HAIR AND NAILS

“Under thy long lockes thou maist have the skull.”

CHAUCER.

- RINGWORM—(1) *Tinea tonsurans*—The fungus—Anatomy—Course—Events—Histology—Detection of the fungus—Ætiology—Prognosis—Treatment—Parasitocides—Irritants—Mode of application—Epilation—Precautions against contagion—(2) *Tinea circinata*—Form and locality—Burmese ringworm—*Tinea marginata*—Treatment of ringworm of the body—(3) *Onychomycosis*—Rarity and obstinacy.
- FAVUS—History—Anatomy—The fungus—Treatment.
- TINEA VERSICOLOR—Names—Parasitic nature—Appearance—Distribution—Diagnosis—Treatment—*Tinea vel Pityriasis rosea*.
- ALOPECIA—(1) Physiological—(2) Febrile and syphilitic—(3) Area—its appearance—locality—spread—question of its parasitic nature—prognosis—diagnosis—(4) Universal alopecia—(5) Congenital alopecia.
- TRICHOCLASIA, or brittleness of the hair—Piedra—Beaded hairs.
- AFFECTIONS OF THE NAILS.

WE have seen in other parts of this work that the human body is liable, beside animal parasites, to the invasion of the lower forms of vegetable life. The Schizomycetes, often spoken of generally as *Bacteria*, are the most important of these, since they form the contagion of some, possibly of all, specific fevers. They are described at p. 14 of the first volume, and are the cause of purulent dermatitis and also of some of the dry forms of eczema described above (pp. 847, 830).

Of less practical importance are the *Fungi* which are parasitic on the human body. Some of these affect the mucous membranes and have been already described in the present volume, as *Oidium albicans* in the mouth, p. 298, and *sarcina* in the stomach, p. 361. We have now to consider diseases of the skin which depend upon the growth of similar microscopic fungi. In most cases the cryptogamic spores and mycelium lodge in the deep hair-sacs of the skin.

We will first take ringworm, the most important of this group, then the remaining parasitic affections, and, lastly, it will be convenient to deal in this chapter with non-parasitic or doubtful affections of the hair, which need to be distinguished from the ringworm.

RINGWORM OF THE SCALP.*—This troublesome disease was only proved to depend on the presence of a cryptogamic parasite in 1844 by Malmsten, the Swedish microscopist. He named the fungus *Trichophyton tonsurans*. It belongs to the Ascomycetes ('Brit. Journ. Derm.,' 1889), and is referred by Sabouraud ('Annales de Derm. et de Syph.,' 1892) to the genus *Botrytis*. He states that the smaller spores without mycelium are most common in the scalp and most obstinate, and that the large spores with mycelium are more common on the beard and on the trunk.

Origin and spread.—We seldom see the earliest stage of the disease, but the first obvious effect of the entrance and growth of the fungus in the hair-sacs is for the affected hairs to lose their glossiness and colour and become dry, shrunken, and brittle. They break short and thus set free fresh spores to spread the contagion. At the same time the growth of mycelium in the hair-sac produces slight irritation, which is increased by the patient's scratching. Moderate hyperæmia and corpuscular exudation follow, the loosened hairs fall out, and so a small bare patch is formed, raised, slightly red, and scaly. The process extends, partly by the spores being conveyed to fresh places, partly by their steady advance to the next adjacent hair-sacs. Thus, one, two, and often numerous round patches are developed, each of which closely resembles the other. The form is often geometrically circular, sometimes oval or irregular; the hair is replaced by a few broken, dark, and thick stumps, which can be recognised by the naked eye, while their characters are still more obvious under a lens; the surface is usually covered with greyish-yellow desquamation, composed of epithelial cells and sebaceous material mixed with broken hairs, spores, and mycelium. The scales are closely adherent, and have a uniform, granular look, which is almost decisive to a practised eye. At the edge of the circle a little redness may sometimes be observed, occasionally a few papules, and, still more rarely, a vesicle or two. In the immediate neighbourhood individual hairs may be found by the aid of the microscope to be already affected by the spreading evil.†

A ringworm patch may increase to several inches in diameter without materially altering its appearance, but more often it is modified as it expands in one of the following ways.

Either from scratching or from the effect of the fungus on the naturally irritable scalp, or as the result of irritant applications, more or less of ordinary superficial dermatitis appears; so that many cases of ringworm appear as impetigo capitis, and their true nature is not manifest until the scabs and crusts have been removed. In neglected cases, moreover, pediculi are not unlikely to breed, and further aggravate and confuse the condition. Such horrible masses of felted hair, mingled with inflammatory products, vegetable and animal parasites, and all kinds of filth, constitute the *plica polonica* of Eastern Europe, which was still sometimes seen at Vienna in 1864.

On the other hand, if the hair is kept short and the head clean, and if the skin is not naturally irritable, the fungus, while spreading at the edges

* *Synonyms.*—*Tinea tonsurans*, the *Porrigio scutulata* of Willan—*Porrigio decalvans* of Gruby.—*Fr.* Teigne tondante.—*Germ.* Herpes tonsurans, Kopfgrind.—*Angl.* The Skall (in part), the Ringworm.

† They may be more easily detected by the naked eye if, as Sir Dyce Duckworth suggested, chloroform be first applied ('Brit. Med. Journ.,' November, 1873). This gives the affected hairs a dry, pale, brittle look, like that of burnt-up hay, apparently owing to its solvent power on the oily constituents of the hair. But this reaction is not decisive alone.

of the patch, appears to exhaust the soil in the centre, and dies away, like the larger cryptogamic fungi which form "fairy-rings" upon the grass. The result is that the middle of the patch is more or less completely bald, with only a few short stumps or thin, feebly-growing hairs, while the circumference is occupied by a zone of flat brownish scales, granular desquamation, papules, and broken hairs. This is the most typical form of traditional ringworm, and probably suggested the specific title *scutulata*. When growing patches of the disease meet, they form figures of 8 or dumb-bell-shaped patches, and, as they still grow and unite with others, irregular gyrate figures, like those of old-standing psoriasis, of erythema marginatum, or of syphilodermia. At last almost the whole scalp may be invaded and reduced to baldness. There is seldom a perfectly smooth clear skin left, as in alopecia areata; a few ill-developed, thin, pale, scattered hairs are almost always to be found. Moreover, the process is seldom or never quite universal; on one or the other temple or on the occiput more or less unaffected portions of hair remain.

Events.—The disease does not spread continuously beyond the scalp, but fresh patches arise, sometimes in the eyebrows, occasionally in the beard, more frequently on the skin of the neck and shoulders, and even on more distant parts. If left to itself the course of the disease is extremely chronic, and shows little or no tendency to recovery—if the patient is a child—until the period of puberty is reached. It must not, however, be supposed that among neglected children in a village or a school, where ringworm has invaded the community and scarcely a child has escaped, the disease constantly assumes the severe and inveterate character above described. A single bald patch may remain for months or years, or it may more or less completely recover, and fresh patches go through the same series of changes; or, what is still more important to notice, the spores falling upon an unfavourable soil continue to multiply, and are thus a fresh source of contagion, but yet do not sufficiently interfere with the nutrition of the hair to produce obvious bald patches. In a family or school in which ringworm has appeared, one may find evidence of its presence in the heads of children who are entirely without the characteristic bald patches.

The fungus.—At one time it was believed (by Hebra and Neumann and by Liveing) that the ringworm parasite was identical with *Penicillium glaucum*, but this has now been proved not to be the case (Thin, 'Proc. Royal Soc.,' 1881). Moreover it is now admitted that the *Trichophyton tonsurans* of Malmsten is specifically distinct from the *Microsporon furfur*, the *M. minutissimum* and the *Achorion Schönleini*, to be afterwards described, and specially identical with the *Microsporon Audouini* of Gruby. All belong to the botanical group *Hyphomyces*.

The division proposed by Sabouraud into a small-celled species (or variety) answering to Gruby's form (*Microsporon Audouini* or *Trichophyton microsporon*), and a large-celled one which he names *Trichophyton megalosporon*, has been generally accepted. A further statement of the French observer is that the large conidia or spores are found to be present when the invasion of the hair begins at the root, and travels upwards into the shaft (*Megalosporon endothrix*), and that the small-spored species is first observed at the mouth of the hair-sac, and thence travels downwards into the root. This is much more doubtful, as is the assertion that the large-spored form begins in the epidermis, and spreads to the hair, while the small-spored form begins in the hair itself. The reader is referred to the *Tricho-*

phyties humaines, with atlas, of Sabouraud, published in 1894, to the criticisms of Drs Colcott Fox and Blaxall in the 'British Journal of Dermatology' (July, Aug., Sept., 1896), and to the monographs on Ringworm by Dr Aldersmith (1897), and by Mr Malcolm Morris (1898). It appears that, at least in England, the microsporon is by far the most frequent in ringworm affecting the scalp in children, and the megalosporon in ringworm of the body, the beard, and the nails.

Diagnosis.—If one of the broken stumps of a ringworm patch be extracted with forceps, and placed in a drop of liquor potassæ under a quarter-inch objective, it may often be at once recognised by its opacity. When less densely packed with spores, or when soaking in potash has cleared it, the condition is equally manifest by the complete destruction of all the normal histological characters of human hair. The cortex and medulla are undistinguishable, the surface is rough, the pigment no longer normally distributed, and the free end, instead of tapering to a point or being transversely cut off, is broken, slightly bulbous, ragged, or split into a sheaf of fibres. A less degree of infection is recognised by a few spores in nucleus-like chains or a little branch of mycelium, in the substance of an apparently healthy hair. The whole of the shaft and the root also are full of the disease; but Dr Frederick Taylor has pointed out that the parasitic fungus does not invade the cutis itself, nor even the outer root-sheath of the follicle, and only slightly affects the adjacent epidermis. The inner root-sheath is full of spores, the outer root-sheath free. (Compare his paper, 'Med.-Chir. Trans.' lxii, with Dr Thin's, *ibid.*, vol. lxi.)

The spores differ from oil-drops, with which they are often confounded, in the following particulars: first, they are uniform in size; secondly, they do not run together; thirdly, they are not perfectly spherical, but some at least perceptibly spheroidal or oval; next, they do not refract light so strongly, and though glistening and having a well-marked outline, the centre is not so bright, nor the circumference so broad and black; they occur in little groups or in chains; lastly, potash, instead of dissolving them by forming a soap, as it does with oil-drops, is powerless to affect their protoplasm, which is protected by a cell wall; thus it only serves to bring them out clearly by making the surrounding keratin and oily matter transparent. Ether is also without effect. Carmine and other staining agents act slowly, but in the end stain the cell. Often the most characteristic objects are not the extracted hairs, but short broken fragments, which are conveyed to the glass slip with scales and *débris* from the scalp.

The recognition of ringworm is in most cases sufficiently easy after a little experience, but we must remember that it may be masked by secondary impetigo, as above described; also, that when of long standing, patches of almost bald skin may simulate the atrophic patches to be described (p. 943) as alopecia areata; and, thirdly, that the trichophyton may exist in hair which, as explained above, does not show the ordinary signs of ringworm visible without a microscope. In all doubtful cases, therefore, we must depend upon careful microscopical observation. This is particularly important when we have to decide whether the disease is cured or not. It is only by taking numerous specimens that we can assure ourselves of the fact. We must sometimes, where to the naked eye the ringworm has disappeared, hunt through a dozen slides without finding a single diseased hair, until in the thirteenth we may find unmistakable evidence of the ringworm being still incompletely cured.

Etiology.—The only efficient cause of ringworm is the growth of the *Trichophyton tonsurans*; and its almost universal spread under favourable circumstances shows that individual difference of soil has but little to do with it. All that we can see are differences in the luxuriance of its growth, in the irritation it occasions, and in the obstinacy with which it clings to the affected scalp.

It has often been stated that ringworm occurs chiefly in pale thin children, who are called “scrofulous” or “strumous,” but have no enlarged glands or any sign of tubercle. There is little evidence for this opinion, or for its supposed predilection for light-haired, “lymphatic” children. One often sees ringworm in those who are the picture of rosy health. It occurs more frequently in light-haired children than others, because most children in England have light hair; but it is common enough in those with brown hair, black hair, or red hair.

What is really important in its ætiology is that it is most frequent between the ages of three or four and nine or ten. It is not very common in infants, and when present is usually cured without difficulty. This probably depends upon the less development of hair. Why ringworm of the scalp is so seldom met with in adults is difficult to say. Not only do mothers and nurses rarely take the disease from their children, but when it does occur it is far more readily cured. In children above ten or twelve years old it is easier of cure than in younger ones, and about fourteen or sixteen years of age its treatment seldom gives trouble, and it sometimes disappears spontaneously.

Among 100 consecutive, mostly private, cases of ringworm under the writer’s care, 13 of the patients were between twelve months and five years old, 34 between five and ten, 40 between ten and fifteen, 3 between fifteen and twenty, 9 between twenty and thirty-four, and one was aged fifty.

Ringworm is probably as common in girls as in boys; but of the above 100 patients, it happened that 63 were male and only 37 female.

It must be remembered that the lower animals are liable to this disease, and the source of contagion may sometimes be a cat or a horse or a hedgehog.

Prognosis.—In infants and in adults ringworm of the scalp is a very manageable disease; in children, though the majority of cases may with care and attention be cured, it often proves obstinate, and now and then, in spite of the best available treatment, may persist for years, and at last yield to advancing age alone. In a school or a family, from a third to a half of the cases will be cured in from three to nine weeks, a few of them by three or four days’ application of the remedy. The majority of the rest will yield to persevering treatment in from three to six or eight months. A few only out of a large number will last beyond this time, and some of these are pretty sure to prove inveterate.

Treatment.—The principle of treatment is the same as that of scabies. In both cases we know the cause of the disease; we know the natural history of the invading organism and the means of checking or destroying it. The difficulty in the case of ringworm is that most frequently before the case comes under our observation the fungus has already fixed itself deeply in the hair-sacs of the scalp, and it is extremely difficult to apply remedies to reach it. It is moreover protected by the epithelial scales which closely surround the hair-bulb, and by the sebaceous and other products which

block its mouth. We shall see that when the same parasitic growth invades the surface of the body its cure is easy.

So great are the practical difficulties of treating ringworm of the scalp that, although with perseverance and skill we can cure the vast majority of cases, and some of them rapidly as well as safely, yet everyone who has had much experience in this disease must have met with cases which are so intractable that, when after many months or even years they at last get well, it is to time and the increasing age of the patient that the cure is due.

Preparations of *mercury* are poisonous to all cryptogamic plants, to fungi as well as to bacteria, and probably the most poisonous is corrosive sublimate. A solution of perchloride of mercury in alcohol, two grains to the ounce, is sometimes rapidly effectual in curing recent cases of ringworm. It should, however, only be applied to separate patches, since there is at least one case on record in which its free use over a child's scalp produced (by some unusual accident in the application, or possibly some idiosyncrasy in the patient) absorption of the drug, and death by mercurial poisoning. Aqueous lotions have the disadvantage of being repelled by the oily sebaceous infiltration of the natural and diseased structures of the scalp. We therefore usually prefer either alcohol or ether, or else lard or vaseline as a vehicle. In early cases of ringworm the white precipitate ointment (*ung. hydrarg. ammon.*) is often completely successful. It should be well rubbed into each patch morning and evening after thorough cleansing with hot soap and water and flannel. Instead of white precipitate ointment the *oleate of mercury*, of the strength of one in twenty or one in thirty-five, is effectual, and by many preferred to the older preparation. The 10 per cent. oleate is too strong unless applied to a very small patch in an elder child.

The effect of contact with white precipitate and with citrine ointment upon the life of the *Trichophyton* has been experimentally studied by Dr Thin. His results confirm those gained from clinical experience, as to the sterilising power of the mercurial ointments, while he found that fats and oils, including oleum ricini, have no such effect in themselves.

Another parasiticide which has become popular is tincture of *iodine*. This also is sometimes effectual with recent cases.

There is, however, another method of destroying the fungus which is painful but often efficacious. It consists in setting up a local inflammation, the products of which destroy the parasite. This plan is most applicable to the first stage of the disorder. If a mercurial application does not prove effectual within a few days, then, with elder children, and especially on the first appearance in the family, it is probably better, after isolating the infected member, to attempt the immediate destruction of the fungus by exciting local inflammation. A stronger solution of iodine acts in this manner, but probably the most effectual and least painful is the blistering fluid made of *cantharides*. The affected spot should be first shaved, including half an inch round it, and a circle of oil be drawn round the margin to prevent the blistering fluid from spreading. The pain of its application does not last long, and in many cases success is immediate and complete.

Too often, however, the fungus has already spread too far to be treated in this decisive manner, which is scarcely applicable except to recent cases with only a single diseased patch.

We will suppose that a child is brought to us with the disease established for several weeks, with numerous rings, and, perhaps, with crusts and

pustules from attempts to cure by various irritant applications. The first step is to have the hair cut quite short over the whole of the scalp. Scabs and crusts must then be removed with the help of poultices, and the whole surface made as clean as possible. We then see the real extent of the primary disease. It is often much less than it at first appears; the secondary superficial dermatitis is readily cured, and the diseased patches are soon ready for treatment. Sometimes we find no impetiginous crusts and little active inflammation, but scattered over the whole scalp small spots of ringworm, while the apparently healthy hair between often furnishes evidence of infection. Under these circumstances the shortest and most effectual way is not merely to cut the hair short, but to shave it completely off. In inveterate cases it is much better to wait until the hair is removed, the crusts or scales got rid of, and the inflamed glands reduced, before beginning active treatment. Meanwhile, the whole scalp should be well anointed morning and evening with carbolic oil, one in fifteen or one in twenty, and the child's head covered with a linen cap both by night and by day. In this way no time is lost, and the spread of the affection to other children is prevented. If, without much active inflammation, there is found considerable accumulation of dead epithelium, and especially when it takes the granular adherent character above described, this must be removed with potash-soap, or other alkaline applications. Dr Foulis ('Brit. Med. Journ.,' 1885, vol. i, p. 536) recommended for this purpose spirits of turpentine rubbed in until the child begins to feel it tingle, and then washed off with abundant warm water and carbolic soap. This is sometimes a rapid and effectual treatment, but it is only applicable when comparatively small patches are affected, and should not be used in the case of young children.

When the way has thus been cleared for parasitocides, we may in the slighter cases obtain good results by rubbing into each patch the white precipitate ointment, as above recommended, anointing the intermediate surface with *carbolic oil*. In many cases, however, this proves inadequate, and we must then use stronger applications, although if the disease is extensive they must be applied only to a limited portion at a time. One part of unguentum hydrarg. nitratis and two of sulphur ointment form an efficient and usually not too severe application. Dr Alder Smith, whose experience of ringworm at Christ's Hospital has been very large, recommends in obstinate cases a mixture of carbolic acid one part, citrine ointment one part, sulphur ointment one part. With children under ten, two or three instead of one part of the sulphur ointment should be used, and it will then cause no pain. Instead of carbolic oil (1 in 10 or 1 in 5) the carbolic glycerine of the British Pharmacopœia (1 in 4 or diluted to 1 in 8) is often preferred. It is preferable where lotions are being used. Another plan is to use carbolic oil (1 in 10) to the generally diseased surfaces and carbolic acid lotion to successive portions; but this is apt to produce more pain and less certain curative effects than the compound ointments.

Oleate of copper (3j to an ounce) was introduced by Shoemaker into practice in America, and has been found very useful by Crocker.

Some writers recommend chrysarobin—a mixture of chrysophan ($C_{30}H_{26}O_7$) and its oxidised form *chrysophanic acid*—the efficient constituent of Goa powder, much used in the East Indies.* As stated in the chapter on psoriasis, it is sometimes a severe irritant, and always stains both the skin

* Chrysarobin is the trade name of Goa powder used at Bombay. It is identical with the araroba powder from the trunk of a leguminous tree (*Andira araroba*) of Brazil.

and linen unpleasantly. Chrysophanic acid has been given by Dr Alder Smith, dissolved in chloroform (seven grains to the ounce), instead of twenty grains to the ounce of lard; and he recommends it in recent cases with only one or two spots as more successful than blistering. At the same time he uses a lotion of hyposulphite of soda (two drachms to the ounce) or of liquor sodæ chlorinatæ (one part in eight). Dr Crocker reported an impartial trial of Goa powder in twenty cases of ringworm; only eleven were slightly improved after three months' treatment, and only two were cured. Moreover, an objection to chrysophanic acid is that it is apt to get into the child's eyes, especially during the night. But Mr Malcolm Morris speaks much more favourably of it. (See Dr Walter Smith's paper in the 'Transactions' of the Derm. Soc. of Great Britain and Ireland, vol. iii, p. 17.)

The writer has seen good results from the ointment of *pyrogallie acid*, which is much used against ringworm in Vienna.

Among the more severe applications is one introduced by Dr Coster, of Hanwell Central London Schools, and afterwards published in the 'Medical Times and Gazette,' vol. i, 1867, p. 34. This *Coster's paste* consists of two drachms of iodine dissolved in an ounce of colourless oil of tar, obtained by distillation from common tar, and known as light oil of wood-tar or rectified spirit of tar, sp. gr. 853 to 867. It is applied with a brush to the affected parts and forms a cake which separates at the end of a week or fortnight. (See a letter by Mr Martindale, in the 'British Medical Journal,' January 19th, 1880.) This was used by Dr Ringer at University College with success. Mr Marrant Baker, at St Bartholomew's, preferred iodine in the same proportion with creasote.

The most severe application is *croton oil*, which produces an artificial pustular dermatitis known as "kerion." A favourite ointment both in Germany and France is that which is also used in the cure of scabies, a combination of sulphur with an alkali (Wilkinson's and Vleminecx's ointment). Hardy gives the formula: Carbonate of potash a quarter to half a gramme, sulphur one to one and a half grammes, lard thirty grammes.

Equal parts of lanolin and olive oil form an excellent vehicle for mercury, copper, sulphur, phenol, or chrysarobin.

Instead of ointments the cure of ringworm has often been attempted with etherial and alcoholic solutions. Cavafy ('Brit. Med. Journ.,' June 4th, 1882) devised a lotion composed of boracic acid, alcohol, and ether, in the following proportions: boracic acid, twenty grains; ether, one drachm; spiritus vini rectific., one ounce. The object, of course, is to dissolve the sebaceous material in the hair-sac and thus enable the boracic acid in solution to soak down to the spores which lurk there. This plan has been adopted and recommended by several dermatologists of experience. The writer has found that this lotion, rubbed into the patches not less than four times a day, has proved cleanly and painless. It sometimes effects speedy cure, but, like all other applications, it not unfrequently disappoints us.

Salicylic acid has also been employed dissolved in alcohol, ether, or chloroform; and corrosive sublimate may be used in alcoholic solution—two grains to the ounce.

If watery lotions are preferred, sulphurous acid gas in solution (*Acidum sulphurosum* of the British Pharmacopœia) is one of the best parasitocides. It must be applied on pieces of rag to each patch. Or the hyposulphite of soda (two drachms to an ounce of water) may be used.

Thymol is another unirritating parasiticide which may be employed. It is soluble in alcohol and ether and in oil of turpentine, or may be used as an ointment, 1—3 drachms to the ounce.

Dr Alder Smith recommends Barff's *boro-glyceride* as one of the best applications if the scalp is tender and sore, especially if impetigo is present.

Oleate of copper is an imitation of the old verdigris ointment, as that was of pennies laid in vinegar. It is of a bright green colour, and less irritating than many metallic preparations when employed as a drachm of the oleate to an ounce of lanolin.

When oily preparations are used, frequent washing is unnecessary and even undesirable—twice a week is enough. With solutions in water, alcohol, or ether, constant cleansing with common or soft soap is absolutely necessary.

Is it desirable to aid the action of parasiticides by removing diseased hairs? This plan of *epilation* is generally carried out both at Paris and Vienna, and is adopted by many English physicians. Others believe that it is ineffectual. The fact is that to pull out all the diseased hairs over an extensive surface affected with ringworm is impossible even by a skilled manipulator. A certain number are sure to break off in the forceps, and still more are too short to be laid hold of. Moreover, the attempt is extremely tedious and painful, and the result insignificant. Where, however, a very small patch is for the first time seen, it is well to pull out at once all the hairs not only from the obviously diseased skin, but from a small circle around, before applying acetic acid, blistering fluid, or any other agent by which we hope to destroy the parasite at once. Again, in chronic and extensive cases, removing loose hairs helps to prevent contagion, to clear the scalp, and also to ensure minute observation and care on the part of the nurse. It is therefore well to give her a pair of broad-tipped, well-roughened, and weak-springed forceps, and to instruct her to remove every morning, after washing the head, as many hairs as may be loose, but not so as to cause the child pain.

Prophylaxis.—While ringworm is under treatment the whole of the child's hair should be kept short—cut, in fact, as close to the head as may be; and this is probably as effectual as shaving. With girls a fringe of hair may be left round the forehead and behind the ears, so that a cap may be worn during the day, and the child's appearance attract no attention out of doors. At night a linen cap should be used. Impervious coverings of gutta percha or oiled silk make the scalp hot, and are unnecessary.

There is no need for a quantity of ointment to be left on the scalp at night. The free application of carbolic oil, or carbolic glycerine, or oleate of mercury to the head is best undertaken in the morning. Although mothers and nurses very rarely suffer from the most assiduous dressing of ringworm, it is well to instruct them to anoint their hands with carbolic oil each time they touch the child's scalp.

With these precautions, and scrupulous avoidance of contact with caps, brushes, &c., it is possible for a child with ringworm to be treated and cured without removal from the family. But if the infected member or members cannot be separated from those who are healthy, they should sleep in separate bedrooms, and, if possible, meet only out of doors. It generally happens that in a family, while most of the cases are cured quickly, there remain one, or perhaps two, extremely obstinate. These may, if necessary, be removed for the sake of treatment; but practically when the child has

once been cured it is little liable to take the disease again, especially if the hair is kept short, if carbolic oil is used as a pomade, and if the nurse (who if at all intelligent will by this time be able to recognise the disorder) is careful to wash and inspect the scalp every week.

The only proof of complete cure depends upon the careful microscopical examination of the hairs, not only from the previously diseased spots, but from the surrounding scalp. When the skin is itself healthy, and the hair which grows on it is soft and downy, when no broken stumps and black points are seen under a lens, and when these good signs are associated with an absence of spores in the hairs examined, we may pronounce the child to be cured. It should, however, not be sent back to school for at least a fortnight after this, and should then be carefully examined again before the risk of relapse can be considered past.

When the ringworm fungus gains admission to the deep hair-sacs of the beard, it presents a somewhat different clinical aspect, and is apt to be inveterate. It is, of course, only met with in adult men, and is frequently complicated by the presence of pyogenic cocci, which produce an appearance indistinguishable except by microscopical examination from the non-parasitic sycosis already spoken of (pp. 919, 921). The patients are usually men under thirty.

RINGWORM OF THE BODY.—*Tinea circinata*.*—This affection occurs in the form of small rings with a red, papular, vesicular, or scaly margin. They are most common on the face and neck, but are sometimes seen on the trunk or limbs. Among 50 cases the writer found the eruption confined to the neck, cheek, or other parts of the face in 23, affecting the trunk in 12, the shoulder, arm, or forearm in 10, and the thigh in 5. The majority of the patients were children under twelve, 1 was fifteen years old, 2 seventeen, and 8 were adults. Ringworm of the body seldom causes much irritation.

The disease is contagious, and if scrapings from the ring are placed in *liq. potassæ* under the microscope, the mycelium and spores are apparent. There is more of the former in proportion to the latter than in ringworm of the scalp, and the fungus is not so readily seen, but after thoroughly soaking in potash it can always be discovered. The large-spored variety is the one usually present both in England and on the Continent.

It often appears in children along with common ringworm, but may also be seen when the scalp is quite free from disease. It occasionally occurs in adults, especially in the form which will presently be described.

Burmese ringworm was described by the late Dr Tilbury Fox as nothing but a somewhat severe and troublesome form of *Tinea circinata* (see his account of this and other exotic forms of ringworm in his work on 'Skin Diseases,' p. 541). *Tinea imbricata* is the name given to the ringworm of Tokelau, Malacca, and other parts of the South Seas by Dr Geo. Turner and Dr Patrick Manson ('Edin. Med. Journ.,' with figs. by Dr McCall Anderson, Sept., 1880; and 'Brit. Journ. of Derm.,' 1892).

Tinea marginata.—There is a form of tinea only observed in adults, and of which the parasitic nature was first recognised by Köbner. It was formerly called *eczema marginatum*.

Its distribution is very characteristic. Unlike all other forms of ringworm, it is symmetrical, and occurs only on the thighs, abdomen, perinæum,

* *Synonyms*.—Herpes circinatus—Vesicular ringworm—Trichophytic circinée.

and buttocks. It begins, probably in all cases, with minute spots, which rapidly form rings; but as these extend and coalesce, they produce gyrate figures, as above explained in the case of psoriasis, erythema, and other disorders which spread at the edge. When a case comes before us it has usually already assumed its characteristic aspect of a somewhat sinuous, broad, yellowish or brownish red, more or less inflamed band, which runs over the upper and inner part of each thigh, passes back to the fold of the nates, or the sacrum, and returns over the lower part of the abdomen or the groin to the pubes. This peculiar distribution no doubt depends upon the mutual contact of the parts, and is aided by the warmth and perspiration which favour the growth of the fungus. The centrifugal spread is that of all forms of tinea, but the central parts are sooner free from the disease, the margin is more inflamed, and the duration more prolonged than in tinea of other parts of the body.

This curious affection is apparently confined to the male sex and adult age. It is most common in those whose occupation necessitates a sitting position for a long time; for instance, in cobblers and cavalry soldiers. There is generally much irritation and discomfort; and, like all long-continued forms of dermatitis, it produces pigmentation, not only in the growing margin, but also upon the inner exhausted surface.

The microscope demonstrates the same mycelium as is found in *Tinea circinata*; but the disease may last for ten years or more, and when of very long standing it is often difficult, and sometimes perhaps impossible, to discover the parasite. Fortunately the aspect and locality are sufficiently characteristic.

Treatment.—*Tinea circinata* is very easy of cure. Among popular domestic remedies, ink is one.* White precipitate ointment or oleate of mercury, verdigris ointment (subacetate of copper two scruples, benzoated lard one ounce), tincture of iodine, boro-glyceride, sulphurous acid in solution may each be employed with a certainty of speedy cure, in striking contrast with their action in ringworm of the scalp. In England there is seldom need for resorting to the more severe parasiticides, but in India Goa powder (chrysarobin, chrysophanic acid) was first introduced for so-called Burmese ringworm.

Ringworm of the body is, of course, contagious, and may not only propagate itself, but may lead to the development of tinea in the scalp. Its easy cure, however, renders precautions by isolation almost unnecessary.

Tinea marginata, however, is, as above stated, very obstinate and difficult of cure. Sulphurous acid of the British Pharmacopœia freshly made, hyposulphate of soda (a drachm to the ounce), boracic acid (ten grains to an ounce of spirit), and corrosive sublimate (two grains to an ounce of water), may each be used with good effect.

In one very obstinate case, in which the patient, there seemed no doubt, had contracted the disease with a pair of knickerbockers which had been mended by a village tailor in Switzerland, most of these remedies were tried ineffectually for some months. At last the effect of pyrogallie acid ointment (half a drachm to the ounce of benzoated lard) was so rapid and unmistakable that the patient complained of my not having prescribed this ointment first instead of last.

Onychomycosis.—It is happily very rare for tinea to attack the nails.

* "How shall we make this mode of writing sink?

A mode, said I? 'tis a disease, I think,

A stubborn tetter that's not cured with ink."—CONGREVE.

When present, ringworm of the nails is usually a complication of ringworm of the scalp. Cases were recorded by Meissner in Vierordt's 'Archiv,' by Virchow and by Bazin, as early as 1853. It was carefully described and the microscopic appearance figured by Neumann ('Hautkrankheiten,' p. 347, figs. 48 and 49), by Dr Purser ('Dubl. Quart. Journ.,' Nov., 1865), and by Dr Fagge ('Guy's Hosp. Rep.,' 3rd series, vol. xv, p. 553, and 'Clin. Trans.,' vol. i, p. 77). The nails become yellowish and brittle, but not rough as when affected by eczema or psoriasis. The fungus is occasionally that of favus (*achorion*, to be next described), but more often that of common ringworm (*trichophyton*). Good models of onychomycosis due to each of these parasites will be found in the Guy's Hospital Museum, Nos. 536 and 537.

It is the most obstinate of all forms of ringworm, and will often persist during the whole of childhood, and only disappear after puberty.

The *treatment* recommended is scraping the affected nail, softening it with alkalies, and when other means fail, complete removal under chloroform, together with the sedulous application of sulphurous acid or hyposulphite of soda; but in one case an eminent dermatologist adopted this method, in addition to every other known parasiticide treatment, for more than ten years without curing the disease.

Tinea erythrasma.—Under this name has been described a slight form of inflammation of the skin, which is due to a fungus, but to one distinct from *tinea versicolor*. It occurs, not on the exposed surfaces of the trunk, but where two parts come in contact, most often on the scrotum and adjacent part of the thigh, sometimes in the cleft of the buttocks or in the axilla. The patches are pigmented, dull reddish yellow or reddish brown, with a slight branny desquamation. The growth of the fungus is evidently favoured by warmth and moisture, but it does not produce the irritation of intertrigo or of *tinea marginata*; it spreads very slowly, and, in fact, is rather a blemish than a disease. The fungus was named by Burchardt and Bärensprung *Microsporon minutissimum*. Dr Payne* has described and figured it, and regards it as a specific organism, though mixed with other epiphytes, without spores, and rather like an involution-form of a bacterium than a true fungus. It consists of jointed threads as well as cocci, and requires staining and a high power to be identified. The patches must be distinguished from other kinds of *tinea* and from syphilitic maculæ. They are readily cured with sulphurous acid or mercurial ointment.

FAVUS.†—This is a rare affection of the scalp and body, due to the presence of a fungus (*Hyphomyces*) named *Achorion Schönleini*.

The disease was recognised and named by Bateman, and was figured by Alibert. But it was not till 1839 that Schönlein published in Müller's 'Archiv' the discovery that the yellow crusts of favus were neither pustular nor sebaceous, but were composed of the mycelium and conidia of a parasitic fungus. This discovery preceded that of Malmsten above mentioned (p. 858), and therefore to Schönlein belongs the merit of opening the whole chapter of cutaneous mycology. Recent observations, however, by Unna, Bodin, and Sabouraud point to more than one origin of the clinical condition favus.

* 'Path. Trans.,' 1886; 'St Thos. Hosp. Rep.,' 1887; and 'Rare Diseases of the Skin,' p. 31.

† *Synonyms*.—*Tinea favosa*—*Porrigio lupinosa*.—*Fr. Teigne favense*.—*Germ. Erbgrind*.

In its earliest stage favus is probably undistinguishable from common ringworm, but very soon a characteristic flat, round, yellow object is seen, depressed in the middle, opaque, adherent, and perfectly dry. Its colour has been compared to a honeycomb (*favus*), and its shape to the disc of a lupine seed. The sight of a single case of the disease, of such models as Nos. 523—527 in the Guy's Hospital Museum, or even of a well-executed drawing, is sufficient to enable any one to recognise favus.

The individual crusts grow, coalesce, and form thick, rugged, porous, yellowish masses, resembling the rind of old worm-eaten cheese. They have a characteristic mouldy odour like that of mice.

The disease may affect any part of the body, but is particularly severe upon the scalp, where it destroys the hair-sacs and often produces complete baldness. It is, as above stated, extremely rare in England, but is less so in Germany, and comparatively common in France. It appears also to be not infrequent in Scotland. Mr Hutchinson published forty-four cases with instructive remarks upon the disease in the 'Med. Times and Gazette' for 1859 (vol. ii, p. 553).

Unusually severe and generalised cases of Favus have been described, and the fungus has even invaded the alimentary canal.

Favus was recorded by Dr Purser, of Dublin, in a cat (1866), and by St Cyr in rabbits and mice ('Ann. de dermatologie et de syphilographie,' 1869). It has since been observed in dogs and in domestic fowls.

The *treatment* is unsatisfactory. Ordinary parasitocides produce improvement, and if perseveringly employed, apparent cure; but relapse is almost sure to occur. The old French treatment of epilation by a cap of pitch-plaster, applied to the head and then forcibly pulled off, is no more effectual than less barbarous methods; but epilation is probably necessary for even a temporary cure (see Dr Bulkley's paper in the 'Arch. of Derm.,' April, 1881). More than one apparent cure by epilation was recorded by Dr Fagge in the 'Guy's Reports' for 1870 (p. 354).

TINEA VERSICOLOR.—This affection, described by Willan as *Pityriasis versicolor*, was formerly named *maculæ hepaticæ*, a translation of the vernacular German name *Leberflechte* (liver spots, *chaleur de foie*). Another name still often applied is *chloasma*, but this is better reserved for true *maculæ* produced by pigment.

In 1846 Eichstädt published in Froriep's 'Journal' the discovery that this affection is due to the presence of a fungus. Now that its real nature is understood, it is curious to read Bateman's remark, that "the causes of this pityriasis are not well ascertained; fruit, mushrooms, sudden alternations of heat and cold, violent exercise with flannel next to the skin, have been mentioned as probable causes; the most extensive eruption I have seen occurred in a Custom-house officer after drinking spirits freely during a day of fasting on the Thames."

Tinea versicolor occurs as yellowish-brown spots of various shades, scarcely rising above the level of the skin, and yielding a branny or furfureous desquamation when scratched. The spots vary from a pin's head to several inches in diameter. As they multiply and coalesce they form larger patches and thin rings, which, when united, produce the gyrate or serpentine outline before described as the result of this mode of development of an eruption. It is rare, however, to see such perfect rings as in

tinea circinata, and the central parts seldom completely recover, but remain more or less discoloured.

The *distribution* of this affection is very characteristic. In the great majority of cases it occupies the chest, it often spreads to the abdomen, and is frequently seen on the back, especially between the shoulders. It may overspread the whole trunk, but rarely descends below the waist or ascends above the neck. Occasionally a patch or two may be also found on the border of the axilla and on the soft skin of the inner part of the arm and on the bend of the elbow. Even when the abdomen is not affected it is common to find this form of tinea on the inner side of the thigh, whence in males it is apt to spread to the scrotum. We may say, therefore, that the affection never occurs upon parts which are exposed to the air, and that its favourite seat is on skin which is the most protected and the most constantly warm and moist.*

On scraping some of the surface and putting the scales in a drop of *liquor potassæ* under a microscope, both spores and mycelium can be seen without difficulty. The spores of the fungus, *Microsporon furfurans*, are somewhat larger than those of *Trichophyton tonsurans*, and occur in heaps, which are surrounded by mycelium threads.

The presence of the fungus is of course the decisive point of diagnosis; but with a little experience the colour, the branny desquamation, and the locality of this affection are sufficiently characteristic.

Sometimes patients complain of the irritation occasioned by tinea versicolor, and it may be accompanied, especially in hot weather, by slight erythematous dermatitis or urticaria, as the result of scratching. Most frequently, however, it produces no symptoms whatever, and is either discovered accidentally, or is regarded only as a disfigurement. The superficial layers of the epidermis are alone affected by the parasite.

The *cause* of this curious affection, or rather of the fungous growth on which it depends, is quite unknown. It is remarkable that it seldom, if ever, occurs in children, and is rare after middle age. It is most often seen in men between twenty and forty, but may also be observed in women, especially under the fold of the mamma. Although the fungus has been proved by experiment to be capable of transmission by direct inoculation, the disease is not practically contagious, or, if at all, to a very small degree.

Treatment.—If left alone tinea versicolor continues indefinitely, but it may be readily removed by any of the milder parasiticides. After thorough washing with hot soap and water, or, if a rapid cure is desired, with soft soap, the affected parts must be well rubbed with oleate of mercury or unguentum æruginis (Θij ad ʒj); or, if preferred, solution of sulphurous acid or hyposulphite of soda (ʒj ad ʒj) may be applied in watery solution.

Tinea rosea (?)—*Pityriasis rosea*.—This affection was first described in 1860 by Gibert as an acute centrifugal erythema of the trunk. Bazin called it *P. rubra maculata et circinata*; Behrend, *Roseola furfuracea herpetiformis*. Vidal regards it as parasitic ('Trans. Intern. Med. Congr.,' vol. iii, p. 133). Some German writers agree in this view, and follow Hebra in classifying it as a variety of ringworm of the body, *Herpes tonsurans maculosus*; but probably the disease described by Gibert, Duhring, and Colcott

* Contrary to the almost invariable rule in Europe and America, true *T. versicolor* of the face is common in Assam (Dr Powell, of Cashar, 'Trans. Derm. Soc. Gt. Brit. and Ire.,' vol. vi, p. 52).

Fox is distinct from any form of *tinea corporis*, and it has therefore for convenience been described in the chapter on Pityriasis (*supra*, p. 876).

It will be convenient to consider in this chapter the remaining affections of the hair, some of which were formerly confounded with ringworm, and are still liable to be mistaken for it.

ALOPECIA.*—Baldness, or loss of hair, when not the result of the presence of a fungus, is the immediate consequence of atrophy of the hair-bulbs, which occasions the premature fall of the hairs from the follicles. When this is only partial, and followed by fresh growth of weak hair, the result is thinness or partial baldness; but when the hair-sac is no longer capable of producing a fresh hair complete alopecia results.

Although a senile change, baldness cannot be considered strictly physiological; for it is often absent even to advanced age in men, it is usually absent and rarely complete in women at any age, and it sometimes occurs very early without any other signs of senile decay. In these cases it is frequently hereditary, but by no means constantly so.

The atrophy of the hair-sacs certainly does not depend upon general deficiency of healthy nutrition, nor upon locally deficient supply of blood. It is not accompanied by anæsthesia, by numbness, or by any other evidence whatever of nervous disorder, so that to ascribe alopecia, whether premature or not, to "vascular" or "neurotrophic disturbance," is an arbitrary hypothesis. It has been asserted that adhesions of the pericranium, and particularly want of mobility of the aponeurosis of the occipito-frontalis, produces alopecia, but many instances disprove the assertion. Neither wearing tight hats, nor going without hats, nor wearing turbans indoors, nor exposure to the sun—nor gout, nor scrofula, nor intemperance, nor abstinence—none of these will in the least explain either senile or premature baldness, for each supposed cause fails on examination.

Alopecia of this quasi-physiological character begins usually in the frontal region, sometimes at the central point at the back of the head from which the hair falls forward, backward, and laterally; and not infrequently in both regions at once. There is often *seborrhœa sicca* or a slight degree of pityriasis which precedes and accompanies baldness; but if this is the cause the thinness of hair can be cured by restoring the skin to a healthy condition, and even if neglected it does not go on to complete alopecia. Moreover, in many cases both of senile and premature baldness the skin is healthy throughout. When the hair has fallen off from the mid-region of the scalp the process almost always ceases, and that on the temples, behind the ears, and on the occiput persists without change. This ordinary alopecia, moreover, never affects the beard, the eyebrows, or other parts of the body.

Many attempts are from time to time made by physicians, as well as by hair-dressers, to check the loss of hair or to restore it. They consist either in applying stimulating lotions, of which cantharides is usually the basis, or in shampooing and manipulating the scalp. It is very rarely that these attempts have even partial success. A process introduced a few years ago by Dr Pincus, of Berlin, promised better, but had not in the sequel fulfilled the expectations of its author.

* *Synonym.*—*Calvities*, or more frequently in classical Latin *calvitium*. *Atrichia* is a modern name. *Alopecia*, ἀλωπηκία, fr. ἀλώπηξ, "quod vulpes hoc malo sæpe corripitur," is the real Greek name. Celsus distinguishes it by its occurrence in patches and only in adults from ὀφίασις, which spreads in a serpentine form on the back of the head in children.

Alopecia as the result of febrile diseases.—Although this often proves the first step of ordinary baldness, yet it is distinguished therefrom by its affecting both sexes and all ages, by the fall of hair not being confined to any region of the scalp, and by its thinning rather than completely stripping the surface affected. Moreover, it is not only secondary in origin, but usually passes away of itself after convalescence, instead of being practically incurable, either by nature or by art.

Syphilitic baldness agrees in these characters, and its frequency, apart from any other affection of the scalp, as well as its early appearance, likewise point to its ætiology as a febrile alopecia.

AREA.*—A curious and not uncommon form of baldness appears in round, smooth patches on the head, most often in children. The skin is often as polished and white as ivory, and the margin is abrupt. There are almost always more patches than one, and as they grow they may unite and form larger *areæ*. The skin looks thin, the patch is depressed, and the hair-sacs and sebaceous glands seem atrophied.

The patient is in most cases at or under puberty, but *area* is also seen in adults, and may then affect the hair of the beard or pubes. The eyelids are sometimes involved with the scalp. The process of atrophy may go on until little hair is left on the head or elsewhere, but it very seldom or never ends in such complete alopecia as will presently be described. So peculiar is the appearance of this disease, that it is less needful to insist upon its distinction from other kinds of alopecia than upon the fact that it is a true alopecia, anatomically identical with the other forms of atrophy of the hair, though differing in its origin and course. It is independent of the presence of a fungus.

Hebra believed at one time in the statement of Gruby that alopecia areata was parasitic, but before long changed his opinion, so that one can only share in the surprise expressed by Kaposi that Hebra was associated with Bazin as a supporter of the parasitic nature of *area* by his disciple Dr Neumann. It is probable that the single observation of Gruby in 1843 ('Comptes Rendus,' xvii), which gave rise to the question, was made upon a case of true ringworm, and that he did not mistake, but misnamed it. The French dermatologists call many cases "*pelade*" or "*teigne pelade*," which in England or Germany would be regarded as true ringworm in its later stages. In M. Hardy's lectures it is not difficult to recognise in the swelling, irritation, and desquamation of the skin, which he describes in *pelade*, the characters of ringworm. The fact seems at present to be that the condition of *area* may, and often is (perhaps most often in France), the final result of ringworm, but that it is also (and in England and America most often) without evidence of this origin. Whether these cases are due to one of the numerous bacteria (not fungi) which have been described remains to be seen.

Apart from the microscopic evidence, the naked-eye appearance and natural history of the disease would almost disprove the parasitic hypo-

* *Synonyms.*—Alopecia areata (Sauvages)—Area Celsi—Porrigio decalvans (Willan)—Teigne pelade (Bazin)—Tinea decalvans. Celsus did not particularly describe this variety of baldness, but applied the word "*area*" ("a bare space," *locus sine ædificio*) to any form of baldness, distinguishing *ἀλωπηκία* and *ὀφίασις* as varieties. The "*porrigio*" of Willan meant any eruption of the scalp, including true ringworm and impetigo or pustular dermatitis, and the term is now almost out of use. The appellation *Tinea* or *Teigne* depends upon the erroneous doctrine of the parasitic nature of the disease.

thesis. The hairs around the affected spot are not swollen at the roots, nor brittle in the shaft, but are either atrophied, like normal hairs which are ready to fall out, or at the margin of the area are thin and shrivelled at the end attacked, so as to be club-shaped or like a note of admiration. There is no evidence of local irritation in the hair-sac. The disease, above all, is not contagious, at least as we observe it in England, and it is not curable by antiparasitic treatment.

Admitting that area is not a true tinea, Mr Hutchinson believes that it may be the result of ringworm, and certainly persons who suffer as adults from area sometimes tell one that they had ringworm in childhood. Whether this occurs too frequently to be regarded as a mere coincidence is doubtful. Certain it is that many, probably most, cases of area have not been preceded by ringworm, and that most cases of ringworm are not followed by area.

Dr Thin ('Proc. R. Soc.,' 1881, No. 217) has figured minute schizomycetes, which he calls *Bacterium decalvans*, but which are rounded rather than rod-like, and probably identical with those described by Dr v. Sehlen in 'Virchow's Archiv.' Even if this were of ætiological importance, it would not make area a true tinea.

The same criticism applies to Sabouraud's hypothesis, that area is caused by one of the microbes of *Seborrhœa capitis*; the latter complaint is far too frequent to be the efficient cause of the former. Moreover, though many cases of area occur in people with scurfy heads, others occur in those who are quite free from seborrhœa.

Another theory is that area is a tropho-neurosis, but of this there is at present no sufficient evidence. There is no anæsthesia of the bald patches to be demonstrated in at least the great majority of cases, and no frequent coincidence with other tropho-neuroses or with "nervous" antecedents.

Area is certainly more common in children and young adults than after thirty. Among 112 patients under the writer's care, 43 were children from four to fifteen, 52 were young men or women from sixteen to thirty, and 17 were above thirty, one being forty-seven and one fifty-eight. In the last case area supervened after ordinary senile alopecia had begun to appear, and the two affections were perfectly distinct ('Guy's Hosp. Rep.,' vol. xlv, p. 373).

Of these 112 cases of area, 72 occurred in male and 40 in female patients. There were several cases of recurrence of the affection in the same patient, and three of its appearance in two or more children in the same family (see Dr Tyson's case, 'Clin. Trans.,' 1886). Such cases may be really due to contagion of a trichophyton.

A second or third attack of area sometimes follows after the first has been completely cured and an interval of time has elapsed.

Treatment.—In many cases area would probably pass away of itself, but recovery is often hastened, if not brought about, by treatment. This consists in local irritants, and, when necessary, internal corroborants. We may begin with a lotion containing $\frac{3}{ss}$ to $\frac{5}{ij}$ of acetum cantharidis to a pint of water. This will often cause slight erythema in children, but in adults and in many children we may increase the strength to two, three, or four ounces with advantage, letting the irritation subside whenever it goes beyond redness. A mild and often efficient application is *linimentum myristicæ* (one part of the expressed oil to three of olive oil). With brown hair the *unguentum iodi* of the Pharmacopœia is a useful application.

Area occurs in persons of all degrees of health, complexion, and temperament; but if the patient is pale and thin, tincture of steel is certainly useful, and bark or cod-liver oil may be prescribed when indicated by some other symptom than the bald patches.

The writer has prescribed *Tr. Jaborandi* with seeming benefit in several cases of area; or a solution of pilocarpine may be injected under the scalp, but both methods often fail. Dr Pringle, among other successful cases, reported one in a woman of sixty.

Universal alopecia.—There are some cases of complete and rapid loss of hair which are neither senile, syphilitic, nor febrile, and which cannot be classed as examples of area. They are distinguished first by the hair falling off almost simultaneously from the whole of the scalp, not gradually from certain regions as in ordinary baldness, nor by the confluence of separate patches, as in area; secondly, by the baldness not being confined to the scalp (nor even to the scalp and beard or eyebrows, as is occasionally the case in area), but affecting the whole of the body; thirdly, by its not following an illness.

In one case of this kind the patient was a young man in robust health, and wearing a full beard. Without any assignable cause he lost the whole of the hair off his body in a very short space of time.

This universal alopecia occurs in both sexes, always beginning in adult life, and usually in young adults. It is quite incurable.

Nine cases have come under the writer's care, the ages being 9, 17 (*bis*), 25 (*bis*), 23, 35, 40, 55: five were men and four were women. In three of them the baldness had begun some years before, then the hair had more or less grown again, and, lastly, a fresh *defluvium capillorum* had ended in total alopecia.

We may at present distinguish these somewhat rare cases of *alopecia universalis acquisita* from the still rarer cases of *congenital alopecia*. In these the nails, as well as the hair, are affected; and like other deficiencies of development, the condition may be hereditary. Such cases are comparable with congenital ichthyosis, especially in such marked examples as the "porcupine boy;" and still more closely with the "hairy family" of Burma, and the blue and hairless horse exhibited a few years ago in this country.

The following striking series of examples of congenital baldness occurred in Guy's Hospital under Dr Fagge. It is remarkable that the development, both of hair and nails, was tardy and imperfect, but not absolutely deficient. The italic letters denote the female sex, as in Mr. Francis Galton's nomenclature.

F. Born without hair or nails. Hair began to grow when he was about twenty-three years of age, and at thirty he had a full head of hair. The finger-nails also grew after puberty, but were always ill-formed, and he never had toe-nails. *F*. Normal.

B. 1. Born without nails or hair; the former appeared while teething, the latter when she was ten years old. *n*. Born without hair and nails; none yet grown.

B. 2. Born with hair but without nails; died, aged seven. B. 3. Born without hair or nails; died, aged five months. B. B. 4—9. Born with normal hair and nails.

B. 10. Born partly bald, with ill-formed nails; he is now twenty-two and has a fair head of hair, but his nails are not good.

The patient herself, then nineteen years old, the eleventh and youngest of this large family, was born without hair or nails. She had, in 1876, only thin lanugo on the scalp and imperfect nails.

TRICHOCLASIA (*Wilson*).—A singular disease of the hair which has been described under this title, and also as *fragilitas crinium* and *Trichorrhæxis nodosa* (Kaposi), is characterised by each hair dilating at intervals and breaking at these enlarged points. The dilated node consists of separated cortical fibres which look very much like the splitting and enlargement of a cane when broken across, and the air which enters between the fibres makes them appear white by reflected light. They have thus a superficial resemblance to the ova of pediculi.

It is almost always confined to the beard, and is non-contagious and non-parasitic. It was described by Devergie as “trichoptylose,” and subsequently by Beigel and by Wilks. The writer has seen three or four cases of it, one of which he figured in the ‘Pathological Transactions’ for 1879 (p. 439), where references will be found to the scanty literature of the subject.*

This is apparently quite distinct from a parasitic affection of the hair known as “Piedra” from its stony hardness, which occurs in the hair of the scalp, among women only, in Central and South America. It has been described by several French writers and by Mr Malcolm Morris in the same volume of the ‘Pathological Transactions’ (p. 441).

A third distinct disease or malformation of the hair consists in “beading,” like that of *Trichoclasia nodosa*; but the hairs break at the internodes, not at the nodes. Cases have been published by Dr Walter Smith, Dr McCall Anderson, and Dr Payne (see the account and figures of the last writer in his ‘Rare Diseases of the Skin’). It is identical with *Monilithrix* or beaded hair, which has been observed as a congenital condition in more than one member of a family.

Lastly, there is a very rare condition known as *Leucotrichia annularis* or ringed hair, first described so long ago as 1846 by Dr Karsch. The hair loses its lustre, and air bubbles form in alternate segments of the shaft. Cases since observed by Wilson and others at home and abroad agree in the patients being young and in other distinctive characters (‘Brit. Journ. of Derm.,’ vols. v, viii, and xiv).

AFFECTIONS OF THE NAILS.—The nails as epidermic appendages share in the disorders of the horny layer and the matrix or nail-bed in those of the *rete mucosum* and *corpus papillare*.

In cases of eczema of the fingers and toes, the nails are seldom affected; but occasionally the inflammation spreads to the matrix, and causes irregularly thickened formation of horny material; or, if more severe, scanty, ill-developed nail-tissue. In cases of chronic eczema of the fingers, the nails sometimes present a curious “pitted” appearance from numerous depressions.

More frequently psoriasis involves the nails, making them thick, rough, and opaque, and loosening their hold on the underlying matrix. Mr Hutchinson has seen this condition without psoriasis of the skin, and finds that it yields to the administration of arsenic. (‘Brit. Journ. of Derm.,’ 1899, p. 306.)

* See also a valuable paper, with fuller references, by Dr T. C. Fox, in the ‘Lancet,’ Dec. 7th, 1878, and Hans v. Hebra, *loc. cit.*, p. 391.

Pityriasis rubra and Pemphigus occasionally spread to the matrix, and cause destruction of the nail-tissue, with more or less complete loss of them.

Syphilitic disease of the nails is not common; most frequently it leads to suppuration (*onychitis*), but sometimes to great thickening, without pain or inflammation.

In cases of peripheral neuritis of the limbs—from leprosy, diabetes, or alcohol—as well as in similar trophoneuroses, as the result of injury to a nerve-trunk, of syringomyelia or pachymeningitis cervicalis, “painless whitlows” form under the nails.

Minor affections of the nails are:—encroachment of the skin, which grows forward like the pterygium in the case of the eyeball; the formation of opaque white spots, which are probably formed in the same way as one kind of white hair, by inclusion between the horny scales of air, a senile change; “reeding” of the nails by longitudinal furrows; and transverse depressed lines which tell of a precedent febrile illness, as long ago described by Wilks; less frequently opaque white lines without furrowing have been seen to follow the like conditions (Langdon Down, ‘Path. Trans.,’ 1870, p. 409).

The curved nails (*ungues adunci*) of phthisis have been recognised from the time of Hippocrates. They are best marked in chronic cases with fibrous transformation and hypertrophy of the right side of the heart. And this fact agrees with the same form of nails being present in cases of chronic bronchitis with emphysema, of inveterate asthma, of empyema, and of valvular disease of the heart, particularly that of congenital origin. The common condition is that of systemic venous congestion from obstruction of the pulmonary circulation. It is most rapidly produced in children with empyema. It is quite distinct from the “filbert” nail of health, and its relation to the “clubbed” fingers which often accompany it is that of the earlier stage in the same process. It affects the nails of the toes as well as those of the fingers.

When neglected, the nails may grow to an extent rivalling those of Nebuchadnezzar—as much as four to six inches, *onychogryphosis* (‘Path. Trans.,’ xii, p. 240). True horns may grow from sebaceous cysts, as in a remarkable case removed by Mr Cock.

CHRONIC DEEP INFLAMMATIONS

WITH CUTANEOUS HYPERTROPHIES AND OTHER CHANGES OF UNCERTAIN NATURE

“His face is all bubuckles and welks and knobs and flames of fire.”

Henry V, iii, 6.

Deep and chronic dermatitis—its definition—its relation to superficial dermatitis—to hypertrophy—and to new growths.

GUTTA ROSEA—*Origin in recurrent erythema—development—localities—causes and pathology—relation to dyspepsia—to drink—to ovarian irritation—treatment—Chilblains.*

EPIDERMIC AND PAPILLARY HYPERTROPHIES—*Callosities and corns—Leucoplakia lingualis et buccalis—Warts—Condylomata and mucous patches.*

ICHTHYOSIS—*Anatomy—varieties—xerodermia—Prognosis and treatment—Ichthyosis intra-uterina—Horns—Hyperkeratosis centrifuga.*

SCLERODERMIA—*Scleriosis and morphæa—History—description—distribution—histology—diagnosis—prognosis and treatment—Sclerema neonatorum—Linear atrophy.*

ELEPHANTIASIS—*Nomenclature—anatomy—pathology—relation to chyluria and filaria sanguinis—clinical characters—Dermatolysis.*

XANTHELASMA—*History—course and symptoms—histology—relation to jaundice—Xanthoma diabeticorum.*

As stated in the introductory chapter, the great majority of affections of the skin consist anatomically in superficial irritative exudation; that is to say, they are confined to the papillary layer of the cutis and the Malpighian layer of the epidermis, with the resulting changes in the cuticle. In no form of superficial dermatitis are the papillæ destroyed; and no scars result.

We have now to speak of a far less frequent kind of inflammation of the skin, which involves, if it does not originate in, the deep layer of the cutis, which destroys the papillæ, which spreads from the skin proper to the subcutaneous connective or adipose tissue, and which after recovery leaves scars behind. Eczema, psoriasis, and their allies—scabies, the erythematous eruptions, and the tineæ—are all, in the sense in which the word is here used, superficial; and however severe and protracted their course, they leave, when cured, either no trace behind or only pigmentation.

When, however, inflammation occupies the hair-sacs and the sebaceous glands a cicatrix is not infrequently the result.

Thus acne in its severe forms leaves scars behind, varying from white spots, very slightly depressed and otherwise inconspicuous, slight local atrophies, up to the hypertrophied scars which sometimes simulate cheloid. The same applies to sycosis, though obvious scarring is less frequent. Some other pustular diseases destroy the papillæ and thus produce scars. This is never true of impetigo, nor of the pustules of scabies or bullæ of pemphigus; but variola, when unmodified by vaccination, almost always leaves indelible traces of its presence—either deep-pitted, depressed, white scars, or more extensive and hypertrophied puckering. The same is true, though less constantly, of varicella, and a deeply pitted cicatrix is the well-known mark of successful vaccination. Lastly, the pustules of zona very often (though by no means constantly) leave more or less marked cicatrices; and sometimes, especially upon the forehead, these are deep and indelible.

But beside these deep pustules, we meet with inflammation of the skin which, uniformly and over large surfaces, penetrates below the papillæ and affects the whole thickness of the integument, together with the subcutaneous tissue. This *deep dermatitis* is usually chronic in course; or, if it shows acute characters, they are repeated again and again, without any tendency for the malady to come to a natural end. Such recurrent subacute diseases become practically chronic, as we see in the case of inflammations of the bronchial tubes, of the conjunctivæ, and of the colon.

Like other chronic inflammations, those of the skin show in many cases little of the classical signs of the process, and are unattended with fever; moreover, the exudation is never purulent, but if œdematous gradually assumes the characters of *œdema durum*; if congestive, those of hypertrophy. The exuded leucocytes, instead of dying and undergoing transformation into pus-cells, become organised into connective-tissue corpuscles, and gradually form fibres. Thus chronic inflammations are closely related to, and often undistinguishable from, *hypertrophy* in the humbler stages of that process, hyperplasia of the connective tissues. An analogy is offered by the case of hypertrophic cirrhosis of the liver.

Again, chronic inflammation is apt to lose the uniform and characteristic qualities which distinguish the catarrhal, adhesive, and suppurative forms of acute inflammation as described in the first volume (p. 51), and either to become fibrous, like cirrhosis of the lung, liver, or kidneys, or else to become the seat of the tubercle bacillus, and thus assume a caseous form.

Moreover, the continued irritation which gives rise to inflammation and thickening of the mucous membrane of the tongue, the lips, the pylorus, or the rectum may in time, by almost imperceptible stages, pass into a *new growth*, perhaps of the most markedly "heterologous" and malignant kind. Warts and other innocent growths, condylomata and syphilitic nodes also arise from and are complicated with chronic dermatitis and cutaneous hypertrophy.

It is therefore pathologically justifiable to associate with *chronic deep inflammations* of the skin, *hypertrophies* and *new growths*; and this arrangement we propose to follow. The present chapter will deal with chronic deep dermatitis and cutaneous hypertrophy, the following three with the special forms of dermatitis produced by the infections of Syphilis, Tubercle, and Leprosy, and the next with new growths of the skin—innocent and malignant.

The only important instance of *acute deep dermatitis* is that afforded by

erysipelas, which has been already treated as a specific disease in the first volume. The deep and acute inflammations which result from burns and other injuries are best studied in surgical text-books.

GUTTA ROSEA.*—This affection in its more obvious forms is well known beyond professional circles. It provoked the well-known descriptions of Bardolph's face, as "the lanthorn in the poop," "an everlasting bonfire light," "sometimes blue and sometimes red."

Course.—The affection begins with slight erythematous redness, usually of the tip of the nose, occurring after food, and combined with local irritation; the heat and itching are felt by the patient, the redness and even slight swelling are visible. The erythema passes off quickly and perhaps may not return for weeks, but gradually becomes more frequent, until it is at last habitual.

The next step is for the congested vessels to fail to recover themselves in the intervals between the successive states of hyperæmia. What was a recurrent subacute erythema now becomes a chronic congestive dermatitis with exacerbations. Frequently-recurrent œdema has moreover ended in hypertrophy, so that the skin and subcutaneous tissue of the affected part are swollen and thickened. Some of the veins become varicose from habitual distension, and remain visible as red tortuous lines. The sebaceous glands are apt to be obstructed or to suppurate without obstruction, and pustules resembling those of inflamed acne result. Hence the common name "acne rosacea." But there are no precedent comedones, and the distribution, ætiology, and entire natural history of the disease are distinct from those of acne.

Hypertrophy may go on until great pendulous masses of thick skin, with the scars of past pustules and abundant fibrous tissue, form unsightly excrescences upon the nose, growing either from the tip, from the alæ, or from the septum.

Distribution.—By far the most frequent and conspicuous seat of gutta rosea is the nose, but it is not the only one. In persons in whom this feature is characteristically affected, we usually find large red pimples with inflamed base and chronic course upon the cheeks, the chin, and other parts of the face. When the nose is only slightly affected, and the rest of the face decidedly, the general aspect is very different from that of the hypertrophied form above described when confined to the nose, but the anatomical condition is essentially the same, and every gradation between the two forms may be observed. Beyond the face, similar recurrent erythema, with more or less of hypertrophy, may be seen in the lobes of the ears, although here it is very rare to see pimples or pustules. Gutta rosea never affects the shoulders or chest, as true acne does.

Causes and pathology.—Gutta rosea is no less distinctive in its ætiology than in its anatomy and distribution. Like other erythemata, gutta rosea is symptomatic (p. 893); it is never the result of local irritants, but always depends upon reflex inhibition of vaso-motor nerves causing active congestion. We saw that the origin of this reflex action is, in some of the most marked forms of erythema, irritation of the primæ viæ by poisons, drugs, or food (p. 894). Gutta rosea is no exception to this rule; almost

* *Synonyms.*—Acne rosea—Acne rosacea.—*Fr.* Couperose—Acné congestive (Hardy).—*Germ.* Kupferose—Erythema angiectaticum (Auspitz).

always in men, and most frequently in women, it is the result of gastric irritation.

Common notoriety affixes the stigma of drink to the possession of a nose like Bardolph's, but it would be no less unjust than uncharitable to assume this as the necessary cause. No doubt the excessive use of alcohol produces most frequently and most readily the gastric irritation which leads to gutta rosea; but marked examples of the disease may be seen in persons of habitual temperance, and even in total abstainers. In women, especially at the period of the menopause, there is apt to be a form of dyspepsia which leads to flushings of the face, not only after every meal, but in the worst cases upon the first morsel of food reaching the stomach; these flushings are felt by the patient and cause great distress. The frequently recurring hyperæmia leads to habitual congestion, pimples, and at last more or less hypertrophy, although in these cases the type is more often that of diffused redness with dilated venules and pimples scattered over the face than of marked local hypertrophy. This, however, is occasionally seen, just as the diffused pimply redness is often the result of tipping.

The only cause for gutta rosea, beside alcoholic or non-alcoholic dyspepsia, is uterine or rather ovarian disturbance; hence the frequency with which the disease occurs when menstruation is becoming irregular before it finally ceases. Moreover, in many cases of gutta rosea the affection is decidedly worse at the menstrual periods, and is associated with dysmenorrhœa. Considering, however, the great number of cases of menstrual disturbance in which no such effect is produced, and the rarity of gutta rosea even in the worst cases of dysmenorrhœa in young women, as well as the frequency of what may be called climacteric dyspepsia, it seems probable that in most cases gastric irritation is the exciting cause of the disease, and that the monthly exacerbations which undoubtedly occur in certain cases are due rather to direct, physiological, vascular excitation at that time over the whole surface than to morbid irritation of a reflex kind from the ovaries.

Gutta rosea is not produced by the most frequent kind of dyspepsia, that of young adults; it is rare before the age of forty, even in persons who drink freely. It is often combined with gout, but is the result of the dyspepsia which, like the gout, is produced by over-feeding and over-drinking, rather than directly connected with excess of urate of soda in the blood. Gutta rosea is far from being confined to the male sex, though the most typical cases from alcohol are more frequent in men.

Treatment.—The rational treatment of this, as of every other disease, depends upon recognising its pathology and origin. The first indication is to remove the gastric irritation which is almost always present, to discover if possible its cause, whether in excess of food, in imperfect and hasty mastication, or in some particular article which acts as a poison. Salt meat, spices, pickles, melted fats, and sauces—any of these may prove to be the offender; but most often it is wine, beer, or spirits. If we fail to discover the cause of the dyspepsia we may yet do good, apart from regulating the diet and the meals, by the exhibition of small doses of soda with rhubarb and calumba; or when gastralgia is marked, by ten grains of sub-nitrate of bismuth as a powder, taken after meals, to which five grains of carbonate of soda may be added if there is obvious acidity. Gentle laxatives are often desirable, and occasional doses of blue pill. In some cases euonymin is particularly valuable, taken in doses of two or three grains every other night; in others a dinner-pill of rhubarb and nux vomica with

henbane and aloes is found useful. Locally, astringent washes, like Goulard lotion, are useful and pleasant; flexible collodion may also be painted over the congested parts at bedtime; but this should be done when the patient is in retirement, for the closely adherent film is unsightly and difficult to remove. In advanced cases, scarification by innumerable punctures with a lancet is sometimes efficacious; and very successful cases have been reported by Mr Squire and Dr Stowers. When the hypertrophied masses are considerable they can only be removed by the knife.

Pernio.*—Chilblains are examples of chronic and rather deep dermatitis with congestion and œdema. They are the result of frequent local erythematous hyperæmia, and are therefore pathologically allied to gutta rosea. The stimulus is not directly that of cold on the tissue of the skin, as in frostbite; but vaso-motor paralysis causes vascular dilatation, probably after primary contraction of the same arterioles. The itching character of the disorder is produced by the secondary hyperæmia.

The fact that chilblains are most common in childhood and youth probably depends on the greater susceptibility of the vaso-motor nervous system at this period of life. Their prevalence in the winter, and particularly in changeable weather, is explained by the same hypothesis of ischæmia followed by congestion. Their localisation follows from the greater exposure of the peripheral parts to cold; for, as is well known, they are most common on the toes, next on the fingers, and sometimes affect the ears, or even the chin and nose. Moreover, venous stagnation is most apt to occur in the parts most distant from the heart.

The empirical treatment of chilblains is also rational; preserving the extremities from cold by woollen clothing and exercise, avoiding the neighbourhood of the fire indoors, and stimulating the local circulation by friction and rubefacients.

Pathologically similar in the local condition of peripheral anæmia, are the coldness of hands and feet which attends a feeble "circulation," the "dead fingers" to which many persons are liable on exposure to cold air or water, and the "local asphyxia" which was described as the slighter degree of Raynaud's disease (*supra*, p. 600).

EPIDERMIC HYPERTROPHIES.—It was observed by John Hunter that internal pressure produces atrophy, as when a tumour or aneurysm presses upon a vertebra; but that external pressure produces hypertrophy, as in pressure upon the skin of a labourer's hand. It is better put by Paget, that continuous pressure produces absorption and atrophy, intermittent pressure produces hypertrophy.

When pressure is continuously applied, as to a lady's foot in China while still growing, atrophy takes place with only moderate distortion of the bones, and without thickening of the skin; but when it is applied only while walking, as by the narrow-toed and high-heeled shoes of a European lady, there ensues, along with a certain amount of distortion, hypertrophy or thickening of the prominent parts of the skin. This is usually accompanied with a chronic deep dermatitis, whereby the papillæ are affected, and a new growth forms, or occasionally a deep bursa results. These products, in

* "Fiunt etiam ex frigore hiberno ulcera, maxime in pueris, et præcipue pedibus digitisque eorum, nonnunquam etiam in manibus, . . . dolor autem modicus, prurigo major est."—CÆLSUS, lib. v, cap. xxviii, § 6.

which chronic inflammation, hypertrophy, and tumour are seen at their point of junction, we know by the names of corn and bunion.

When, without unnatural pressure or distortion, the hand or foot or any other part is exposed to intermittent pressure, the result is something short of this. It is a pure hypertrophy affecting only the epidermis (*callositas*, *tyloma*). Such is the case in the thickened skin of the ball of the foot and the heel in adults, and of the palm of the hand in all who do manual labour. In children the thickening is but very slight, probably an inherited character, since we find it in all plantigrade animals. In adults the degree of it varies with the habits of the individual. This most purely physiological form is seen in those races who go barefoot, for wherever shoes are worn there is a chance of corns appearing even on the sole of the foot. Precisely similar callosities appear in the middle of the palm in workmen who use screwdrivers, gimlets, and augers, in the cleft between the finger and the thumb in shoemakers, and others who habitually pass a strap or cord in this position, over the patella in those who frequently kneel, and on the back of the neck, especially over the seventh vertebra, in those who carry burdens on their shoulders, as may be often observed in railway porters.

In such a callosity it will be found on section that the horny layer or cuticle is enormously thickened, the Malpighian layer slightly, if at all, and the cutis vera quite unaffected. Hence these callosities appear lighter than the rest of the skin in negroes.

*Tylosis** was the name given by Hebra to great epidermic thickening, without inflammation. It usually affects the palms and soles. A marked case is described and figured by Dr Crocker in the 'British Journal of Dermatology' (vol. iii, p. 109). This was an instance of tylosis following over-secretion of sweat, but more often the condition is a congenital exaggeration of the natural thickness of the palmar and plantar epidermis. In a case reported in the same article, blisters formed every autumn, perhaps from retained sweat; and the patient could trace the same malformation in his mother and grandfather, in three of his own children, and in a grandchild.

In other cases tylosis (or keratosis) of the palms and soles is the result of arsenic;† and may be either diffuse or discrete. In the latter form, the corns are found also on the back of the hands, the fingers, and the toes.

The corn (*clavus*, *cor*, *Hühnerauge*), as was first shown by the anatomical researches of Gustav Simon, consists of a diseased growth of the horny cuticle into the subjacent living Malpighian and papillary layers. The horny downgrowth is of a more or less conical shape, and causes atrophy of the immediately adjacent papillæ, but at the same time a thickened layer of cutis forms around by true chronic inflammation. Here the cuticle is but slightly thickened, and not hard, as in the central part, and the papillæ become gradually hypertrophied. Occasionally the original central hardening appears never to take place, especially in the soft parts of the skin between the toes, which are continually in contact and moistened with per-

* *Tylosis palmæ manus plana* (Hebra); *Keratosis manuum aut pedum*, *Ichthyosis palmaris* (Auspitz); *Callositas*. *Τύλωμα* and *τύλωσις* are both genuine Greek derivatives of *τύλος* or *τύλη*, from *τυλόω*, to make callous. So Theocritus, *μακέλα τετυλωμένος ἔνδοθι χεῖρας*, i. e. hands made hard on the palms by using a pickaxe.

† This interesting fact seems to have been first observed by E. Wilson in 1873, but its general recognition is certainly due to Mr Hutchinson ('Path. Trans.,' 1888). See cases by Brooke, Crocker and Pringle, with plates ('Brit. Journ. Derm.,' 1891); Rasch and Payne (*ibid.*, 1895); Mackenzie (1896), and Nielsen's article in the Sydenham Society's volume for 1900, with references.

spiration. The result is what is known as a "soft corn." There may be a mere horny plug pressing on the skin beneath, without exciting inflammation around, as occurs most frequently on the naturally thickened skin of the ball of the great toe or heel. In this case the resulting pain is that of an occasional sharp prick, when the pointed, hard, horny plug is suddenly driven home by accidental pressure, and is very different from the continual, wearing, and disabling pain of a *clavus mollis*. The commonest kind of corn, partaking of both characters, combines the discomfort of each.

When a cyst or bursa forms beneath the corn and increases so as to become obvious it is called a *bunion*. A small cyst is often to be found beneath an ordinary corn of old standing and large dimensions, but the large cysts seldom form except over the metatarso-phalangeal joint of the great toe, when this has been rendered artificially prominent by the distortion of short, narrow-toed, and high-heeled shoes. The bursa from time to time inflames, and the tension then occasions severe pain, although suppuration is rare.

Treatment.—The proper treatment of corns is prevention. Children's shoes should be made low in the heel, broad in the tread, straight on the inner side, and the right and left shoe not directly but conversely similar. In being measured for shoes one should not sit but stand, so that the weight of the body may expand the foot into the natural shape and size which it then assumes. In a perfect covering for the foot similar expansion is afforded by the elasticity of the upper leather, and the yielding of a thick and soft stocking, but the sole should slightly project, so as to equal the largest length and width of the foot. Even in adult life the trouble of insisting upon boots being properly made is well repaid by the increased comfort and ability to walk, and the disappearance of acquired corns and distortions.

In bad cases of bunion it is well for the patient to wear stockings with divided toes like a glove, and to have a stout vertical piece of leather fixed so as to separate the great toe from the rest, and to press it outwards into its natural position—a plan devised by Aston Key.

Besides removing the thickened epidermis and extracting the conical plug of hard keratin from time to time, relief may be obtained by treatment with salicylic acid, either 2 per cent., mixed with mutton suet (the ointment in use in the German army), or in the stronger proportion of five or ten grains to the ounce of vaseline.

In many cases, however, beside the presence of both hard and soft corns, the whole foot is tender and painful. The remedy then consists, first, in large and low shoes, so as to diminish the heat and moisture; secondly, in thick and loosely knitted stockings, which are at once absorbent and pervious; and, thirdly, in soaking the foot night and morning in alum lotions, or brine, or solution of tannin. Thread and cotton coverings for the foot should never be worn; when wool or merino-mixture cannot be borne, silk is the only proper substitute.

To prevent blisters and excoriations thick woollen stockings should be well soaped within. But in hard marching it has been found by the experience of soldiers that a better plan is to bandage the feet with rolls of linen well oiled or soaped, a foot covering which never wears into holes, which is easily washed, and which fits every foot.

Until comparatively lately the shoes supplied to the English army were symmetrical—that is to say, there was no difference between right and left.

This is now happily corrected, owing to the efforts of Dr Parkes and other medical reformers; at present our soldiers are probably better shod than the French with their shoes and gaiters, or the Prussians with their high boots. The best foot covering of all is, perhaps, a kind of sandal worn by the Spanish infantry. In the handsome and serviceable costume of the Hungarian army (now, unfortunately, altered) an excellent laced boot is worn, much like that of our own troops, but somewhat higher, like a shooting boot, without the addition of a leather legging. The importance of anatomical knowledge in clothing is conspicuous in an army; but in civil life also, apart from artistic considerations, the misery and ill-temper produced by ill-fitting shoes render the subject one of serious importance.

Tylosis is best treated by soaking the parts in hot water, with the use of alkaline soap. Or the hardened skin may be covered with salicylic acid as an ointment or plaster. The best method of applying this valuable keratolytic agent is probably that introduced by Unna, as a plaster made by saturating chloroform solution of gutta percha with salicylic acid (see Dr Thin's paper, with four cases so treated, 'Clin. Trans.,' xvii, 9). But the writer has seen excellent results from an ointment composed of ac. salic. ʒss to an ounce of vaseline or lanolin.

Leucoplakia buccalis.*—Closely allied anatomically to corns and callosities, consisting, like them, in hypertrophy of epithelium, are the milk-white patches or corns upon the prominent parts of the heart, both auricle and ventricle, and the thick gristle-like white fibrous patches on the surface of the spleen, with similar conditions less frequently met with in the pleura and the peritoneum.

Still more closely connected with corns are the white patches upon the mucous membrane of the tongue and inner lining of the cheeks. These patches have been erroneously described as "psoriasis of the tongue" and "ichthyosis." They are of much diagnostic interest. They sometimes occur as the result of irritation from a rough tooth. They also are produced, or at least aggravated, by smoking, not by the chemical action of nicotine, but by the heat of a cigar or pipe or by the friction. Very similar patches may be the result of syphilis, but these may generally be recognised by their being unsymmetrical, and not confined to the mucous membrane but dipping beneath it; moreover in most cases there is either an ulcer on the patch, or more or less contraction around it from previous loss of substance. The diagnosis from syphilis, however, is sometimes difficult. Such patches of leucoma are not unfrequently met with in cases of lichen planus (p. 863).

These white patches may be the seat of subsequent cancer; they are not its first stage, for they may last many years before malignant action appears, but they are the seat of irritative proliferation of cells, which only needs the determining condition, whatever it may be, to produce carcinoma.†

The treatment is, first, to remove any source of irritation, as rough teeth, smoking, or the use of pepper, hot soups, and, perhaps, carbonic acid in water; secondly, to apply such local remedies as borax and honey, or chlorate of potash. Balsam of Peru has been recommended by Dr

* *Synonyms*.—Ichthyosis linguæ—Psoriasis buccal (Bazin)—Psoriasis linguæ—Leucoma—Tyloma oris—Keratosi linguæ et oris—Leuco-kératose buccale (Besnier).

† See the discussion on this affection at the International Medical Congress, 1881 (vol. iii, p. 171), introduced by the late Dr Schwimmer, of Buda-Pesth, by whom the term *Leukoplakia buccalis*, now generally accepted, was introduced.

Rosenberg ('Therapeutische Monatsheft,' 1888, No. 10), applied two or three times a day.

WARTS.—*Verrucæ—papillomata—verruæ—Warzen*.—These are small cutaneous tumours consisting in overgrowth of the papillæ of the cutis.

A vertical section shows that the horny layer of epidermis is unaffected or is somewhat thinner than usual. The Malpighian layer is sometimes slightly thickened, and in many cases is the seat of more abundant pigment than usual. There is seldom or never any evidence of inflammation, the process is one of hypertrophy and new growth.

Warts are very rarely painful, but their removal is desired because of their unsightliness and inconvenience, or because of the pain occasioned when they are accidentally pressed upon. They are sometimes single, more often multiple, and in rare instances occur in innumerable multitudes. They appear to be never congenital, but are most common in children; they are comparatively rare after early adult life, but may reappear as pigmented warts in old age. We can sometimes trace their origin to certain definite sources, usually some form of local irritation.

The most common seat of warts is on the hands—not the palm, but the fingers, the dorsum, and the wrist. They may also occur on the arms, the face, not unfrequently on the scalp, and more rarely on the trunk or lower extremities. They are decidedly rare on the feet, but are not uncommon on the penis and vulva, around the anus, at the orifice of the lips and on the mucous membrane of the mouth. A similar condition occurs also in the œsophagus, especially in certain cases where pressure has produced irritation of the mucous membrane, and also where chronic cardiac disease has led to its habitual congestion. Warts are usually of a rounded, hemispherical, or pointed shape, but sometimes are flat at the surface; and by growth or coalescence a large flat warty mass may be formed, which is called a *condyloma*.

Pathologically we may recognise the following varieties of papillomata:

1. The innocent and painless warts of youth, easily removed, and not recurrent. They are almost always found on the hands. When one of them appears, others quickly follow, and their prevalence among children of the same age has led to the popular belief that they are contagious. Dr Payne has recorded a case in which he was himself the recipient of the contagion ('Brit. Journ. Derm.,' vol. iii, p. 185). If warts are caused by bacteria, none has yet been identified.

These multiple warts disappear as readily as they come, with the help of a charm or without. They may, however, be removed by salicylic acid dissolved in collodion or gutta percha, or by frequent applications of nitric or strong acetic acid.

2. Small multiple warts, usually flatter, and of a pinkish colour, thus differing from the yellowish tint of those first described. Beside their size and colour, they are distinguished by occurring in large numbers so as to simulate papular dermatitis. Again, if rather large, discrete, and somewhat flat they may simulate molluscum, a variety of which has been named "*verrucosum*" from this resemblance. These multiple warts are usually seen covering the arms, but may also be met with on the neck, face, and forehead. In one of the writer's patients, a girl of eighteen, they covered the back of both hands; in another, a healthy woman of twenty-eight, they closely resembled lichen planus of the wrist; in a third, a young man of

twenty, they occupied the neck and left side of the nose, where we counted more than three dozen. They were also present, though less numerous, on both hands and forearms. All appeared in six months, starting with one large one on the pomum Adami. This patient had had warts on his thumbs when a boy.

3. The warts of old age (*Verruca senilis pigmentosa*), usually few in number, large and deeply pigmented. They are apt to occur around the orifices of the body, on the eyelids, the lips, the genitals, and around the anus. Similar papillomata occur on the tongue and mucous membrane of the mouth. They are very liable to degeneration, and often become the seat of rodent ulcer or more ordinary epithelial cancer, after having existed for months or years without showing a sign of malignancy. The abdomen of an old woman under the writer's care suffering from internal cancer was covered with these pigmented warts, but they were themselves non-cancerous (Mary Ward, March, 1886).*

4. Warts following gonorrhœa: multiple and confined to the glans and skin of the penis.

Condylomata and mucous patches.—The composite warts, known as condylomata, hard condylomata, Spitzcondylom (*C. acutum*), are true papillomata in structure, but are always local, never scattered about as other warts are. They occur most frequently about the anus and genital organs. They are certainly not always syphilitic; and they may follow the irritation caused by the discharge of a soft chancre or a gonorrhœa, in the latter case being identical in all but size with gonorrhœal warts. They may also occur in the cleft of the nates as the result of friction from riding, when there is not the least probability of other than mechanical origin.

The latter variety only needs dryness and protection, with the help of citrine or some other mild mercurial ointment. The harder venereal warts and condylomata must be removed by curved scissors, or with nitric acid, arsenical paste, or some other caustic application.

On the other hand, the soft condylomata (*plaques muqueuses*, or mucous patches) are believed to be always syphilitic, and they almost alone of secondary lesions have the power of transmitting the virus. They occur on the lips, on the mucous membrane of the tongue, cheeks, palate, and tonsils, occasionally on the eyelid, sometimes on the female mamma, and frequently around the anus and vulva. Here they may grow to great hypertrophic masses, the tertiary *syphilis vegetans* of authors. They are best treated by dusting with calomel, and occasional application of nitrate of silver.

ICHTHYOSIS.—This is a very remarkable and in its fully developed form a rare affection. It is an example rather of hypertrophy and malformation than of chronic inflammation. It was classed by Willan among his squamæ, and its name, "the fish-skin disease," was given for the same reason. The scales of ichthyosis are, however, very different from the branny desquamation which is the last stage of eczema (p. 825), from the large pearly coherent scales of psoriasis, and from the thin squames of pityriasis rubra. In the most marked cases the surface of the skin rather resembles the rough, dark, and scaly surface of the bark of a tree, or it may be compared to the rugged hide of an elephant; and sometimes the horny excrescences make it feel like the prickly skin of certain sharks; indeed, persons affected with an

* A singular case of multiple pigmented warts occupying one half of the body in a girl of seventeen came under the writer's notice in 1883 and 1887, and is briefly recorded in the 'Guy's Hosp. Rep.,' vol. xlv, p. 408.

extreme degree of ichthyosis have been exhibited as "porcupine men" (*Ichthyosis hystrix*).

The malformation is probably always congenital, but does not become obvious until infancy is past, although the mother will generally admit that the infant's skin was from the first more rough, dry, and hard than that of other children.

Ichthyosis is most marked upon the limbs, but its characteristic feature is that it is almost universal. In a fully developed case no portion of the whole body is absolutely healthy. The parts least affected are the scalp, face, palms, soles, genital organs, and the flexures of the joints; in other words, the thinnest portions of the skin. The greatest accumulation of horny epidermis is on the outer side of the arms and legs, and especially about the elbows and knees; but the back, the nates, and the whole of the trunk are often scarcely less affected. The scales are not large and are more adherent than those of psoriasis, so that, considering the thickness and extent of the disease, there is less free desquamation than might be expected. The surface is dry as well as rough; there is almost complete absence of perspiration; the sebaceous glands, instead of their natural lubricating oil, secrete a thick material (*seborrhœa sicca*) which helps to form the bulk of the crusts, and gives them more power of attracting and retaining dirt. The difficulty of keeping the rough scaly skin clean is extreme, so that children affected with it have a dingy appearance, which in some cases and in the worst parts becomes almost black. The name "ichthyosis nigra" has been very unnecessarily applied to this condition; for there is no deposition of pigment, and mere friction will sometimes rub off the superficial dirty scales from the most exposed parts, and leave a grey abraded surface which is characteristic and hideous enough. The thick dry skin is apt to crack in a somewhat regular square fashion like crocodile-skin or the horny plates of certain kinds of armadillo (*I. scutulée*), and these cracks may penetrate to the cutis and become painful bleeding rhagades.

Except for this accident, ichthyosis is painless, and apparently does not affect the general health. One or two children under the writer's care affected with it have been remarkably plump, rosy, and in other respects well developed and healthy.

As above remarked, ichthyosis is a congenital disease or rather malformation, and it is frequently seen in families, as in the famous case of John and Richard Lambert, two brothers who were exhibited as the porcupine men, and whose father had a similar state of skin.

Among 15 patients reported by the writer in the 'Guy's Hospital Reports' (vol. xlvi, p. 389) eleven were under eighteen, and the rest aged between twenty-one and thirty. One was a case of ichthyosis or tylosis of the palms, and was present in a brother and sister of the patient although not in his parents.

Histology.—On vertical section the diseased masses are seen to consist of wavy layers of horny scales exactly like those of the thicker parts of the cuticle. A section of the skin shows that the Malpighian layer of epidermis is proportionately small, and that the ridge-and-furrow cells ("prickle-cells") have more or less completely disappeared; in other words, the keratinous transformation of epithelium is here more rapid than usual. The cutis is unaffected, except the papillæ, which are elongated; but this is a secondary change—they are really thinned out and not hypertrophied. According to Esoff the sweat-glands have disappeared or only exist as cysts; but this

appears to be only exceptional. The sebaceous glands and the hair-sacs are unaffected, but the hairs are atrophied, tufted at the root, and easily shed.

Erasmus Wilson's "false ichthyosis," the *ichthyosis sebacea* of other authors, is seborrhœa sicca corporis. In true ichthyosis there is no doubt a certain amount of sebum, which is mixed with the epidermic masses, and can be extracted by ether in the form of stearine and cholesterine; but this is not the essential part of the disease.

Nor is there any need to continue the distinctions of Devergie and other writers into *Ichthyosis alba*, *I. brunnea vel nigra*, and *I. hystrix*; or Alibert's of "Ichthyose nacrée" and "I. cornée."

In certain cases of ichthyosis hystrix, however, there are papillomata mingled with the epidermic lesions (*I. verrucosa*), and this makes a real distinction. In a case of this kind, a boy in Philip Ward in 1887, the horny warts were arranged in long stripes down his arms and legs, while the scalp, the palms, and the soles were alone free. In some of these cases the stripes follow more or less closely the nerve-trunks of the limb, and this has (perhaps too hastily) been admitted as a proof of a nervous origin of the lesion.

Xerodermia.—We must recognise as true ichthyosis, though of a much milder form, that affection of the skin which was named by Wilson "xeroderma."* This dryness of the skin is accompanied by roughness, to be felt rather than seen, chiefly on the outer side of the arms and legs. There is but little desquamation, and the morbid change is so slight that it is difficult to believe it can be essentially the same as that which produced the porcupine men. But of this there is no doubt, for we meet with every gradation between the two conditions. On the one hand, such an extreme degree of the affection as the *ichthyosis hystrix* of Tilesius is extremely rare; and, on the other, even the slighter forms of xerodermia are more extensive, obstinate, and clinically important than they at first sight appear. At the same time we may admit two groups of the affection; the more severe, which corresponds with the classical description of ichthyosis, and the milder forms, for which the term xerodermia might be used if it had not since been unluckily "conveyed" to a totally different and malignant form of cutaneous disease (*v. infra*, p. 1008). Each case has its own characters from an early period, and when once established in the second or third year of life, does not usually become much worse. Both alike are congenital malformations, both have the same distribution and probably the same histology.

The chief importance of this remarkable disease, even in its mildest form, and quite apart from the hideous deformity of the worst kinds, is that the dry, harsh, unlubricated skin is extremely disposed to superficial dermatitis; or, as it is usually put, ichthyosis and xerodermia are often complicated by eczema.

Treatment.—The first indication is to cure the inflamed, red or weeping patches, and the deep painful fissures by the same methods which have been above described for the treatment of eczema rubrum, madidans, and rimosum. The second indication is to supply the deficient natural lubricant of the skin by oils or ointment; suppleness is thus restored, the characteristic dryness is removed, and the liability to dermatitis reduced to normal limits. In the more severe cases, however, it is necessary, before this can

* In this, as in other similar compounds, the name of the disease, the condition of the derma, should be spelt with *i*. So sclerodermia, pachydermia, etc., words analogous in formation to anæmia and anuria.

be done, to remove the products of disease; and for this purpose warm baths, alkaline baths, friction with soap and water, and particularly with soft soap, are the measures which are necessary. The only caution is not to be too vigorous in softening and removing the diseased epidermis until local inflammation has been relieved. From time to time the process of cure may have to be interrupted, and the tender skin soothed by lead or boric acid ointments or by olive oil. Dr Fagge, as also Dr Liveing, recommend glycerine of starch, but often oil is more soothing than glycerine in any form.

It is astonishing what excellent results may be obtained, even in the worst cases of ichthyosis, when treated with perseverance and with an intelligent appreciation of the object in view. Within a few weeks children, whose portraits would almost go side by side with that of the porcupine men, present an appearance which it requires the scrutiny of an experienced eye to recognise as more than "a little roughness of the skin."

Salicylic acid may be employed in obstinate cases, and "ichthyol" (a mineral oil obtained from fossiliferous rocks) has been strongly recommended for this, as for most other cutaneous diseases.

The disease, however, is relieved, not cured. As soon as the patient is neglected it returns as before, and he can only maintain his skin in bearable condition by constant attention to cleanliness, by frequent warm baths, and continual inunction. Internally antimonial wine was recommended by Dr Fagge ('Guy's Hosp. Rep.,' 1870), and many physicians administer cod-liver oil.

The term *ichthyosis congenita* has been applied to a rare and remarkable form of disease described by Lebert, in 1864, as *keratosis diffusa intra-uterina*. It affects the whole of the skin with thickening of the epidermis, which is too small for the body, so that the child is literally hide-bound. Numerous and deep fissures result, and the appearance which ensues has been described as the "harlequin foetus." Such children die before birth or only survive it a day or two.

The horny layer is greatly thickened, the papillæ and the rest of the cutis unaffected, the sebaceous glands are atrophied, and the ducts of the sweat-glands much dilated.

Cases of this curious and very rare affection have been described by several authors. A good account of it is given by Hans von Hebra in his 'Krankhafte Veränderungen der Haut,' p. 348; and by Thibierge in the 'Dict. Encycl. des Sc. Méd.' Mr Bland-Sutton, who believes that it consists essentially in a perverted secretion of the vernix caseosa, has figured a case in a foetal calf in the forty-second volume of the 'Pathological Transactions.'

CORNU CUTANEUM (*ichthyosis cornea* of Willan and Bateman*) is the name applied to those remarkable cases of horny growth which have been figured as "freaks of nature." They are occasionally seen in old women, less often in old men, and very rarely indeed in early life.

Lebert collected 109 cases. Most often they spring from a sebaceous cyst. They may occur anywhere, on the lip or the glans penis, and sometimes are followed by cancer.

* Bateman objected to calling them horns on the ground that they have no connection with the bones or other part beneath, and are of purely cuticular growth. But this is the only ground on which we call them true horns and not exostoses or antlers. What he meant was that they have no bony core as the horns of ruminants; but they are exactly identical in structure with that of the rhinoceros.

The growth can always be completely removed, and shows no tendency to return; although, as Bateman remarks, if merely sawn or broken off, it invariably sprouts again, like hair or nails.

Two remarkable cases of cutaneous horns, one on the neck and the other on the hand, were modelled by the late Mr Towne for the Guy's Hospital Museum (Nos. 333 to 339).

Hyperkeratosis centrifuga.—Under this title or as *Porokeratosis* (to which adjectives descriptive of its supposed origin in the sweat-pores, *ποροι*, or of its excentric spread, or of its tendency to atrophy have been added), Mibelli described a curious affection in 1893, which is distinct from Ichthyosis (even *I. linearis neuropathica* or *I. hystrix* in stripes, p. 959), from any variety of Lichen, from other keratoses, and from *Porokeratosis scutularis* of Unna. The little horny growths appear on the back of the hands, and the face, and also on the palms, the legs, and feet. As they spread the centre becomes depressed, and they at last become confluent, and "figured" or outlined. The process begins in the mouths of the sweat-ducts and the hair-sacs. No signs of inflammation or of bacterial origin have been discovered. But the most remarkable feature of the affection is that it has a family distribution and is hereditary.*

SCLERODERMIA.†—This is a rare but interesting disease. Pathologically, it is a chronic, deep induration and thickening of the skin, followed by atrophy, and often ending in complete involution.

One of Dr Fagge's best contributions to dermatology was his masterly account of this disease in the 'Guy's Hospital Reports' for 1867, in which he conclusively proved the essential identity of the diffused sclerodermia of authors with the circumscribed sclerodermia which was also known as *Addison's keloid*, and is synonymous with many cases described as *morphœa* by older writers. In a second paper he showed that cases of sclerodermia may undergo a gradual process of recovery (*ibid.*, vol. xv, p. 297).

Course.—The disease begins very gradually in a hardening of the deeper layers of the skin. The epidermis is unaffected, the surface smooth and not elevated, the colour unaltered; but the patient finds that the part is stiff, and on feeling it a more or less marked induration is easily recognised. The skin cannot be pinched up into folds as in health, and instead of the natural elasticity there is a characteristic hardness.

In the circumscribed form the edges are well defined, so that it feels as if a disc of hard, smooth leather were let into the skin. In the diffused form the stiffness and induration become gradually less and less, until they are lost in the natural softness of the skin; but even then one may generally find some directions in which the sclerosed patch has a more definite edge. Sooner or later the local appearances become more marked; the affected skin turns white, or assumes a sallow tint, or becomes pigmented with a pale yellowish brown, which is usually most marked towards the borders, and is never uniformly diffused over the entire patch. In the early stage

* The writer has not seen a case, and takes these facts from an excellent article—with histological drawings, bibliography, and genealogical tree of eleven cases—by Dr Gilchrist, of the Johns Hopkins University, 'Journ. Cut. and Gen.-ur. Dis.,' April, 1899.

† *Synonyms*.—Sclérémie (Alibert)—Sclèrème des adults (Thirial)—including "Addison's keloid" and Morphœa. Vitiligo (in part), *Cutis tensa*. Morphœa, or Morphea, or Morfea, a Low Latin word of uncertain derivation, corrupted into *morpheus*, was very widely applied in popular usage, but in Holland's translation of Pliny is used as a translation of *vitiligo*.

a slight rosy circle may be observed around the patch, occasionally forming a distinct "lilac ring" in the circumscribed form (*morphœa*), or a more ill-defined and irregular blush in the diffused (*scleriosis*). The smooth white patch, with its colour heightened by the pink margin, has been often compared to an ivory disc. There is no œdema of the integument at any period, and this alone suffices to distinguish sclerodermia from pachydermia (*elephantiasis*), with which Rasmussen once associated it.

A patch of morphœa may go on increasing until a disc several inches in diameter is formed, or it may lose its distinctive characters and pass into the diffused variety.

Diffused scleriosis usually has its own characters from the beginning, and slowly extends, with no definite margin, until it involves a considerable part of one limb, or one side of the neck, or half the trunk. The surface is then as hard as a board, and as unyielding to the touch. After a time contraction begins to appear, and scar-like bands vary the surface of the disease. This, together with increasing pigmentation, gives it some resemblance to the contracted cicatrices from a scald or burn, and explains Addison's application of the term keloid to the affection.

A rare form of disease, described as guttate Sclerodermia, is associated with nævus-like spots of dilated vessels. (See Dr Pringle's case, 'Clin. Trans.,' vol. xix, p. 313; and Dr Perry's, 'Brit. Journ. Derm.,' vol. x, p. 54.)

Locality.—A patch of morphœa most often develops on the trunk, particularly on the skin of the female mamma, where such parchment-like *plaques*, or ivory-indurations, like the skin frozen by an ether spray, have been sometimes called "vitiligo."

Diffused sclerodermia may be seen on the scalp, the forehead, the chin, or other parts of the face; and the expressionless mask-like aspect it gives to the features is very striking, particularly since the immobility is not uniform, but affects one side or certain features only. Scleriosis is also frequent in the arms, hands, and fingers, which become contracted and useless,* and on the side of the neck, where a distortion may be produced which resembles torticollis; or it may invade extensive regions of the trunk or lower extremities. In the well-marked case of a young and healthy soldier, reported by Dr Curran ('Edin. Med. Journ.,' 1871), the disease covered the whole surface of the body. It has been unsymmetrical in all the cases seen by the present writer, and in the numerous drawings, as well as models, in the museum of Guy's Hospital. But in a case described by Dr Van Harlingen in a negro (1873) the disease was symmetrical, and he regards this as the rule. Occasionally, but only as an exception, its progress may be traced in the course of a cutaneous nerve.

Cases have been described in which sclerodermia or a similar affection involves the mucous membrane of the mouth.

Symptoms.—Sometimes patients complain of considerable pain as well as stiffness in the affected parts, but this is often completely absent. There is little or no itching, and, as a rule, no accompanying inflammation or pyrexia. Sometimes, however, deep and very intractable circumscribed ulcers form on the sclerotic patches, as in a remarkable case brought by Mr Marrant Baker before the Pathological Society (vol. xxxii, p. 261). There

* Sclerodermia of the fingers has been named "*Sclérodactylie*" by Ball, Hallopeau, and other French writers; this local form is particularly liable to ulceration or even gangrene. In some cases, beside these features, cyanosis of the extremities approaches the condition described by Raynaud, or Sclerodermia and Raynaud's disease may co-exist.

is no hyperæsthesia, nor true anæsthesia—at least, as a rule; but patients may complain that they do not feel so distinctly as on the normal skin. The hide-bound state of the affected parts makes them almost immoveable.

Histology.—There is scanty evidence of a true inflammatory process in this singular disease, nor does there seem to be anything which can fairly be called a new growth; the epidermis is unaffected, the papillæ are atrophied only in the later stages of the affection, the hair-sacs and sebaceous sweat glands throughout are normal, as also are the unstriped muscles of the cutis. The seat is primarily in the deeper layer of the cutis and the subcutaneous tissue. Here the fibrous bundles become thicker and the fat between the meshes is absorbed, while increased pigment is gradually deposited both in the papillæ and in the cutis. No cell proliferation was detected by Chiari or by later observers; but the arteries are found much thickened, with the lumen diminished. The process of mingled hypertrophy and atrophy leads to the characteristic results, both of the earlier and later stages of the disease. In the earlier stage, by compressing the blood-vessels the peculiar pallor is produced; and, by the increase of fibrous tissue and disappearance of fat, the scleriosis of the later stages. Beside the pigmentation, the affected parts sink below the level of the healthy skin, instead of being, as at first, on the same level, and the contraction leads to the cicatrix-like bands which crumple the fingers and deform the face or breast.*

Etiology.—The true cause of sclerodermia is quite unknown. It is more common in women than in men. Putting together 22 cases reported from Guy's Hospital (seven by Addison, three by Fagge, and twelve by the present writer, 'Reports' for 1867, 1870, and 1889), nineteen of the patients were women and only three men. In 40 cases collected by Rasmussen the numbers were thirty women to ten men. It has been observed at all ages, including children under six and adults up to seventy. Among our twenty-two patients, ten were between eight and twenty years old, six between twenty-five and forty, and six between fifty-five and sixty-four.

Prognosis.—Dr Fagge made the remarkable discovery that sclerodermia, both in its circumscribed and diffused forms, is liable to spontaneous involution. He tracked one of the most marked cases described by Addison, and found that the patient's skin had recovered its normal condition. The same thing has been repeatedly observed since, although it is too much to say that complete recovery is an invariable or even a frequent result. The disease, at all events, shows no tendency to develop into any active or malignant form, and beyond the disfigurement and disablement due to contractions and the occasional pain, the most serious result is the occasional supervention of ulcers as above noticed.

No efficient *treatment* has been devised. Emollient oils, warm douches, and massage have been tried with some apparent benefit. Electricity has also been employed, either in the form of continuous galvanism to the affected patches, or by interrupted galvanism to the neck in the somewhat vague hope of stimulating the cervical sympathetic, and the equally vague expectation that occasional stimulation of the cervical sympathetic would have any effect upon the disease.

Sclerema neonatorum.—This affection, sometimes called Thirial's dis-

* See the valuable paper on "Sclerodermia" by Dr Rasmussen, of Copenhagen ('Edin. Med. Journ.,' Sept., 1867); and another by Dr Van Harlingen, of Philadelphia, with a full list of references ('American Journ. of Syphilis and Dermatology,' October, 1873). See also an account of the histology of Morphea by Dr Crocker, 'Path. Trans.,' 1880.

ease, is best named as he called it, *sclerème*, in distinction from the sclerodermia or scleriosis just described. It is the condition which is known as "hide-bound" in new-born children, affecting the whole of the surface, and characterised not only by hardness, want of elasticity, and pallor, but also by œdema. The temperature is lowered and the child generally dies within a fortnight.

Linear atrophy.—Somewhat resembling sclerodermia in appearance, and perhaps also in pathology, is a curious affection of the skin, which takes the form either of long streaks, generally broader in the middle than at the ends, or less frequently of round, more or less regular patches; in both cases it appears like a scar, for there is loss of pigment and atrophy of the cutis vera. It was first described by Wilks ('Guy's Hosp. Reports,' 3rd series, vol. vii, p. 298) as an idiopathic affection which exactly resembles the cicatricial marks caused by overstretching of the skin and rupture of its deeper layers—well known under the name of *lineæ gravidarum* as a result of abdominal distension from pregnancy, but also seen in ascites or whenever the abdominal skin is similarly stretched, and over joints which have enlarged and stretched the skin. The spots are palpably depressed below the level of the healthy surface, and—microscopically—the papillæ are found atrophied or vanished, the epidermis in both its layers thinned, and the subcutaneous tissue and glands atrophied. This curious affection, which may be idiopathic, has been seen upon the hips, the leg, the knee, the ankle, and the hand. In the early stage the marks are somewhat pink, but there are no signs of inflammation, no pain, or any other symptoms. In a case described by Dr Liveing the maculæ were at first slightly red and raised above the skin; they occupied the upper part of the sternum and neck, and after passing into the atrophic stage, underwent gradual involution.

In a girl who lay ill with renal dropsy in Miriam Ward in 1886, there was during her illness and after recovery a remarkable zebra-like development of atrophic stripes on the forearms, loins, and hips. A still more extensive case of the same kind has since occurred in a youth of nineteen, who recovered after more than a year's illness from tubal nephritis with extreme anasarca and ascites.

There are some good models of these *striæ atrophicæ* in the Guy's Hospital Museum, Nos. 340—347.

ELEPHANTIASIS.*—This, among many other names, has been given to a curious form of chronic inflammation with hypertrophy of the skin, chiefly met with in tropical climates. It is not necessary to enter the tangled labyrinth in which this, like so many other names of cutaneous diseases, is involved. It will suffice to say that the Greek word elephantiasis was used by Aretæus and by Celsus for the very different disease described by the Evangelist Luke as lepra, and throughout the Middle Ages as leprosy. Thus *Elephantiasis Græcorum* was the technical term for leprosy down to very recent times. The allusion was to the magnitude and monstrosity of the disease (*cf. infra*, p. 991, *note*); but unfortunately the comparison was supposed to be with an elephant's hide, and since the legs affected with pachydermia have some resemblance to the thick and shapeless limbs of an elephant, the two diseases and their names were long hopelessly confused.

* *Synonyms*.—Elephantiasis Arabum or Elephas—Bucnemia or Bouknemia—Arabic, Dal fyl—Pachydermia—Barbadoes leg.

The most important pathological fact about elephantiasis, using the term as applied by the Arabian translators of the Greek authors, is that it is hypertrophy dependent upon recurrent deep dermatitis, which we may compare to that of gutta rosea and of pernio. All observers in countries where the disease is endemic agree that it begins and is accompanied by recurrent attacks of what has been called erysipelas, each attack leaving the tissues more thickened and infiltrated. Inflammatory oedema of the skin and subcutaneous tissue is the characteristic lesion. This gradually becomes *oedema durum*, and no longer yields to pressure, the infiltrated tissues undergo hypertrophy, and masses of fibrous tissue are thus produced, which may be described as a diffused new growth. The skin itself appears at first to be unaffected, at least in its papillary and epidermic layers; but after a time it also hypertrophies, the papillæ becoming enlarged and the surface coarse, thick, scaly, and pigmented.

Histology.—On section, the hypertrophy of the deep layer of the cutis, and the massive fibrous bands of white and elastic tissue, with oedematous connective and adipose tissue, are very characteristic; the lymph-spaces of the cutis are enlarged, and the lymphatic vessels are frequently found dilated and varicose. Occasionally an ulcer will accidentally open one of these enlarged lymphatics; and a discharge of normal lymph, more or less milky if it has passed through several lymph-glands, is poured out.

The disease does not spread to the deeper fasciæ or bones, and it never affects internal organs or leads to any but local results.

Such a condition is occasionally seen as the result of long-continued inflammatory dropsy of one limb. An example in a case of old dermatitis of originally syphilitic origin is figured in the 'Guy's Hospital Reports' for 1877, pl. ii. A similar result may also be seen in cases of enormous obesity and general hypertrophy of fat and subcutaneous tissues. Or, again, it may be the result of local pressure upon the veins and lymphatics, as by enlarged inguinal glands or other tumours. But in many hot countries, particularly the West India Islands, the Cape Colony, Egypt, South America, China and Japan, and also in the Pacific Islands—elephantiasis is idiopathic and endemic. Dr Turner, of Samoa, sent the writer photographs of this disease, which presents exactly the same features there as in the other races and climates where it is found.

The *distribution* of elephantiasis is almost limited to the legs and scrotum. Sometimes only one foot is affected, sometimes the thigh remains free. One leg may entirely escape while the other forms a huge tumour, and the scrotum may be diseased independently or along with the legs. Lymph-scrotum is the name given to elephantiasis scroti (p. 715). The size of these scrotal tumours is sometimes enormous, the mass reaches to the ground, the penis is completely lost within it, and the whole weight may exceed that of the rest of the patient; yet the organs involved in this monstrous tumour are, when dissected out, found perfectly normal, except that the tunica vaginalis is often the seat of hydrocele. Many remarkable cases with numerous illustrations will be found recorded in Esmarch's and Kulenkampff's monograph, 'Die Elephantiasistischen Formen,' 1885.

Ulceration of the unwieldy mass of flesh often occurs, and the pain and discharge of the ulcers may produce a certain amount of cachexia.

The *cause* of the disease was until lately unknown, but owing to the remarkable discoveries made by Dr Lewis, Dr Manson, and other observers, it is now known that a certain proportion of cases of elephantiasis, particu-

larly when it affects the scrotum, coincide with chyluria and the presence of a parasitic worm in the blood (*Filaria hæmatobia*, *supra*, p. 472). It is supposed that the lymph-channels are mechanically blocked by the parasites; this leads to œdema and inflammation on the one hand, and, when rupture into the urinary tract occurs, to chyluria on the other.

There is no doubt, however, that many cases of elephantiasis have been observed in which no filariæ could be detected in the blood. See a case with discharge of milky lymph recorded by Dr Wagstaffe ('Path. Trans.,' 1875, p. 215), and in the same volume one with great lymphatic dilatation figured by Mr Stewart, as well as a third case of ordinary pachydermia with histological details by Mr Butlin.

The *treatment* of this disease is purely surgical. There appears to be little or no power of restraining its course until the tumour is sufficiently large to be removed. From the famous cases of Clot Bey in Egypt to those of Dr Turner in Samoa and other medical missionaries in China, the removal of these frightful masses of flesh has been one of the most brilliant benefits conferred by European surgery.

XANTHELASMA.*—This remarkable affection was originally described by Addison and Gull under the name of *Vitiligoidea*. Afterwards Dr Pavy, Dr G. H. Barlow, Dr Fagge, and many others published similar cases. See 'Guy's Hosp. Rep.' for 1851 (plates); *ibid.*, 1866 (plates); *ibid.*, 1877, with thirty-eight tabulated cases; and 'Path. Trans.,' 1866, p. 277 (plates); *ibid.*, 1868, p. 436; *ibid.*, 1882, p. 376, with thirty-six cases of multiple xanthelasma.

Xanthoma shows itself in two anatomical forms. One is the commoner, and was first figured and briefly mentioned by Rayer as yellow patches on the eyelids in 1835 (see the writer's paper in the 'Guy's Hosp. Rep.' for 1877, xxii, p. 97); these flat patches (*Vitiligoidea plana* of Addison and Gull) cannot be detected by the finger, although they look raised and have defined margins.

The first indication of xanthelasma is the appearance in one of the upper eyelids, just above the internal canthus, of a yellow, cream-coloured, or washleather-like patch. Afterwards similar ones come out in the same neighbourhood, and these may ultimately coalesce so as to form a broad ring surrounding the eyes. Like patches may show themselves elsewhere—on the surface of the body, on the backs of the hands, on the scrotum, and also on the palms and soles, where they either present a peculiar dotted appearance, or form long streaks following the creases of the skin. This plane variety of xanthelasma may affect mucous membranes as well as the skin. It occurs in the gums and palate, and in the larynx and trachea; Dr Legg has seen it on the side of the tongue, and in two cases Dr Fagge found it in the lining of the bile-ducts.

The second and rarer form of xanthelasma consists of raised solid nodules or tumours (*Vitiligoidea tuberosa*). These make their appearance later than the flat patches. They occur on the ears and on the limbs, especially on the extensor surfaces; they form aggregated tubera on the olecranon, and swellings on the knuckles not unlike those of gout. They are

* *Synonyms*.—Plaques jaunâtres des paupières (Rayer)—*Vitiligoidea plana et tuberosa* (Addison and Gull)—Xanthelasma, *i. e.* yellow laminae, from *ξανθός* and *ελασμα* (Wilson)—*Molluscum lipomatodes* (Wilson). The term Xanthoma (yellow tumour) was suggested by Dr Frank Smith, of Sheffield, in 1869, and has been generally adopted in Germany.

occasionally found, not in the skin itself, but in the adjacent tendons of the extensor muscles of the fingers.

Two cases are reported in 'Virchow's Archiv' (1883 and 1885) in which the eyelids were unaffected, but nodules and tumours occupied the elbows, fingers, knees, and buttocks.

When the flat plates and the nodules are found in the same patient, the term *Xanthelasma multiplex* has been applied. Twenty-three cases are tabulated in the thirty-third volume of the 'Pathological Transactions,' p. 381. See also Dr Payne's case ('St Thos. Hosp. Reports,' vol. xiii).

Symptoms and course.—Xanthelasma is important not only because of its pathological interest, but because it is often attended with much suffering to the patient. The parts affected with it are sometimes exceedingly tender. A patient of Dr Fagge's was unable to stand, or even to sit with comfort, on account of the pain produced by the slightest pressure on the xanthelasmic patches, and for a similar reason she could not use her needle. In this case most of the raised tubera gradually disappeared, and the pains were in great part removed.

Similar involution has been observed in other cases, *e. g.* a remarkable one associated with icterus from atrophy of the liver, recorded by the late Dr Frank Smith, of Sheffield ('Path. Trans.,' xxviii, 236).

Histology.—Microscopical examination shows that xanthelasma is essentially a chronic deep dermatitis with early fatty degeneration, the yellow colour depending upon the presence of innumerable fatty granules in the tissue. In the nodules there is also present a dense fibrous tissue. The minute structure of xanthelasma is thus identical with that of atheroma in an artery—a comparison first made by Mr Howse. Some dermatologists, however, taking the less frequent tuberoso form as the type, describe the disease as a new growth, and call it Xanthoma (p. 966, *note*). The distinction between chronic inflammation, hypertrophy, and granuloma is, as we have already found, by no means easy to make in every case, and we may regard xanthoma as one of the transition forms between the chronic dermatitis described in this chapter, and the granulomata of syphilis, lupus, and leprosy.

In the areolæ of the connective tissue affected with xanthelasma are numerous nucleated cells filled with separate oil-drops, and these do not coalesce into the single large drop which fills a normal cell of adipose tissue. Such abnormal fat-cells are called xanthoma-corpules, and seem to be characteristic and constant.

In palpebral xanthelasma, Dr Politzer believes that the fibres of the orbicularis muscle undergo fatty degeneration and cause the yellow streaks.

Pathology.—The multiple and tuberoso form of the disease is most frequently seen in chronic cases of *jaundice*, from whatever cause it may arise, although it may also be found, particularly in children, in cases entirely free from icterus. Of eight cases of infantile xanthelasma multiplex, not one was associated with jaundice; and in these the eyelids were not affected, as they almost always are in adults ('Path. Trans.,' vol. xxxiii, p. 383). Usually xanthoma does not make its appearance until the patient has been jaundiced for a year or more, but in one case it began within six months after the jaundice, or perhaps even earlier. The more common *plane* form of xanthelasma which is confined to the eyelids has been shown by Mr Hutchinson to occur frequently in those who have suffered from sick headaches ('Med.-Chir. Trans.,' 1871).

Xanthelasma has been observed more frequently in women than in men, in the proportion of three to two. It occurs, like atheroma, most often in adults over forty years of age; but several examples are recorded in children, and two congenital cases by Dr Stephen Mackenzie and Sir Thos. Barlow ('Path. Trans.,' 1882 and 1884). Sir Wm. Church met with six cases of the affection in the same family ('St Barth. Hosp. Rep.,' vol. x), and several cases of the same kind have been since reported here and in America.

No plan of treatment is known except incision; but it is only in exceptional cases that symptoms arise, and there is some reason to hope for spontaneous recovery.

Xanthoma diabeticorum is the name given to a curious and rare affection of the skin, which is only doubtfully related to classical xanthoma. The earliest case was the fifth in Addison and Gull's original paper. The patient was a man aged twenty-seven, and during the course of diabetes a papular eruption appeared on his arms, and rapidly spread over the trunk, the head, and the limbs. The papules were large, yellowish, and mottled, and looked like pustules. After several weeks these "tubercles" began to subside, leaving no obvious change behind them. This is certainly very different from the affection above described, and Dr Fagge ('Path. Trans.,' xix) and the present writer ('Guy's Hosp. Rep.,' 1877, p. 131) agreed in excluding the case from the group.

Several similar cases have been since reported by Bristowe (as "ke-loid"), by Malcolm Morris ('Path. Trans.,' 1883) and Thos. Barlow ('Brit. Journ. Derm.,' vol. i, and vol. iii, p. 106), by A. R. Robinson (from New York), and by Gendre and Besnier from Paris ('Ann. de Derm. et de Syph.,' 1889). They have also been called diabetic "lichen" and "acne." The papules are red, with a yellow head, as if they contained a drop of pus; and sometimes coalesce. They are more often painful or irritable than are patches of xanthelasma.

The histology of these papules is different from that of xanthoma ('Path. Trans.,' xvii, p. 414, and xxxiv, p. 284), and so are their anatomy, distribution, origin, and course. They occur chiefly on the limbs, but also on the buttocks and on the head. They appear somewhat abruptly, last a long time, and then gradually fade away.

Several instances have now been reported of a similar cutaneous disorder without diabetes:—Cavafy's case, 'Brit. Journ. Derm.,' vol. i, p. 76, and also those of Bristowe, Hutchinson, Besnier, and Vidal.

Most of the patients were men between twenty-five and fifty. Histological examination shows that the papules are those of deep dermatitis, the whole corium being filled with exudation cells. The yellow colour is probably due to fatty degeneration, but in most cases is deeper and brighter yellow than we see in ordinary xanthelasma. Dr Norman Walker, of Edinburgh, collected 29 cases, making 30 with an original one (*ibid.*, Dec., 1897, vol. ix, p. 461), from 1851 downwards. In most of these, but with many exceptions, there was glycosuria or complete diabetes. All were adults. He describes the structure as a new formation of connective tissue without corpuscular exudation, and with abundant fatty degeneration. Like other observers, he found no characteristic microbes.

SYPHILODERMIA

“Indignas premeret pestis cum tabida fauces,
Inque ipsos vultus serperet atra Lues.”

MART., i, 79.

*Importance of lues as a cause of cutaneous lesions—their pathological character—
The roseolous exanthem—the papular—squamous—pustular—bullous—
macular—Alopecia—Tertiary lesions—Cicatrices—Diagnosis of syphilitic
dermatoses—Hereditary syphilodermia—Treatment.*

THERE is no doubt that the only satisfactory classification of diseases, whether of the skin or of other organs, would be an ætiological one; that is to say, one which expresses their true origin and natural history, not as so-called morbid entities, but as derangements more or less extensive and profound of physiological function. For if we know the origin of a disease, we not only understand its nature or pathology, and may hope to adopt rational means for its cure, but we also may still more confidently hope to prevent, which is better than curing it. The objections to the supposed ætiology of diseases which we have often urged throughout this book, are that causes are often assumed instead of proved, and that no cause can be logically admitted which is not an invariable antecedent of the effect. Hence it is that we demur to explaining inflammations as due to cold, that we doubt the origin of rheumatic fever from bacterial infection, and cannot accept a gouty diathesis, a scrofulous tendency, or a neurotic disposition without a careful examination of the grounds on which such causes are assigned. The same reasonable scepticism applies to the dartrous or arthritic diathesis which was invented as a cause for many diseases of the skin, or to the ready invocation of the nervous system to explain dermatoses. Just as no one believes firmly and intelligently in the use of any remedies if he believes in all, so we shall not either appreciate or trust to true ætiology until we have separated it from what is spurious. Enteric fever and scabies were both at one time ascribed to meteoric causes, to the influence of the seasons, to diathesis, to dyscrasia of the blood and humours, to chills, to mental affections, and to all the other commonplaces of unscientific speculation. We now know that their single, constant, and exclusive cause is the transference, in the one case of a plant, in the other of an animal, from one human organism to another.

When the true ætiology of any disease is ascertained, we know its con-

stant and necessary antecedent, and can hope to prevent or to cure it by rational methods.

In the time of Willan, psoriasis, for example, was regarded from the anatomical point of view as a scaly disease caused (among many vague and uncertain conditions) by the presence of syphilis. We now know that true psoriasis is a definite and well-marked derangement of cutaneous nutrition, with its own natural history and characters, and that it is never due to syphilis; that what used to be called syphilitic psoriasis is no more entitled to the name than is a rash caused by belladonna to that of scarlatina. If diagnosis is not to be mere learned or unlearned trifling, it means drawing a distinction—not between one pimple and another, but between morbid processes as distinct in their origin, nature, and practical consequences as poisoning by a drug, invasion by a fungus, and infection with a bacillus.

The separation by Bielt, therefore, and his successors in France of the syphilides from all other cutaneous diseases was a great improvement upon Willan's pathology, an advance none the less important because it led to an extension of the same excellent principle to tuberculous diseases.

We have already in the first volume stated the general character of syphilitic infection, its mode of transference, generalisation, and transmission to a second generation. We have now to deal in particular with its manifestations upon the skin.*

These lesions are multiform. Syphilis is one of the specific febrile diseases, and its earliest effect on the integument is the production of an exanthem or specific rash strictly comparable to those of measles, typhus, and smallpox. The syphilitic process is, however, drawn out far beyond that of any other member of its class; and its exanthem is accordingly protracted in duration and extremely varied in form. It is as if a case of variola lasted not for a few weeks, but for as many months. We should then see an early roseolous rash, followed after an interval by the appearance of papules with characteristic distribution and anatomy; afterwards vesicles would succeed, then pustules, crusts, hæmorrhages, and finally cicatrization; while the orderly march of the eruption would be interrupted or modified by local conditions during its tardy and irregular progress. Hence it is that the cutaneous lesions of syphilis present so multiform a variety, and are so irregular in their appearance, locality, and course.

Another important pathological point is that syphilis, like many diseases, has sequelæ, and is itself so chronic that it is often difficult to say when the disease is exhausted and the sequelæ have begun. Compared with the secondary lesions, which form part of a prolonged exanthem, the latter forms of syphilodermia may be called sequelæ; but, nevertheless, they are certainly parts of the true syphilitic process, and may in their turn be distinguished from affections like tabes dorsalis and paralytic dementia, which correspond more strictly to what in other cases we call sequelæ—results, but not parts of the original disease—"parasyphilides."

The syphilitic exanthem ("syphilitic roseola").—The rash is erythematous in form, like that of measles and scarlatina; it occurs in patches, not uniformly; it is not raised above the skin, and disappears on pressure; the colour is in most cases an inflammatory redness tinged with brown pigmentation, so as to produce a tint which differs from the bright red of

* *Synonyms.*—Syphilis cutanea—Lues (*sc. venerea*) in cute—Les syphilides—Dermato-syphilis.

scarlatina, and from the purplish or rose-tinted hue of measles by the admixture of a yellowish tint, producing a salmon-coloured, coppery shade, which French writers compare to that of the lean of uncooked ham. The patches are not sharply defined at the margin, and are irregular in shape. The most characteristic distribution is on the trunk, particularly the front and sides of the chest, on the abdomen, and on the neck. The rash is not infrequently seen upon the face, but is rare upon the limbs, and seldom or never reaches the hands or feet. It appears at a variable time after the primary infection, usually three or four weeks, but sometimes much later. It takes, as a rule, several days, and often a week, to develop; when it comes out unusually quickly it may simulate the rash of measles, or that produced by copaiba or some other drug.

The papular form ("syphilitic lichen").—Sometimes as a part of the early exanthem, more frequently after an interval, papules show themselves, large, pointed, discrete, and very early covered with small scales. They are more like those of psoriasis than of papular erythema or lichen planus, and are larger and less confluent than those of eczema. They appear on the trunk and limbs, and also on the forehead, at the roots of the hair. Like the exanthem, they produce as a rule neither pain nor irritation.

One variety of papular rash may occur early in the post-erythematous series. It is marked by each papule being formed around a hair sac. Hence Dr Bäumer has proposed to call it the *follicular* syphilide. The papules are small and pointed, "miliary." They may be discrete or clustered, they appear in successive crops, and may last for several weeks or even months. They sometimes become scaly, sometimes vesicular, but most often, perhaps, pustular.

Squamous syphiloderma (formerly known as "syphilitic psoriasis") most frequently occurs as a further development of the papular eruption just described. The scales are much smaller than those of psoriasis, and have a dirty yellowish colour instead of a silvery lustre. The distribution of the scaly syphilide is much like that of the papular stage, except that it frequently affects the palms and soles even thus early. The trunk, limbs, and face, the scalp, and the genital organs may all be the seat of this form of eruption; but it affects the flexor rather than the extensor surfaces, and avoids the regions peculiar to psoriasis, the elbows and the knees. At this stage Mr Hutchinson describes the occasional appearance of a kind of erythematous ring on the arms and trunk, which appears again and again, when the patient has left his bed or has taken his bath, and lasts for a short time.

Pustular syphilide (syphilitic "impetigo" and "ecthyma").—Venereal pustules are usually developed out of the papular stage, but sometimes seem to come independently; they are often large and discrete, whence they have been called by the obsolete name of ecthyma. They are frequently mixed with papules and scales and injected maculæ, giving the polymorphic aspect which is characteristic of syphilis. This pustular eruption is irregular in locality, but is frequent on the scalp, face, and trunk, and comparatively rare upon the limbs. The pustules are very commonly followed by minute white scars, which are often useful in subsequent diagnosis. Crusts form much as they do with eczema and impetigo, but instead of being green or yellow they are usually a reddish brown.

The pustular eruption of syphilis has often to be carefully distinguished from varicella and from the modified form of smallpox.

A form of pustular syphilide, which Bäumler has designated "follicular," is that in which each pustule is pierced by a hair. It has a swollen dark reddish base, forms a minute yellowish crust, and leaves a white cicatrix. They come out in great numbers on the face, chest, shoulders, and limbs. This is the form which was formerly known as "syphilitic acne."

Vesicular and bullous syphilides.—The older dermatologists did not admit "syphilitic eczema," and there is no doubt that vesicles are extremely rare as the result of syphilis.*

Bullæ are not at all uncommon among the later secondary lesions. They become pustular, and form massive conical crusts, sometimes with a curious resemblance in form and colour to a limpet shell. This is the eruption which was formerly known as *rupia*. When the crust is removed ulceration is found beneath it. A bullous syphilide is also common in congenital cases upon the palms and soles; it used to be known as "pemphigus neonatorum syphiliticus."

Macular syphilides.—All the eruptions just described are marked more or less decisively by the coppery pigmentation characteristic of the disease, but somewhat late in the secondary stage maculæ without any other lesion begin to appear. These primary syphilitic stains are much more common in women than in men. Hardy described them as usually appearing on the neck and front of the chest, consisting of irregular spots of a *café au lait* colour, as large as a franc or half-franc piece, and often confluent.

In the Guy's museum is a model, together with drawings, taken from a typical case of this form of syphilide which was under Dr Barlow's care in 1856. The patient, a woman, had dark brown maculæ scattered over her chest and arms; it was eight months since the beginning of the syphilis, and the stains faded under treatment.

We shall see in a subsequent chapter that increased pigmentation (melasma, melanoderma) frequently coincides with an adjacent deficiency of pigment (leucoderma), and this is what often occurs with the pigmentary syphilide just described, defined white patches appearing in the diffused dark patch. Varieties of aspect and distribution have been distinguished as "marble-like," "macular," and "lace-like."

Beside the most frequent position on the neck, it may appear on the flanks, or the chest, or abdomen.

Its period is often early, from three to six months after infection, less frequently somewhat later, but when once developed it continues unaltered for three or four years or even longer. It then disappears spontaneously. Its appearance usually coincides with that of mucous patches and condylomata, but sometimes is as late as tertiary nodes.

Syphilitic alopecia is an early symptom, and is not always dependent upon a scaly or pustular affection of the scalp; when this is absent we are probably right in regarding the loss of hair as of the same nature as that which often follows many of the specific fevers. It is distinguished from *alopecia præmatura* by not specially affecting the forehead or the back of the head. It is distinguished from *area* by the absence of circumscribed smooth hairless patches. The hair becomes thin, and forms irregular bald patches in various places.

* Hutchinson speaks of a form of syphilide which is attended with clusters of vesicles like those of shingles, but which is bilateral and widely distributed; and Hardy describes three varieties of syphilide, which he terms eczematous, varioliform, and herpetiform.

Tertiary dermatosyphilis.—The pustular and especially the rupial eruptions often come so late in the secondary period that they are accompanied by syphilitic ulcers, and, again, true gummata may occasionally appear in the skin at a comparatively early period; in fact, the yellow nodules of early syphilitic iritis are considered by good pathologists to be themselves minute gummata (vol. i, p. 288). We cannot, therefore, draw an absolute line between the preceding forms of syphilodermia as symptoms, and the tertiary lesions as sequelæ; but whether we use the word tertiary or speak only of late symptoms, the distinction is practical and important between the roseolous, macular, papular, squamous, and pustular syphilides on the one hand, and the later nodes and ulcers on the other. The former are forms of dermatitis, the latter of granuloma. The former are associated with sore throat and iritis, the latter with the visceral affections described in the first volume (p. 290).

Syphilitic *condylomata* and mucous patches have been sufficiently described already (pp. 884-5).

The syphilitic *ulcer* may begin in a cutaneous gumma or beneath a rupial crust, or in a patch of syphilitic scales or pustules. It spreads from the margin, or, as the phrase is now applied, it is serpiginous. It is usually, but not always, multiple; the edges are rounded and punched out. It causes remarkably little irritation or pain; it may occur upon any part of the body, trunk, head, or limbs; it may often be distinguished by its presence upon parts which are little liable to other forms of ulcer. Hence an ulcer of the arm is more likely to be syphilitic than one of the leg, and an ulcer over the fibula than one above the inner ankle.

Syphilitic cicatrices follow the pustular, the rupial, and the ulcerating lesions. They are often rounded or horseshoe in form, pigmented, smooth, and atrophic rather than hypertrophic. Their irregular localisation is, perhaps, the best evidence of their nature.

Diagnosis.—It is most important to distinguish syphilitic diseases of the skin from those of traumatic, febrile, and idiopathic origin; for, apart from other considerations, an error in diagnosis is fatal to successful treatment.

The rules for diagnosis of syphilis so far as relates to the skin may be briefly given as follows.

Firstly, what is called *history* is a most fallacious guide. In hospital practice the writer was accustomed to neglect it entirely; and although, when the patient's statements are carefully sifted, they have a certain value, the greatest mistakes are those which are due to reliance upon this misleading character; for we must always remember that even the most truthful patient may be quite deceived in supposing that he had syphilis, when he suffered from a different venereal malady, or may have been erroneously informed that a venereal sore was non-specific; and, as we insisted before, syphilis is, in a considerable minority of cases, a non-venereal disease, and in a certain number may be transmitted without any primary lesions at all (*cf.* vol. i, p. 291).

Secondly, the elementary lesions of dermatosyphilis are *multiform*, not mere stages of inflammation, as in eczema or erythema multiforme, but diverse from the beginning.

Thirdly, the *colour* is an important character, but is sometimes absent from the earliest exanthem, when its presence would be most useful for

diagnosis. Even in subsequent forms it is occasionally much less marked than usual, and may be simulated by certain cases of chronic eczema or psoriasis, or still more frequently by lichen planus.

The irregular *distribution* of the syphilides is an important aid to diagnosis. Syphilis in its early stage may, if extensive, be symmetrical in the same sense as measles. But true symmetry, when homologous regions are independently affected—such as we see in psoriasis and eczema—is seldom or never seen in syphilis, with the exception of that which affects the palms and soles.

The *absence of itching* from the earlier, and of pain from the later eruptions is very remarkable, and almost though not absolutely constant.

Lastly, the presence of *concomitant lesions* in other organs than the skin, the eyes, the throat, the lymph glands or the testes, the presence of scars or maculæ, will often render a doubtful diagnosis certain, or their absence will be decisive against a suspicious eruption being specific. Nevertheless we must be on our guard against the common fallacy of supposing because a patient has had syphilis or is even suffering from syphilitic lesions at the time of examination, that any affection of his skin must needs own the same cause. We have only to remember that a man suffering from syphilis may be attacked by smallpox, typhus, or scabies, and that a patient suffering from chronic psoriasis or varicose ulcers may contract lues, to see the fallacy of trusting to what after all is only a probability.

Congenital syphilodermia has essentially the same characters as that due to an acquired lesion; in infants the commonest eruption is a coppery, blotchy rash on the nates and thighs. The bullous syphilide of the soles is almost peculiar to the congenital disease; this is often accompanied with mucous tubercles of the anus. In later childhood, typical gummata may develop and leave the characteristic ulcers (*cf.* vol. i, p. 357).

The *prognosis* and *treatment* of dermato-syphilis is that of the general disease of which it is part, and has been already fully discussed in the first volume. Mercury is almost always indicated, and iodide of potassium or sodium should only be substituted when cutaneous gummata or tertiary ulcers are present. If the ulcer does not speedily heal, the dose of iodide should be increased. Phagedænic ulceration always calls for treatment by opium.

Locally, the slighter forms of eruption need no special treatment. Ulcers and raw surfaces should have black wash or red ointment applied, and condylomata should be dusted with calomel. If there is a sloughing or otherwise unhealthy surface, iodoform is often used with great benefit.

LUPUS

AND ITS ALLIES

“Heus tibi autem—Quidnam est?—Lupus in fabula.”

TERENCE, *Adelphi*, iv, i, 31.

Definition, history, and nomenclature—Anatomy, histology, and local course—A tuberculous lesion—Distribution—Symptoms—Age, etc.—Diagnosis from cancer, rodent ulcer, and syphilis—Pathology—Relation to tubercle and scrofula, to phthisis and other granulomata—Clinical course and prognosis—Treatment, local, internal, by inoculation with tuberculin and by light. Tuberculous ulcers—Bazin’s erythema induratum—Tuberculosis verrucosa. Lupus-erythematosus—Pathology—Locality—Course—Histology—Treatment. Disseminated erythematous lupus. Rhinoscleroma.

Of all diseases which affect the skin alone, lupus is the most destructive. Unlike syphilis, leprosy, and malignant growths, it affects only the skin and the adjacent mucous membranes. Its seat is the deep layer of the cutis, and the epidermis is only involved subsequently. It also spreads to the subcutaneous tissue and occasionally to adjacent mucous membranes, but rarely to the cartilages or fascia propria beneath the skin; and it never attacks muscles, bones, or other deep structures. Lupus is at once a chronic deep dermatitis of a special kind and a new growth in the modern sense of the word. Virchow included it with tubercle, leprosy, and syphilis among the Granulomata, or new growths which consist of the same corpuscular elements which form the granulations of a healing ulcer. By the French writers it was assumed to be tuberculous in nature, and was included in the family of Scrofulides. This view, though not shared by Hebra or the earlier English dermatologists, is now amply justified and universally accepted. In earlier times lupus was confounded with ordinary chronic ulcers, with cancer, with leprosy, and most of all with the later forms of lues. The lupus exedens of older writers was in most cases tertiary syphilis, *e. g.* the woodcut given as a type of the disease in Druitt’s ‘Vade Mecum.’

Lupus* was first carefully defined and described by Willan, and was

* *Synonyms.*—Tuberculosis cutis—Noli me tangere—Tentigo prava—Impetigo rodens—Herpes exedens.

The word “lupus,” which has been traced back by Virchow to the school of Salerno in the thirteenth century (‘Archiv,’ vol. xxxii, 1865), expresses the destructive ravages of the disease. It is applied to an incurable ulcer by Alexis of Piedmont (1578). The dramatist Webster uses the English equivalent in the phrase “the ulcerous wolf.”

figured by Bateman in his sixty-seventh plate. He noticed its characteristic preference for the face, and placed it among Tubercula, *i. e.* new growths, not tuberculous in the modern sense. The distinctions subsequently introduced—*Lupus exedens* and *L. non exedens* (Rayer), the lupus “qui détruit en surface—en profondeur—avec hypertrophie” (Cazenave and Schedel)—*Lupus serpiginosus*, *syphiliticus*, and *vulgaris*, with many others—are unnecessary or mischievous. Only one aberrant form, or rather allied disease, *Lupus erythematosus*, need be separately described or named.

Anatomy.—Lupus begins by the formation of minute nodules of granulation tissue in the deeper layer of the cutis. These can be felt like shot in the skin, although without hardness, and show as reddish spots which mark the “macular” stage. When exposed, they are found to be vascular; and when several are seen together, a yellowish tint is observable on pressure, which with their soft translucent appearance has led to their being compared to apple jelly.

Histologically, they consist at first of small nucleated lymph-like cells, with very scanty stroma. As the disease goes on, these granulation nodules unite, and undergo changes in two directions. The intercellular substance may become a stroma of delicate connective tissue. This may increase and acquire firmness until it becomes fibrous tissue with spindle-shaped corpuscles; and finally may form a dense, contracted tissue which resembles an atrophic or hypertrophic cicatrix. More frequently, however, either universally, or with only a certain amount of the fibrous transformation just described, the new-formed lupus tissue breaks down, the nodules become confluent, the cells undergo fatty degeneration, ulceration destroys the new growths, the epidermis gives way, and an ulcer results. The floor of this ulcer is formed by lupous nodules, which can be distinguished by the naked eye from the healthy granulations of a healing sore. The edges are somewhat raised and can generally be felt to consist of nodules which have not yet softened. The pus secreted is usually thin and scanty. While fresh deposition of nodules and fresh softening and ulceration ensue, there is usually some effort at repair by the fibrous transformation above described.

The skin around, though red and slightly swollen, does not feel hot, and the redness is of a venous tint.

The whole process is strikingly similar to that which occurs in the lungs during the course of phthisis. There, also, we have minute nodules of granulation tissue, which have been described both as new growths and as inflammatory. There, also, the nodules undergo softening and ulceration; the ulceration spreads, with chronic inflammation and continual deposit of fresh tubercles. There, also, the ulcerative process is rarely unaccompanied by some amount of fibrous transformation, which in favourable cases leads to the involution of the disease and the formation of a cicatrix. The bearing of this resemblance on the theory of lupus will be presently seen.

The process above described is extremely slow. We may watch lupus for more than a year before it ulcerates. It usually begins at a single spot and spreads irregularly therefrom, sometimes in a serpiginous form and comparatively swiftly, more often with an irregular rounded shape. It is rare for two independent foci of lupus to be seen, but this may sometimes occur. Whether in separate patches or as a single spreading surface, the disease is decidedly unsymmetrical, unless it happens to begin in the median line.

The epidermis is usually more or less thickened, particularly the deepest

layer; but the original seat of the process is between the papillary and the deeper layer of the cutis, whence it spreads downwards as well as upwards, until the whole thickness of the true skin is infiltrated. According to Auspitz, the sebaceous glands are destroyed, the sweat-glands are unchanged, the hair-follicles disappear or are transformed into cysts. Rindfleisch taught that lupus begins in the sebaceous glands—not the erythematous or so-called sebaceous form, but lupus vulgaris; and he even called it an adenoma. This no doubt was a mistake; any inflammatory disease will show most exudation in the more vascular parts of an organ, and the most vascular parts of the skin are the papillæ, the hair-sacs, and the sebaceous glands; but the glands themselves are not involved except as a secondary result in the disease. Neither Neumann nor subsequent histologists agree with Rindfleisch.

Giant-cells are frequently observed (Friedländer in 'Virchow's Archiv,' 1874; and Thin, 'Med.-Chir. Trans.,' vol. lxii). Hence lupus resembles tuberculous lesions in histology as well as in anatomy. Finally, the characteristic bacilli of tubercle (vol. i, p. 368) are present. They were discovered by Doutrelepon in 1883.

Locality.—Lupus by preference attacks the face, particularly the alæ of the nose, the edges of the lips, the cheeks, the eyelids, and the conjunctiva. It also occurs upon the ears and spreads to the neck. It is rarely seen on the scalp and is not common on the trunk and limbs; the hands and feet are almost exempt. But there is probably no part of the body on which lupus has not been observed; and although it is rare to see two lupous ulcers at once, it often, after appearing and being cured upon the face, reappears in another region. In Vienna the trunk and the buttocks appear to be more often the seat of lupus than the arms and legs.

Lupus also affects the mucous membrane of the nose, the lips, the hard and soft palate, and the larynx (*cf.* p. 128). It very rarely affects the tongue* or the other mucous membranes; but lupus vaginæ was described by Matthews Duncan, and it has been recorded not only on the scrotum but on the prepuce. When the disease is unusually virulent it may occasionally penetrate from the skin or mucous membrane to the cartilages of nose and ears, and even the tendons and ligaments of joints.

Symptoms.—As the progress of the disease is slow and its local signs torpid, so its symptoms are but slight. It is astonishing how little pain is felt even when extensive tracts of the skin are deeply ulcerated; as we shall presently see, the remedy is far more painful than the disease. The general health is also unaffected; so that, except for the disfigurement, lupus would be one of the most easily borne of all serious and destructive diseases. It is remarkable that patients suffering from lupus very seldom have a history of tuberculosis in the family—not more often than healthy persons. Nor are they, except in rare cases, affected by phthisis and other forms of internal tubercle. Most remarkable of all is the fact that the adjacent lymph-glands are scarcely ever the seat of secondary caseous inflammation by infection from lupus.

Ætiology.—Though no doubt lupus is always due to tuberculous invasion, the disease certainly is not "catching" or contagious in the ordinary sense of the word.†

* Leloir's case, quoted by Besnier and Doyon (tome ii, p. 449), is one of the very few on record of lingual lupus.

† There are, however, cases of inoculation recorded, *e.g.* the policeman who struck a consumptive burglar on the teeth and got lupus of the hand. Jadassohn described a case from tattooing, done by a consumptive operator.

With respect to *age*, it is, as commonly seen, a disease of young adult life. But it has then lasted in most cases for several years, and it may occur in young children. We have observed it in those not above four or five years old, but it is more common about puberty, and usually begins at from fourteen or fifteen to twenty. After thirty it is certainly rare for it to begin, though cases of undoubted lupus may be occasionally observed which start even later. According to Hebra and his disciples, it always begins before puberty, occasionally in infancy, but usually from the fifth to the ninth or tenth year.

In young subjects it shows an almost constant tendency to spread, even when repair, as generally happens, is to some extent occurring at the same time; but after thirty lupus tends to undergo involution, and if left to itself will in certain cases end in cicatrices, disfiguring or disabling the patient, but no longer active.

Lupus is not hereditary, and has never been seen at birth, nor does it often affect more than one member of the same family. It is probably equally common in both sexes.

Devergie noticed that lupus is more often seen amongst hospital patients than in private practice, and this is certainly true in England as well as in France. It is more common in Vienna than in London.*

Diagnosis.—The fact of lupus being a deep inflammation of the skin, and leaving scars, at once distinguishes it from eczema and all the superficial forms of dermatitis enumerated in the earlier chapters of this section; nor is there much practical difficulty in the diagnosis from varicose and traumatic or accidental ulcers. The real difficulties of diagnosis arise between lupus, syphilis, rodent ulcer, and cancer of the skin.

Lupus is distinguished from *cancer* by the absence of pain, by its slow progress, by its beginning early in life, by the presence of granulations, the absence of hæmorrhage, and the nodulated, but not uniformly and densely infiltrated, edge. Invasion of deeper structures and secondary enlargement of the corresponding lymph-glands decides the case to be cancerous, but our object is to establish a diagnosis long before this point has been reached.

Rodent ulcer is covered by an adherent reddish-brown scab, which when present is characteristic; but it may have been removed by accident, by poulticing or by other remedies, before the lesion is seen. The edges are neither thick, hard, and infiltrated like those of epithelial cancer, nor do they contain little nodules, as in lupus. Granulations are absent, the ulcer being of the kind known as “indolent,” while that of lupus is what is called “weak.” Like lupus, its favourite seat is on the face, but in the neighbourhood of the eyes rather than on the cheeks and nose; it is always single, and never extends so widely as lupus; it makes no attempt at spontaneous cicatrization; and it only occurs in those who are past middle life.

Syphilitic ulcers have an undermined, not an infiltrated or nodular edge; the colour of the surrounding skin is brownish or yellowish, whereas that of lupus is of a more venous, *i. e.* purplish red. In both there may be considerable crusts, forming what is described as “rupia” and “ecthyma” in the one case, and as “lupus pustulosus et crustaceus” in the other; but when these are removed, the more characteristic ulcerated surface beneath will be seen. The scars which result when a syphilitic ulcer is healed often resemble those of lupus, but they are less apt to be hypertrophied, they

* It appears to be rare in New England, where consumption is very common.

are much more pigmented, and seldom present the pink aspect and enlarged veins which are often seen after lupus is healed.

Moreover, tertiary syphilitic ulcers begin as a rule with the formation of a gumma in or under the skin, deeper than the nodules of lupus and less early affecting the epithelium. In the nose this distinction is most applicable—lupus begins at the edge of the nostril and slowly creeps on, only affecting the cartilages (if it does at all) in its latest stages; whereas syphilis begins in the perichondrium or periosteum, and has already destroyed much underlying tissue before the ulcer on the skin appears. The nose which has lost its tip or alæ has usually been affected by lupus; that which has lost its bridge, by syphilis. Extensive disease of the skin, with the cartilages and septum intact, is most likely lupus; a small ulcer with a deep foul cavity beneath it, and exposed bone and cartilage, is almost certainly syphilis. The frightful cases of destruction of the greater part of the face and opening of the orbit, pharynx, and posterior nares, which figured in museums and plates as lupus exedens, were tertiary syphilis, neglected or ill-treated, cases which the better diagnosis and improved therapeutics of modern times have happily banished from civilised countries.

Apart from the local characters, diagnosis between syphilis and lupus will be much helped by remembering that syphilitic ulcers are frequently multiple, lupus very rarely so; and that secondary implication of lymph-glands, with characteristic induration, is common in syphilis—rare and only as the accidental consequence of temporary inflammation, which renders them soft and painful, in lupus. Again, the syphilitic ulcer is usually accompanied by other cutaneous lesions; the lupous ulcer has no complication. Syphilis begins after puberty, often long after; lupus before puberty or shortly after. Lastly, lupus is a disease of the skin and nothing else, whereas syphilitic gummata and ulcers will be generally accompanied by other signs of lues in the bones, glands, tongue, or viscera.

Of the cutaneous lesions of congenital syphilis, the early coppery rashes have no resemblance to lupus, and the later gummatous ulcers are like those of tertiary acquired syphilis, and unlike the ulcers of lupus.

Pathology—relation to syphilis.—Although it is scarcely possible to confound lupus with the ordinary lesions of congenital syphilis, it was once supposed by respectable authors that lupus is the result of inherited syphilis.* But no one pretends that a child with a syphilitic father or brothers is thereby *prevented* from becoming the subject of lupus, and if so the two diseases must occasionally occur in the same family or the same individual. Persons affected with lupus may acquire syphilis, and persons who inherit syphilis may be attacked by lupus. Owing to Mr Hutchinson's classical observations, we can now recognise congenital syphilis not only in infants, but in later life; and we find that such persons are not more liable to lupus than others.

The assumption that there is such a thing as "syphilitic lupus," a kind of hybrid between two diatheses, is also unjustified; and, like similar diagnoses of "rheumatic gout" or hybrids of scarlatina and measles, is practically mischievous. At the same time the diagnosis between syphilis

* Hebra himself was led into this opinion by a striking case in his own practice, where a syphilitic father had borne to him, first, a stillborn child; secondly, one which died in a few months with the ordinary marks of inherited syphilis; then a third who survived, after suffering from congenital syphilis of the skin and bones; while the last, who was born apparently healthy, remained so for some years, but became before puberty the subject of typical lupus.

and lupus is often difficult, and even with care and experience one may mistake the one for the other—at least the present writer has done so.

Relation to tubercle.—The French school have always held lupus to be a “scrofulide.” This word was invented by Bazin and Hardy in imitation of the Syphilides of Biett and Alibert. Bazin included among “Scrofulides bénignes,” chilblains, erythema, strophulus, prurigo, lichen, eczema, impetigo, and some forms of acne. These were justly excluded by the sounder judgment of M. Hardy, who precisely defined his scrofulides, and regarded their relation to scrofula as that of the syphilides to syphilis, never developing without it, and diagnostic of its presence. He included lupus, which Biett had, with his usual good sense, separated from all other diseases, while Alibert had lumped it with eczema and psoriasis among the “dartres.” Cazenave described four species of scrofulide under lupus: erythematous, tubercular, ulcerous, and hypertrophic. Bazin’s division of “Scrofulides malignes” was into erythematous, tubercular, and *scrofulide crustacée ulcéreuse*. Hardy addressed the same reproach to the third of Bazin’s as to the last two of Cazenave’s species, and himself described the following five varieties of scrofulides.*

1. *Scr. érythémateuse*.—This corresponds to erythematous lupus, an undoubtedly distinct affection, which will be described below.

2. *Scr. cornée et acnéique*.—This is not what other French writers describe as *acné cornée*, a curious affection of the sebaceous glands unaccompanied with inflammation, also called “ichthyosis follicularis” (p. 915). The *scrofulide cornée* of Hardy consists of groups of comedones, placed on a purplish-red patch, and is followed by depressed cicatrices unpreceded by ulceration. It appears upon the face, has a very slow course, and occurs (we may presume) only in persons who for some other reason are entitled to the epithet scrofulous. It corresponds to Devergie’s *Herpès crétacé*, and to Chausit’s *Acné atrophique*.

This affection would now probably also be called lupus erythematosus or lupus sebaceus.

3. *Scr. pustuleuse*.—This is the most frequent variety; it begins either by a number of pin’s-head pustules grouped on a small red patch, lasting from a week to a fortnight and leaving a yellow scab, or else with a large pustule, like that of ecthyma, which when ruptured gives place to a dark prominent crust, which other dermatologists would name rupia. The part usually affected is the nose, the course is very slow and unaccompanied by itching or pain, but the most characteristic point is that when the crusts, which are very adherent, are removed, ulceration is found beneath. This ulceration is not deep, the surface is pale and sometimes presents little hard, dry, rough, warty nodules, which led Hardy originally to describe the variety as “scrofulide verruqueuse.”

This lesion would by German, English, and American dermatologists be recognised as a form of lupus (*Impetigo rodens*), which, as above described, frequently begins in pustules and is accompanied by large scabs. The slow course of the disease makes the subsequent ulcerated stage much more familiar, but even if watched from the beginning, cases of lupus with pustules and large prominent crusts are seen in London, in a minority not less, perhaps, than a third or fourth of the whole.

* The later volume (‘Maladies de la Peau’), published in 1886, did not on this subject deviate from that which was taught in 1864.

4. *Scr. tuberculeuse** is divided again into a superficial and a deep variety, and the former distinguished as sometimes disseminated over various parts of the body and sometimes localised, sometimes inconspicuous and ending in a light atrophic scar, sometimes hypertrophied, especially when it affects the genital organs. M. Hardy speaks of the deeper tubercular scrofulides as producing “ces vastes destructions, ces plaies épouvantables et hideuses qu’on ne rencontre que trop souvent à la face,” and as occasionally proving fatal, with profuse suppuration, cachexia, and hectic; while, if it heals, large cicatrices follow on the eyelids, lips, neck, ears, or nostrils, like those produced by severe burns.

This is obviously lupus exedens in its severest and most destructive form, but not differing from the slighter forms accompanied with true ulceration, except in degree. Moreover, even when untreated, the ravages of lupus, however hideous, are more remarkable for their contrast with the deeper destruction of syphilis and cancer than for their extent and severity compared with other diseases of the skin.

5. *Scr. phlegmoneuse*.—This is a superficial ulcer which begins in a phlegmon as big as an almond or a nut; this gradually softens, fluctuates, acquires a purplish-red colour, and at last discharges a little thin pus; a scab forms, and this process may be repeated and become chronic until a large surface becomes ulcerated. The disease appears chiefly on the face, but also on the trunk and limbs. It also leaves a scar, at first violet-coloured, afterwards pale, irregular, and reticulated.

This somewhat rare variety will be recognised as what Hilton and other surgeons of his day used to describe as “scrofulous ulcer.” No doubt it deserves separate mention, and whether regarded histologically, or from the point of view of pathology and treatment, it is closely allied to lupus. When, however, it occurs on other parts than the face, the primary abscess is often due to suppuration of a tuberculous lymph-gland, of which there are not a few too small to be recognised by the anatomist, but apparent when enlarged by the hypertrophy of Hodgkin’s disease or by caseous inflammation.

It is remarkable that the rare papular affection of the skin described by Hebra as lichen scrofulosorum (p. 860), and also the dry, harsh condition called pityriasis tabescentium, are not included in the above account of scrofulides.

The term Scrofula originally denoted a swollen neck, which in some children makes the head pass into the shoulders with scarcely any constriction, as it does in a pig (*scrofa*). This usually depends on a chronic caseous enlargement, with characteristic suppuration and subsequent cicatrices, of the cervical lymph-glands. The word *struma* also meant a swollen neck, and while in England it is used as a vague synonym of scrofula, which had better be discarded, in Germany it is applied to another cause of a chronic swollen neck, namely, bronchocele or goitre.

The caseous degeneration is, as Virchow long ago pointed out, not characteristic; for it may occur in arteries as atheroma, in the skin as xanthelasma, in the middle of tumours and even of cancers. Most cases of caseous disease of the lympharia will, on careful examination, be found not to be idiopathic, but secondary to mucous or cutaneous irritation. If indurated lympharia are discovered, we at once seek for a primary affection in a chancre, if cancerous in a primary tumour or epithelial surface, if suppu-

* *I. e.* nodular, tubercular—not tuberculous, as the two words are used at present.

rating in a primary wound or inflammation of the skin or mucous membrane. In the same way caseous lymph-glands can generally be traced to chronic tuberculous inflammation of the surface from which they receive their lymph. In the neck they are most frequently traceable to the throat with its tonsils and other lymphatic organs, more rarely to the scalp, the teeth, or the ear; bronchial lymph-glands become caseous in consequence of chronic or repeated subacute tuberculous broncho-pneumonia; mesenteric lympharia in consequence of chronic or subacute tuberculous enteritis. These three groups of lympharia in the neck, the thorax, and the abdomen are the principal seats of so-called scrofula, and the reason is probably because the mucous membrane of the fauces, the bronchial tubes, and the small intestine is pre-eminently rich in adenoid or lymphatic tissue. In all genuine cases of scrofula, whether affecting the bones and joints, the lymph-glands or the skin, tubercle bacilli are to be found, and thus the term has become superfluous. Watson's classical account of the two types of scrofulous children left one sceptical of the same morbid disposition showing itself in such opposite ways; and it is now clear that "pretty scrofula" was in most cases tuberculosis, and "ugly scrofula" inherited syphilis.

So that the relation between scrofula and lupus is really that both are tuberculous.

The nodules and granules of lupus contain a bacillus which in form, size, and reaction to staining agents is indistinguishable from that found in phthisical sputum. It is present, however, in small numbers, and sometimes needs prolonged search before its presence is ascertained; but present it always is.

This review of the doctrines of the French school on this subject certainly entirely justifies them; and the accuracy of the descriptions above given (*cf.* Besnier, 'Ann. de Derm. et de Syph.' tom. vi, pp. 1—8).

Nevertheless it is extremely rare to see lupus among the countless victims of phthisis, *i. e.* of chronic tuberculous inflammation of both lungs. Nor, looking at the question from the opposite point of view, has the writer found among patients with lupus, either in hospital practice in London or in the large numbers under Hebra's own treatment, any considerable number of cases of phthisis;* and yet phthisis is remarkably common both in England and in Vienna, so that it has been regarded by English writers as the characteristic scourge of this country, and by Austrian writers as so peculiar to Vienna that its prevalence has been explained by the geological condition of the soil.

Relation to granulomata.—Auspitz—who widely departed from Hebra's classification—puts lupus among what he styles "chorio-blastosen," or anomalies of growth of the corium and subcutaneous tissue. He subdivides this group into simple hypertrophic (*macrosomia*) and paratypical or abnormal growths, which include the granulomata. Here lupus finds a place side by side with leprosy, scrofuloderma papulosa (or lichen scrofulosus), and scrofuloderma pustulosa (or acne cachecticorum), scrofuloderma ulcerosa (or tuberculous ulcers of the skin), tuberculosis cutis (as a separate condition), syphilis, and lastly rhinoscleroma.

This is closely following Virchow's arrangement of tubercle, lupus, and leprosy among the granulomata.

Neisser placed lupus with tuberculosis of the skin and scrofuloderma

* The writer once had a patient, aged thirty, who suffered from lupus for eighteen years, who had hæmoptysis at intervals since he was seventeen, and who then showed the physical signs of phthisis; but such cases are in his experience decidedly rare.

in one division of a group of chronic infectious diseases of the skin, which includes in addition leprosy, syphilis, glanders, rhinoscleroma, and framboesia. He, however, excluded erythematous lupus from the group.*

Lupus may be defined as a chronic deep dermatitis of the granuloma type, depending on the presence of the tuberculous bacillus. But there would be no advantage in forsaking the well-established, familiar, short, and expressive name lupus for that of cutaneous tuberculosis.

Moreover, there are other cutaneous disorders which have more or less close association with tubercle. There are tuberculous ulcers which are multiple, and have neither the aspect, the distribution, nor the course of lupus (p. 985).

Another tuberculous lesion is the wart-like growth known as *Verruca necrogenica*; also Lupus erythematosus (p. 987), Bazin's disease (p. 986), Rhinoscleroma (p. 990), and "Lichen scrofulosorum" (p. 860), which all have more or less close relation to tubercle.

Why tuberculous ulceration of the skin should be so shallow and so slow is hard to say. Perhaps the exposure of the face to cold air may prevent the tuberculous process going on as rapidly as it does in the lungs and internal mucous membranes.

Clinical course and prognosis.—Lupus is one of the most chronic of diseases. It creeps on, usually with an imperfect attempt at healing, sometimes retreating until it almost disappears, and then again advancing with a persistence and rapidity foreign to its usual character. In the end, if left to itself, it probably heals, leaving, however, indelible marks of its presence in hideous scars, contracted limbs, distorted features, or obliterated orifices. It is singularly free both from pain and from irritation, and never affects internal organs. It does not produce secondary caseous inflammation of the lymph-glands which correspond to the affected skin, and never leads to general tuberculosis of the internal organs. Happily it is amenable to the efficient treatment which has been established within the last forty years, so that the prognosis almost entirely depends upon the early recognition of the disease by a skilled practitioner.

Treatment.—Bateman remarks that he knows "no medicine which has been of any essential service in the cure of lupus," and that "it requires the constant assistance of the surgeon." Wilson, in the first edition of his treatise (1842), by a remarkable omission mentions neither the disease nor the name; in the later ones he recommends caustic applications and a prolonged course of liquor arsen. et hydrarg. iodic., *i. e.* Donovan's solution. The usual practice of the earlier English dermatologists appears to have been to use arsenic and so-called tonics. It was Hebra who, regarding lupus, like most other diseases of the skin, as a purely local lesion, resolutely attacked the diseased tissue, and by destroying it produced a healthy inflammation which ended in cure. The determination with which he carried out this method often led to the most remarkable success. Tilbury Fox introduced the Viennese treatment into England, and maintained that the real treatment of lupus consists in destruction of the diseased tissue by caustics. In France, Hardy recommended iodine—one part dissolved in thirty of water with the help of three of potassium iodide. Even this he admits is useless in most cases, and recourse must then be had to stronger caustics, as *chloride of zinc* or *potassa fusa*.

* Plumbe spoke of lupus as a strumous affection. Erasmus Wilson maintained the same relation; Dr Fagge said that "it is apt to occur in scrofulous persons," and Dr Living that "it belongs rather to the scrofulous diathesis."

Often less severe measures suffice, and Hebra himself accomplished admirable results with the solid *lunar caustic*. A strong solution of the same silver salt (a drachm to the ounce) may sometimes be substituted with good effect. The acid nitrate of mercury may also be applied, especially to small and comparatively superficial spots.

A more satisfactory method of treating cases of ulcerative lupus than any by chemical caustic is by *scraping* with the sharp spoon introduced by Volkmann, of Halle.* Chloroform should be given and the whole of the diseased surface scraped away. It is astonishing how boldly a skilful surgeon can use the instrument, employing enough force to remove all the diseased tissue without injuring the more resistant healthy cutis which surrounds it. Indeed, Hebra's use of the pointed nitrate of silver pencil almost converted it into a scraping or mechanically as well as chemically destructive agent. The hæmorrhage produced by these operations is less than would be supposed. While it is almost always necessary to repeat the application of a caustic, one advantage of the scraping is that it is sometimes sufficient after a single sitting, and seldom requires more than two or three. The saving of time as well as of pain to the patient is great.

Caustic potash, applied as it used to be in stick, is not only extremely painful, but even with the greatest care will destroy healthy as well as diseased tissue. Hebra's *arsenical paste* is less destructive, but causes great inflammation as well as pain, and is every way inferior to scraping.

The *pyrogallie acid* introduced by Järisch is probably the next best local application. It should be used as an ointment of 10 per cent., which is better than solution or plasters. It causes, however, considerable pain.

Another plan of treatment, also introduced by Volkmann, and carried out by Vidal and Besnier in France, by Mr Squire and Dr Stowers in this country, is *scarification*, or, as the operation is now performed, minute stabs with a lancet, or an instrument made for the purpose. The section of immense numbers of blood-vessels produces temporary hæmorrhage, but afterwards obliteration of their channels and anæmia of the lupus spots.

The *galvanic cautery*, though sometimes applicable and less painful than would have been supposed, has the same drawback as caustic potash and sulphuric acid—that is, it destroys diseased and healthy tissues alike.

The Paquelin knife combines cutting and burning, and, though still used in Vienna, will probably soon follow similar methods into disuse.

Whatever agent be chosen, it is of paramount importance, for the successful treatment of lupus, to recognise its character as a new growth which must be destroyed. So long as any of the granulations remain it is liable to return. Once rooted out, it is rare for this to happen, or even for it to appear in another part of the skin. Occasionally the knife may be employed to excise part of the tip of an ear or some other circumscribed piece of skin; but scraping or caustic, or the two combined, are in a great majority of cases as effectual, and the results are better. Indeed, when early and thoroughly treated, lupus becomes a manageable disease, and the cicatrices which result are often surprisingly slight.

In the more superficial forms of lupus such vigorous means are generally unnecessary, though wherever ulcers or granulations are seen, their destruction by some means or other is the only thorough method of cure. The milder applications which have been recommended, such as tincture of

* See his paper on "Lupus and its Treatment," translated for the Sydenham Society in 'German Clinical Lectures,' 1876.

iodine, iodoform ointment (half a drachm to an ounce), resorcin ointment (a drachm to the ounce), and strong solution of nitrate of silver (a drachm to an ounce), may probably stop the disease at an early stage. They certainly check its progress, and may be usefully employed whenever more decisive treatment is counter-indicated or postponed.

Since Finsen, of Copenhagen, in 1897, introduced the treatment of lupus by light, considerable, and sometimes brilliant, success has followed it. If solar light is attainable, the actinic rays of the violet part of the spectrum should alone be used, so as to prevent heating and burning of the skin. If the arc-light (50 or 60 ampères) is used, the affected skin is first compressed by a transparent lens, to cause local anæmia, for a sitting of from half an hour to an hour each day. The objections to the treatment are the length of the cure, which needs twelve months more or less, and the expense of the apparatus.

The Röntgen rays have also been used, but with less success and greater drawbacks, particularly the severe pain.

Although local treatment is essential for lupus, and is often sufficient without any other methods, many dermatologists strongly recommend the internal administration of *cod-liver oil*. Even Hebra admitted its value, and used to apply it locally to the sores as well as internally. It is unwise to trust to this remedy without attempting local measures as well; but wherever swollen glands or phthisical symptoms are present, or when want of weight and flabby muscles show malnutrition, *oleum morrhuæ* ought undoubtedly to be given.

Some of the most scaly forms of lupus are said to be cured by arsenic, but if we cannot recognise such transition forms as are called psoriasis-lupus, we may suspect these cases of being really psoriasis, and not lupus at all, just as serpiginous lupus is often difficult to distinguish from syphiloderma, and owing to this difficulty has sometimes been supposed to be cured by iodide of potassium.

Treatment by inoculation.—Injection with Koch's tuberculin has been widely tried as a treatment for lupus, and with almost as unfavourable results as it gives in phthisis. The injection is followed by marked febrile reaction, confirming the conclusion above reached, that lupus is truly tuberculous. The bacilli are not killed, but this is of less importance where they are so few. The granulation tissue becomes more vascular, swollen, and painful, as in tuberculus lungs, lymph-glands, and joints. After repeated injections tolerance is established, and the local excitement subsides.

In some few cases the result is decided improvement. In most there is no visible change, and in some the effect is injurious.

The new tuberculin has been tried with some misgivings, and certainly causes less harm than the original one: whether it does more good is doubtful.

Tuberculous ulceration of the skin.—It may seem contradictory, after the evidence above given, that lupus is itself an ulceration which is accompanied by the characteristic microbe of tubercle, to distinguish another disease which is also ulcerative and also tuberculous. But clinically we are bound to follow the natural history of diseases rather than their anatomy or origin, and those which differ in aspect, course, and treatment demand separate notice.

Lupus, though a deep inflammation, is not exclusively ulcerative; scales,

pustules, and other inflammatory products make up much of the diseased structure; and although tuberculous in nature, it is not often associated with other forms of tubercle in the bones, joints, and lungs. But there has long been recognised an ulcer which is distinct in appearance from lupus, which is more like that of syphilis, and which is characteristic of the condition known as tuberculosis of the lymph-glands and joints, the lungs, and the serous membranes.

Tubercular ulceration is usually multiple; the ulcers are rounded, without the thickened border of lupus, often somewhat undermined, not sloughing or phagedænic, but with pale, large, œdematous granulations. They are situated on the face, trunk, or limbs, most often on the last, and are irregularly placed. They may occasionally be seen on the lips, or about the genital and anal orifices. They are more sensitive than the sores of lupus or of syphilis. They are most often seen in children or very young adults,* and are frequently accompanied by signs of caries, by caseous glands, or by other tuberculous lesions.

The local appearance, the locality, and concomitants are unlike those of lupus vulgaris or lupus erythematosus. Confusion with acquired or congenital syphilis is easier. But the ulcers are not the result of a sloughing gumma; they have a purplish cyanotic, not a brownish, coppery border; they are more painful, and they are accompanied by the concomitant lesions of tuberculosis, and not of syphilis.

The treatment is chiefly internal, by oleum morrhuæ, steel, good food, and good air, with soothing or gently stimulant local applications.

The only other cutaneous lesions which the writer has seen accompanying the ulceration are—the curious dry condition known as pityriasis tabescentium (p. 917), and pustules, which might be called impetigo or ecthyma, scattered over the limbs.

Bazin's disease (*érythème induré scrofuleux*, as he called it, or Latinised as *erythema induratum*), has no relation to Erythema as above defined (pp. 893-5), for it is chronic and ulcerative, and most, if not all of the cases described under this title are better called indolent tuberculous ulcers of the legs, with unusual amount of precedent venous congestion, œdema, and induration. (The best account of it is by Dr Colcott Fox, in the 'British Journal of Dermatology' for 1893, p. 225.) Anyone looking at the coloured lithograph which accompanies this article will recognise the undermined, indolent, cyanotic ulcers of "scrofula"—as unlike lupus as syphilis.

It has been confounded with erythema nodosum, and with gummatous ulcers, but is utterly unlike the former; and though it sometimes looks at first sight like tertiary ulceration, the colour, uniformity, and locality seldom fail to distinguish it, apart from the presence of other tuberculous, and absence of syphilitic lesions.

Although the calf of the legs is the most frequent seat of the disease, it may be found elsewhere about the knees, and occasionally on the upper extremities.

It is most common in girls and young women, and I have only seen it in such patients; but it has been recorded in boys and in women as old as fifty. It is said to be worse in winter.

Its progress is very slow, and the treatment unsatisfactory. Bandaging

* A remarkable case in an aged woman, reported by Dr van Harlingen, of Philadelphia ('Arch. of Derm.,' April, 1879), may perhaps be regarded as a senile and malignant form of this disease.

or elevation of the affected limb, gently stimulating applications locally, and abundant food, with tincture of steel and quinine, are the best treatment.

Under the title *Tuberculosis verrucosa cutis*, Riehl and Paltauf described in 1886 an infectious disease derived from handling the skin of tuberculous animals, either dead or alive. It is intermediate between tuberculous dissection-warts (*verruca necrogenica*—Wilks, 'Guy's Hosp. Rep.' vol. viii, p. 263), lupus verrucosus, and tuberculous ulceration.

Lupus-erythematosus.*—The essential nature of this somewhat rare disease still admits of doubt. There is no question that the sebaceous glands are early, if not originally its seat; it is equally certain that a slow chronic dermatitis, accompanied with a violet or rose-tinted erythematous blush, is always present. One seldom fails to discover evidence of a destructive process in the papillary layer; usually as a smooth, thin, atrophied aspect of the affected skin, and sometimes as obviously cicatricial patches.

It is still usually associated with lupus, as above described, the two forms being distinguished as *lupus vulgaris*, *exedens*, or *exulcerans* on the one hand, and *lupus erythematosus*, *erythematodes*, *sebaceus*, or *non-exedens* on the other.† It, however, differs from *lupus vulgaris*, in the age of the patients, in its locality and course, and in the absence of tubercle-bacilli.

Some good observers regard true lupus and this affection as altogether distinct and unrelated both in pathology and in clinical course; but totally different as a severe case of ulcerated lupus of the face appears when compared with one of the disease under discussion, there are not unfrequent instances of the two being apparently connected by such as those named by Leloir *Lupus vulgaire érythématoïde*. Lupus erythematosus in some cases simulates nævus. See the account of patients under the care of Mr MacCarthy and Mr Higgins, given by Mr Hutchinson in his 'Lectures on Clinical Surgery,' vol. i, p. 284. This does not, however, correspond with the telangiectic variety of Lupus erythematosus described by Crocker. The latter writer also distinguishes a rare nodular form which approaches common lupus in character.

The *locality* of this affection is very characteristic. It almost always occupies the face, and usually the bridge of the nose, together with both cheeks; for, in contradistinction to ordinary lupus, it is remarkably symmetrical. The figure produced by this distribution has been compared to a butterfly, a bat, or the sphenoid bone, and when once seen is easily recognised. Lupus erythematosus is also found on the ears, and sometimes on the scalp. The hair is then destroyed, a sufficient proof that lupus erythe-

* *Synonyms*.—This curious affection was first described by Bielt and named *Érythème centrifuge*. It was called by Hebra *Seborrhæa congestiva* (1845), and the same view of its nature has led to the titles *Lupus sebaceus* and *Lupus acnéique* (Hardy). It has also been named *Scrofulide érythémateuse* and *Lupus de Cazenave*. It is, however, more generally recognised by Cazenave's name, Lupus erythematosus (1850). Unna has proposed the title "ulcerethema centrifugum."

Certainly this disease is not a mere form of Lupus, like *Eczema madidans*, *Psoriasis guttata*, *Lepra anæsthetica*, but needs the adjective to give it any meaning, like *Lichen-planus* or *Pityriasis-rubra*.

† In favour of this view see Mr Hutchinson's 23rd lecture ('On Certain Rare Diseases of the Skin'). For arguments in favour of a more complete severance of lupus erythematosus from true lupus see Kaposi's and Veiel's papers ('Trans. Intern. Med. Congr.,' vol. iii, pp. 162, 167), with comments by Schwimmer and Thin; also Dr Payne's remarks (St Thomas's Hosp. Rep., vol. xiii). The contrast of opinion on this point is exhibited in the text of the French translation of Kaposi by Besnier and Doyon compared with the notes of the learned translators.

matusus is not, as it was classed in 'Ziemssen's Cyclopædia,' a superficial dermatitis. It occasionally appears upon the limbs, sometimes preserving its symmetry, but sometimes being confined to one hand. On the trunk and legs it is certainly rare, but in one patient of the writer's it spread over the shoulders and buttocks, and in one or two recorded cases it has covered trunk and face and limbs. Two separate patches are far more often seen than in ordinary lupus.

It is seldom that we see the beginning of this disease. It shows itself as an erythematous patch, not unlike an early stage of *tinea circinata*. It spreads at the edge (whence Biett's epithet *centrifuge*), which is marked by injection, swelling, and desquamation, while the centre becomes pale, smooth, and slightly depressed. The sebaceous glands are enlarged, sometimes prominent, resembling *acne punctata*, sometimes forming black comedones within the affected surface. It thus spreads until it has attained the form and dimensions above described as characteristic. Sometimes, however, fresh spots occur at a distance, and this is decidedly more frequent than with ordinary lupus. The small injected patches are usually covered with minute scales of dried sebum, which suggested the epithet *herpes crétacé* to Devergie (p. 980).

On making a microscopic section of the diseased skin, dilated blood-vessels are found, and infiltration of the cutis with leucocytes. The congestion and proliferation is most abundant around the sebaceous glands. The exudation-cells never become caseous, or soften down so as to form the granulations and pus of an ulcer. They gradually become transformed into connective-tissue corpuscles; and as the fibres thus formed take their place, the papillæ atrophy and the glands shrink and disappear. These histological characters appear to show that no sharp line of distinction can be drawn between chronic deep-seated inflammation with hypertrophy and consecutive atrophy on the one hand, and development of such simpler forms of new growth as lupus, tubercle, and syphilis. On the other hand, there is a clearly marked line between deep inflammations with destruction and atrophy on the one hand, and superficial inflammations which do not destroy the papillæ and are never followed by ulceration or cicatrices on the other.

No tubercle bacilli nor other specific microbes are found in the diseased skin. Nevertheless, as Besnier and Hutchinson first noticed, phthisis is more frequent in the families of patients suffering from *L. erythematosus* than in those of *Lupus* itself or than the frequency of phthisis would explain.*

Lupus erythematosus occurs chiefly in young adults, but sometimes in children and aged persons. Most of the patients are between twenty and forty, and the average age of this affection is certainly later than that of ordinary lupus. It is commoner in women than in men.

The diagnosis of *Lupus erythematosus* in the most characteristic discoid form is usually easy. But it may simulate *lupus vulgaris*, *nævus*, ringworm, *acne*, *psoriasis*, and chilblains on the hands and ears.

Its course is extremely chronic, with liability to occasional exacerbations. Like ordinary lupus, it is accompanied by neither pain nor itching.

The *treatment* of erythematous lupus is seldom satisfactory. Alteratives of a stimulant kind do more good than caustics. Hebra's diachylon oint-

* See Boeck's paper in the New Syd. Soc. vol. for 1900, and the opposing view expressed by Unna ('Brit. Journ. of Derm.,' 1898, p. 373).

ment or solution of soft soap (sp. sap. alk.) is sometimes sufficient. Iodide of mercury ointment (one to fifteen) was recommended by Cazenave, and pyrogallic acid is sometimes useful; but the effects of iodoform ointment (gr. xv— $\bar{3}j$) have in the writer's experience been better. In some cases mild applications can alone be borne. In others, again, the treatment which dermatologists, whatever name they give it, are led to adopt is the scarification, or scraping, or the galvanic cautery used against Lupus.

Mr Hutchinson strongly recommends the continued use of an ointment consisting of half a drachm of liquor picis carbonis to an ounce of vaseline; salicylic acid 5 p. c., or resorcin 3 p. c. in collodion. The ung. liq. picis carbonis ($\bar{3}ij$ — $\bar{3}j$) of the Guy's Pharmacopœia is stronger, and is also a useful remedy. Ichthyol is also recommended by some writers, but its alleged efficacy, whether used internally or locally, makes one sceptical of either. Internal treatment by arsenic has never seemed to the writer to be effectual; but some good observers speak highly of quinine, and others of salicylate of soda. (See an admirable paper by Prof. White, of Harvard, 'Journ. Cut. and Gen. Skin Dis.,' Oct., 1898; and Unna's list of remedies, 'Brit. Journ. of Derm.,' vol. x, p. 374.)

Finsen's light treatment has been employed in cases of erythematous lupus, and in some cases with good results (p. 985).

Lupus erythematosus disseminatus.—Kaposi named lupus erythematosus as above described *discoïd*, in order to distinguish it from a rare and remarkable form of disease, which he regards, and probably with justice, as nearly related. The latter he has named the "disseminated" or "aggregated" variety of lupus erythematosus. Here the patches do not grow by the enlargement of the circumference, but by fresh ones appearing. Moreover the disease is not confined to the face, but is seen upon the trunk; the course is sometimes acute, and the whole character of the disease is far more severe than that of ordinary erythematous lupus, or even of lupus exedens; there is considerable pain, and sometimes synovitis; there is high temperature, with nervous symptoms which sometimes end in coma, and in not a few cases the result has been fatal. Cæsar Boeck saw two well-marked cases of this curious disease in Norway.

The acute form is, however, the exception. More often the disease persists with more or less frequent exacerbations, the face appearing as if affected with constant erysipelas. Here also the end is usually death, either from marasmus or from an intercurrent disease.

The writer has only seen one example of this remarkable affection, which occurred in the practice of Dr Cavafy at St George's Hospital. The patient was a woman between thirty and forty; the affection occupied not only the face, head, and neck, but the greater part of the back and trunk. It looked like erythema of a somewhat gyrate form, but there was unquestionable scarring. The patient succumbed to pneumonia.

Rhinoscleroma.—This uncouth epithet was applied in 1870 by Hebra and Kaposi to a newly recognised form of disease—a hard, smooth infiltration or new growth of the septum of the nose and the adjacent tissues of the alæ nasi and of the upper lip. It has a general resemblance both to lupus and to syphilis, but is said not to be prone to ulceration—a characteristic which would also distinguish it from epithelial cancer. It may be regarded as a granuloma, and so far allied with lupus; but no tubercle

bacilli have been discovered, and only in some cases diplo- or streptococci.

Mr Hutchinson has not seen any case which corresponds with the fourteen or fifteen seen in Vienna, but thinks he has observed cases of lupus which by their unusual hardness and other characters approached rhinoscleroma. He showed a case in an old woman of sixty-eight to the Dermatological Society in April, 1883. A few additional cases have been published in Austria, reference to which will be found at p. 496 of Hans von Hebra's 'Krankhafte Veränd. d. Haut.' The writer has seen some cases which may have been rhinoscleroma, but only one which was certain. The first occurred before 1870, in a young man at Vienna, which was diagnosed and treated as tertiary syphilis; it consisted in "a thickening and stony-hard induration of the nose," which had lasted for six years when seen. A second case seen a few months later, recorded as one of "syphilitic sclerosis of the nose," not nearly so hard, and accompanied by redness and ulceration, is more doubtful. A third was seen at Guy's Hospital, in November, 1886, in a man of thirty-five; there was dense induration of the upper lip, with surrounding œdema and a little superficial ulceration; it had lasted several years. He also saw the typical and very remarkable case in a patient of Dr Payne's, a young man from South America, which will be found described and figured in the 'Path. Trans.' for 1885. Here the palate and larynx were also affected; and the local ulcerative condition and histological characters were not unlike those of some forms of sarcoma. Dr S. Davies has recorded a well-marked instance from Egypt ('Brit. Med. Journ.,' May 29th, 1886).

Rhinoscleroma not only attacks the nose and lip, but may spread to the gums, tongue, palate, and even to the pharynx and larynx.

The ivory-like induration, the singular locality, and the absence of ulceration separate it from lupus; and von Frisch discovered a bacterium not identical with that of lupus ('Ziemssen's Hdbh.,' xiv, 713). Since then Cornil and Alvarez and others have seen it ('Ann. de Derm. et de Syph.,' vi, No. 4, 1885). It is figured in Dr Payne's 'Manual of General Pathology' (p. 672, and *front.*, fig. 1). It occurs in twos or fours and encapsuled.

The histological characters are not distinctive, for Kaposi found only infiltration of the cutis with very minute leucocytes. Geber recognised giant-cells and spindle-cells ('Arch. f. Derm. u. Syph.,' 1872). Payne found large nidus-cells beside granulation tissue, and "nests" like those of epithelial cancer. In a doubtful case, brought by Mr Marrant Baker before the Pathological Society in 1881, Mr Hutchinson, Dr Cavafy, and the writer were appointed a committee, and drew up a report, which will be found at p. 262 of the 'Transactions' for that year. A figure is given at p. 458 of 'Ziemssen's Handbuch' by Schwimmer and Babes.

Rhinoscleroma has returned after removal in cases reported from Germany and from Italy; but is said to have been favourably influenced by the injection of an alkaline solution of salicylic acid as a germicide.

It may be primary in pharynx and larynx. Hence some modern Germans call it "Sclerom," not Rhinosclerom (see Payne's article in 'Allbutt's System').

LEPROSY*

“Est Elephas morbus qui propter flumina Nili
Gignitur Ægypto in media, neque præterea usquam.”

LUCRETIVS, *De Rerum Nat.*, lib. vi, 1112.

History and terminology—Geographical distribution—Anatomical lesions and course—Histology—The Bacillus lepræ—Symptoms and event—Ætiology—Treatment—Other exotic diseases—Frambæsia—Acrodynia, etc.

THIS disease, interesting from an historical point of view, is still of practical importance in many parts of the world. Leprosy is much more than a disease of the skin, for it involves nerves and joints and bones; but its first symptoms are cutaneous, and, like syphilis, it may be suffered to remain as a chapter in dermatology.

Nomenclature.—The name given to the disease by the Greeks was *elephantiasis*; and it was divided into *alphos*, *melas*, and *leukos*.

Celsus, however, who describes *alphos*, *melas*, and *leuce* as species of *Vitiligo* (lib. v, cap. xxviii, § 19), portrays leprosy separately and distinctly as a disease affecting the bones and the whole body, and almost unknown in Italy, “*quem ἐλεφαντίασιν Græci vocant*” (lib. iii, cap. xxv).

The term applied by Willan and Bateman to leprosy was “*elephantiasis Græcorum*,” while they unfortunately used “*lepra*” for part of the innocent white scaly disease which the ancients would possibly have called *lepra* or *alphos*, but which all modern dermatologists call *psoriasis*.

No doubt many other cutaneous affections, obstinate chronic eczema, syphilis, lupus, and psoriasis, were confounded with leprosy in ancient times; but there is no question that one and the same destructive form of disease has existed in Palestine under the Mosaic law, in Western Europe during the Middle Ages, and at the present day in many other parts of the globe; and this is best named by its historical title, leprosy.

* *Synonyms.*—*Lepra vera*—*Lepra Arabum*—*Elephantiasis Græcorum*—*Leontiasis*—*Satyriasis*—*Morbus Herculeus*.—*Fr.* La lèpre.—*Germ.* Aussatz.—*Norweg.* Spedalskhed.

It must be remembered that the terms *Elephas* and *Elephantiasis* do not primarily refer to rough skin or huge and shapeless limbs, but to the magnitude of the disease. “*Elephantiasis a magnitudine et diuturnitate nomen accepit*” (Aëtius).

“Est lepræ species elephantiasisque vocatur,

Quæ cunctis morbis major sic esse videtur,

Ut major cunctis elephas animantibus extat” (Macer Floridus, 1160).

Aretæus says it is called *elephas* partly because it is unlike anything else, partly because it is black and terrible, and partly because the skin is rough and cracked (‘*De Morb. Chron.*,’ lib. ii, cap. xiii).

Leprosy appears to have been rare in ancient Greece, and it seems to be not certain that the Septuagint translators were correct in rendering *zaraath* of the Hebrew Scriptures by the Greek word λέπρα. The latter term, however, is constantly applied to leprosy in the New Testament. It refers to the scaly surface often seen. The Arabic name of true leprosy, according to Dr Greenhill, is *Judzam* (= *lepra Arabum*). *Barat* (= *leuce* = *vitaligo*), or "white leprosy," is nothing but leucodermia. In the Middle Ages leprosy was known to the school of Salerno as *mal morto* and *mal di San Lazzaro*.

Distribution.—Norway is the only European country in which leprosy is still common; there were 900 lepers in the asylums at Bergen and Molde in 1884; it is there known as *Spedalskhed*. It is also found here and there in Sicily and in Malta (28 lepers were reported in the latter island in 1886), in certain parts of Portugal, in the Levant, in the Crimea, and at Astrakan; it is more common in Syria, Arabia (Palgrave), Persia, Bengal, S. India, Burma, and Siam; in Japan and in China, where it is said to have been known for ages; in Egypt, Nubia, the Soudan, the Cape Colony (where it co-exists with elephantiasis Arabum), and on most parts of the African coast (though apparently it is rare in the interior); in Madagascar and the Mauritius, St Helena, the Canary Islands, and the Azores; in New Brunswick, Mexico, and the West Indies (especially Trinidad), Central America, Ecuador, British Guiana and Surinam, Bahia, and the coast of Brazil; in New Zealand, the Sandwich Islands, and some other parts of the Pacific.*

Unhappily, while leprosy has receded from civilised Europe during the last four hundred years, there is reason to fear that it is reappearing in new countries. Thus it has been introduced within historical times into the Sandwich Islands, and within the last twenty years cases have been reported from various parts of the United States, and from Australia. In Minnesota it appears to have been introduced by Norwegian immigrants, in New South Wales by Chinese labourers; but this explanation of its spread, whether by contact or inheritance, does not apply to certain isolated cases observed. For instance, Dr George Dock sent the writer in 1889 a careful account of two cases, one in a German, the other in an Alsatian, which occurred at Galveston on the coast of Texas, neither of whom had had intercourse with lepers from Mexico, China, or South America. It had not spread to the wives or families of either of these patients.

Leprosy has not been observed in any of the lower animals.

Varieties.—Leprosy is essentially one and the same disease, but one of two forms is usually predominant—the *nodular* or "tubercular" (more common in Norway and less so in the tropics), and the *anæsthetic*. The two, however, are often combined. Either may be preceded or accompanied by pigment spots, which have led to a third species being formed—*lepra maculosa*. All end in an ulcerative stage, and all may lead to loss of members—*lepra mutilans*. "Black leprosy" is the only genuine form; "white leprosy" is not leprosy at all, but leucodermia.

Anatomy.—The disease begins insidiously, usually as an erythematous redness, but in some cases with an outbreak of bullæ resembling those of

* See Dr Ransome's maps and statistics ('Brit. Med. Journ.,' March 1st, 1890), and Dr P. S. Abraham's pamphlet with a map (Epidemiological Soc., 1889); also a report on leprosy by the College of Physicians which was issued as a blue-book in 1867. In 1874 Vandyke Carter published an official report upon leprosy in India, and more recently Beaven Rake contributed valuable clinical and pathological information on the subject from Trinidad, and Hillis from Guiana.

pemphigus. There follows the appearance of red or violet patches, varying from a finger-nail to the palm of the hand in size, and gradually becoming darker in colour. At the same places, or independently, appear flat, firm, raised nodules, consisting of an infiltration of the deeper parts of the skin. The lymph-glands at the same time enlarge. These nodules of tubercular leprosy may shrink and be absorbed, leaving atrophied and sometimes pigmented spots; but more often they soften and ulcerate. The leprosy ulcers secrete but little pus, they show few and feeble granulations, but they slowly increase both in extent and depth.

Localization.—The leprosy patches usually appear first on the face or limbs, and afterwards on the trunk. When fully developed in the face the disease produces a singular deformity, which the ancients described as like the face of a lion (*leontiasis*) or a satyr (*satyriasis*), and which, once seen, is never forgotten. The disease also affects the neck, shoulders, back, chest, and abdomen, but is most frequent in the extremities, especially on the extensor surface. Nodules occasionally occur upon the palm and sole. The hands and feet are swollen and distorted, with thickened and rough skin; the ulcers burrow deeply, and invade tendons, bones, and fibrous tissues, until at last toes, fingers, or the entire hand or foot undergo gradual necrosis and fall off. The scalp and genitals are very seldom attacked.

Some of the mucous membranes are also affected, particularly those of the mouth, nostrils, and larynx, and even the conjunctivæ. Moreover the disease involves the great nerve-trunks—where the leprosy nodules can often be felt during life—as well as the liver, spleen, and other viscera.*

Histology.—Microscopical investigations showed that the disease consists in infiltration of the deepest layers of the cutis with granulation tissue. Leprosy was therefore classed by Virchow in proximity to lupus, from which, however, it is widely separated by its clinical course, geographical distribution, and natural history.

A bacillus was discovered by Hansen, of Bergen, in 1874, which he described and figured in the 'Quart. Journ. of Micr. Sci.' for 1880 (vol. xx, p. 92).† These microbes appear constantly in leprosy nodules, and in great abundance. The *Bacillus lepræ* is 5 μ long, very slender, and immobile. It stains like the bacillus of lupus, *i. e.* of tubercle (see Crookshank's 'Bacteriology,' pl. 23). Attempts at cultivation have hitherto failed.

Diagnosis.—The signs of leprosy are well marked and characteristic: but it must be distinguished from Lupus by its extent, its nodules, and its anæsthesia, and from Syphilis by its uniformity and signs of neuritis. This peripheral neuritis, with ulceration and bullæ, makes sometimes a close approach to the symptoms of syringomyelia; and probably some cases of "Morvan's disease" were really leprosy (vol. i, p. 633, *note*). The blood in leprosy is not characteristic. At first the erythrocytes are normal or excessive in number; afterwards there is secondary anæmia without leucocytosis (von Limbeck).

Course.—Leprosy is sometimes preceded by prodroma of the invasion of the infection—headache, nausea, and other febrile symptoms. The dis-

* Schäffer, 'Lepra,' vol. i, p. 11, with plates.

† They have since been found by Doutrelepont, Neisser, Cornil and Babes, Köbner, Dr Hillis ('Path. Trans.,' 1883, pl. xxii), Dr Thin ('Med.-Chir. Trans.,' vol. lxxvi), Dr L. J. Steven, of Glasgow ('Brit. Med. Journ.,' ii, 1885), Drs Klein and Gibbes, Dr Rake ('Path. Trans.,' 1887), and all modern observers. See Dr Thin's monograph on Leprosy, 1891.

ease, when established, is extremely slow in its progress, and it resembles syphilis and lupus in producing but little pain. Patches of anæsthesia are generally to be found, and may be followed by ulceration before tubercles appear. It is said that in rare instances hyperæsthesia precedes or takes the place of loss of sensibility. The anæsthetic spots usually show some amount of atrophy, and the hairs of those parts are small and deficient in colour. The anæsthesia and bullæ and ulceration are due to peripheral neuritis of the nerve-trunks.

While this terrible disease goes on its course, interrupted from time to time by temporary improvement and healing of the ulcers, but never more than checked, the general condition of the patient is wonderfully little affected. Even perspiration takes place much as usual. The hair, however, is gradually lost, not only that of the scalp, but also the beard, eyebrows, and eyelashes. There is no fever, the temperature is usually subnormal, and the patient suffers much from cold. The pulse is slow, and the appetite and organic functions, including the quality of the urine, are very little altered. There appears to be no foundation whatever for the assertion of the ancient physicians that the sexual instinct is increased; perhaps the name *satyriasis*, first applied to the distorted and hideous features of the sufferer, was afterwards misinterpreted.

Death seldom occurs directly from leprosy, for there is neither excessive pain nor hæmorrhage nor invasion of vital organs to cause it; but when once fallen into a condition of anæmia and marasmus, the miserable leper is cut off by some intercurrent affection—pleurisy, pneumonia, dysentery, or Bright's disease, all of which have been recorded by the Norwegian pathologists Boeck and Danielssen, but none with sufficient frequency to show more than an accidental connection with leprosy. The patients become dropsical in the later stages; and they are often cut off by phthisis, in which the leprous bacilli appear to take the place of those of tubercle.*

Ætiology.—The cause of the former prevalence and present restriction of leprosy is entirely unknown. It has probably existed from the earliest times, and has only disappeared from civilised Europe within the last 400 years. We may hope that it is in slow but steady process of extinction in other regions.

Notwithstanding the presence of the *Bacillus lepræ*, the disease is not, under its usual conditions, contagious; it is not transmissible by living in the same house, by contact, or even by sexual intercourse. It is, however, possible that contact of actually ulcerating leprous nodules with a fissured skin or mucous membrane might produce the disease, and there is reason to believe that a contagious quality is more marked when the disease is newly introduced. Köbner and Rake were unsuccessful in attempts to propagate it in animals by inoculation. A case of supposed inoculation in a man in 1886, at Honolulu, appears to be inconclusive. Sir Wm. Gairdner, however, published a case which seems to show that leprosy may be inoculated by vaccination ('Brit. Med. Journ.,' February 5th and June 11th, 1887).

Whether or not it is under any circumstances contagious, leprosy is still believed to be *hereditary*. Its occurrence in persons of pure European parentage is excessively rare. Patients in England are usually either half-castes or persons who were born and lived in India, and one of whose parents

* Some lepers survive to a considerable age. Thus at the Lepers' Home in Jamaica in 1901 a woman had been an inmate for sixteen years; and another, who had lost her hands and feet, was probably sixty years old ('Guy's Hosp. Gazette,' February 15th, 1902).

was perhaps of mixed blood. The Norwegian emigrants to Wisconsin and Minnesota included 160 lepers, but the disease was found to have died out among them by Dr Hansen, of Bergen ('Arch. f. Derm. u. Syph.,' 1889).

It is doubtful whether leprosy has any predilection for castes or races as such, although at the present day it is, as above stated, almost confined to certain of the dark races of mankind, and where prevalent is rare among the well-fed and well-cared-for classes.

Mr Hutchinson believes that leprosy depends upon eating fish, probably fish in a state of decomposition. This view certainly agrees with its presence not only on the sea-coast, but also in the neighbourhood of great rivers and inland lakes; and it also accords with the large consumption of salt fish in the Middle Ages, when it formed the principal animal food throughout the winter. The disease, however, does not appear in many parts where fish, both fresh and putrid, is eaten, and it is prevalent in certain districts where fish do not form an article of diet. (See Dr Abraham's paper in the 'Practitioner,' 1889, and the discussion at the Congress at Berlin in 1897.)

Leprosy appears to be more common in men than in women—in Bombay, according to the late Dr Carter, very much so. It usually begins about the time of puberty or in young adults. No congenital case appears to have been recorded.

Treatment.—This is unfortunately almost hopeless, and we must rather look to the gradual rooting out of the disease by improved conditions of life, and by segregation of cases as they occur, than to therapeutics. Various drugs have been vaunted from time to time as specifics, but have all in turn been discredited. Cod-liver oil is the only internal remedy which can be said to do more than alleviate symptoms. Externally Gurjun and Chaulmoogra oils have been supposed to be valuable. The writer has tried the former in three cases with no benefit. Dr Liveing's much larger experience makes it probable that the latter is sometimes of service. Excision and scraping of the leprous nodules was recommended by Dr Rake.

Injections with tuberculin have proved useless and dangerous; but Haslund and Crocker have seen benefit from intra-muscular injections of perchloride of mercury ('Derm. Zeitschrift,' vi, 1, 1901).

Mr Hutchinson has recorded a case of gradual spontaneous recovery ('Med.-Chir. Trans.,' lxii, p. 331); and the writer once had a somewhat similar case of a man who developed leprosy after living in Brazil, and became very much better in England. But temporary quiescence of the disease is very common.

Leprosy is the only exotic disease of the skin of practical importance to practitioners in England.

Framboesia or Yaws, apparently a contagious malady, and by some authors believed to be nothing but Syphilodermia, was known to Bateman, and was described at length by Kaposi in Hebra's 'Handbook.' The best account in English is in Numa Rat's monograph (1891). Yaws is endemic on the West Coast of Africa, and appears to be identical with what is known as *Pian* in Java. Less clear is its relation to *Parangi*, a cutaneous disease endemic in Ceylon, and to *Verrugas* in Peru. An account of parangi by Dr Christie appeared in Dr McAll Anderson's 'Diseases of the Skin.'

Radesyge in Norway is, according to Hebra, lupus. *Sibbens*, the old Scottish name of a disease of the skin, was syphilis.

Aleppo evil, the Oriental sore, known also as *bouton d'Alep* or *de Biskra*, and the Penjdeh or *Delhi boil*, have been ascribed to syphilis, but without proof. It is now said to be dependent on a special bacterium and capable of inoculation in animals (Heidenreich, 'Centrbltt. f. Bact. u. Parasitenkunde,' January 25th, 1889).

Pellagra, an epidemic erythema, was first observed in Lombardy, and connected with eating diseased maize; it seems to be identical with *Acro-dynia*, described by Alibert as epidemic in Paris during 1828 and 1829. Winternitz ("Eine klinische Studie ü. das Pellagra," 'Vierteljahresschrift f. Derm. u. Syph.,' 1876) doubts the existence of the former. *Acro-dynia* seems to be endemic in the Levant (Behrend, 'Hautkrankheiten,' p. 154).

The best account of *Pellagra* in English is given by Dr Sandwith, who has studied it in Egypt ('Brit. Journ. Dermat.' for 1898, vol. x, p. 395).

Ringworm appears in peculiar forms in certain foreign countries. Burmese ringworm has been already referred to (p. 937); and Dr Anderson has published an interesting account, with figures, of *Tinea imbricata* from Tokelau, in the South Seas ('Edin. Med. Journ.,' Sept., 1880).

NEW GROWTHS

"Il y a des blessures, dont . . . la cicatrice reste."

VOLTAIRE, *Tancred*.

Cheloid—Terminology and history—Appearance, course, and symptoms—Histology—Relation to scars—Distribution—Prognosis and treatment.

Multiple fibroma—Anatomy and distribution of the tumours—Their course and treatment—*Neuroma*—*Myoma*.

Molluscum contagiosum—Distinction from fibroma and from sebaceous cysts.

Adenoma—*Colloid milium*—*A. sudoriparum*—*A. sebaceum*.

Angioma or vascular *nævus*—*Elephantiasis teleangiectodes*—*Lymphangioma*—*Angioceratoma*.

Mycosis fungoides—Anatomy, clinical characters, and course—Precedent dermatitis.

Kaposi's xeroderma maligna—age—primary condition—later course.

Sarcoma, *carcinoma*, and rodent ulcer of the skin.

PASSING from the granulomata, with deep intractable ulceration, of lupus and leprosy, we now come to the new growths or tumours of the skin in a more restricted sense.

The relation between chronic inflammation, hypertrophy, and new growth is so close, that lupus, tertiary syphilis, and leprosy might be classed either as deep destructive forms of dermatitis or as cutaneous granulomata; while condylomata, gutta rosea, xanthelasma, and elephantiasis are almost as much new growths as inflammations. But we have now to treat of neoplasms which are neither hypertrophies nor inflammations nor granuloma.

As in other parts of the body, the tumours of the skin are clinically "innocent," "malignant," or "semi-malignant;" while anatomically they are distinguished as "homologous" or "heterologous" (*cf.* vol. i, p. 71).

CHELOID.—In the 'Arbre des Dermatoses' of Alibert appears, among many other fantastic names, a new term for what was an undescribed disease—*Kéloïde*. The etymology of the word was long a puzzle. It was supposed by some to be derived from *κηλῖς*, a mark; by others from *κήλη*, a tumour, as in *hydrocele* and *sarcocele*. Being taken by Addison in the former sense—*quasi ustione facta macula*—as meaning a scar from a burn, it was transferred to the curious affection long known as "Addison's keloid," but better named *morphœa* or *circumscribed scleroderma*, described above (p. 961). The late Dr Fagge made clear that Alibert was not thinking of either *κηλῖς* or *κήλη*, but meant by the word "*kéloïde*" to denote the claw-

like offshoots which characterise the disease in question, and intended to derive it from *χρλή*, a crab's claw. The right spelling is now generally used, and Alibert's is recognised as the only "true" cheloid.

Alibert described it as "cancroïde," and Bazin and other French dermatologists have hence called it malignant, and regarded it as closely allied to epithelioma of the skin; but it is possible that by "cancroïde" Alibert did not mean "cancer-like," but "crab-like;" at all events, it is not a cancrroid tumour in the modern sense of the word (vol. i, p. 93).

Bielt and Lebert afterwards published cases; Addison gave an account of Alibert's disease in the 'Med.-Chir. Trans.' for 1854 (reprinted in his 'Collected Works'), and a paper by Dieburg appeared in the 'Deutsche Klinik' for 1852, No. 33. Since that time cheloid, though a rare disease, has been observed and described both clinically and histologically in all parts of Europe and America. The 'Clinical Transactions' for 1880 contain a remarkable case of multiple cheloid of the face following smallpox by Dr Goodhart, and a commentary by a committee on the subject. See Dr Walter Smith's paper ('Brit. Journ. Derm.,' 1157), and De Amicis, *ibid.*, p. 408.

A series of excellent models of cheloid, Nos. 454 to 466, were made by Mr Towne for the Guy's Hospital Museum.

Anatomy and course.—The affection begins as a pink, smooth, slightly raised, flat nodule, which increases in extent without becoming relatively more prominent. It is remarkably firm in feel. The centre becomes paler, and is sometimes depressed, while the raised edges are surrounded by a slightly injected border; the epidermis is completely adherent; it is in and not under the skin. Sometimes, however, especially in the later stages, it spreads to the subcutaneous tissue and forms adhesions to the deeper parts, but it never invades more than the integument.

The most characteristic part of the disease is the presence of radiating bands, which appear after a time, run across the original nodule, and afterwards project from its edge. These are the claw-like processes observed by Alibert. They undergo contraction in the same way as the cicatrices of a wound, and the whole tumour is sometimes puckered and deformed by this process. In the earlier period the nodule might pass for an hypertrophied scar; in the later stages it still more closely resembles a large indurated and contracted cicatrix, as from a deep burn or a syphilitic ulcer or a carbuncle.

The tumour is usually single, but two or more may exist on the same patient, as in Dr Goodhart's case, and in a young man under the writer's care in 1888. The disease is of very slow growth. It occurs most often in young adults of either sex. From the commencement it is usually attended with pricking and itching, with a sense of constriction, and sometimes there are severe stabbing pains. It is almost always tender to the touch, but in certain cases is quite free from pain.

Histology.—Microscopic sections show that the epidermis is thin, but otherwise unaffected; the papillæ are destroyed, and the cutis vera and subcutaneous tissue occupied by bands of dense fibrous tissue, which are quite indistinguishable from those of a true scar; only in the peripheral parts of the growth is small-celled tissue to be found together with spindle-cells, the stages no doubt which precede the fibrous scar-like texture of the mass of the growth. As in all cicatrices, the sweat-glands, hair-sacs, and sebaceous sacs are destroyed in the process. Dr Warren, of Boston, published a valuable histological account of cheloid in the 'Transactions of

the k. k. Akad. d. Wissensch.,' Vienna, March, 1868. See also that by Babes in 'Ziemssen's Handbuch,' xiv, p. 434, and by Hyde and Montgomery (1901), p. 541.

Locality.—Cheloid tumours occur most frequently in the skin over the sternum. They have also been observed on the abdomen, neck, shoulders, arms, and face. They are usually single, and very rarely more than two in number, except in the case of cicatricial, so-called false cheloid, when the new growth may appear in as many scars as were originally present.

We had once in hospital a well-marked case of cheloid affecting the pubes in a patient of Mr Bryant—a man who probably had never had ulceration, syphilitic or other, of this part.

Diagnosis.—Neither in the histology nor in the symptoms does there seem to be any obvious distinction between a cheloid tumour and an hypertrophied and painful scar. Hebra, in fact, defines cheloid as an idiopathic or primary cicatrix. Others have maintained that all cheloid tumours are hypertrophied scars; and undoubtedly they often arise from ordinary cicatrices, or from the slight marks left after leech-bites or acne pustules. They often occur on the shoulders, where the cicatrices of acne are usually the deepest and most extensive.*

Dr Robert Liveing, while admitting that cheloid growths often begin in scars, finds the distinction between them and hypertrophied cicatrices in two points: first, that the bands of fibrous tissue in cheloid run in definite parallel or radiating bundles, whereas those of cicatrix form an irregular network; secondly, that the cheloid growth invades healthy tissues, which hypertrophied scars never do, and that this is the case even when cheloid appears in a previous scar. The new growth can be distinguished as it invades the old cicatricial tissue.

Pathologically true cheloid appears not to be a mere hypertrophy nor a granuloma, but a fibro-cellular new growth, a true sarcoma, at first consisting chiefly of spindle-cells, subsequently more exclusively of fibres. It is very apt to return after removal, but it does not reappear in the neighbouring lymph-glands or in the viscera.

Traumatic or false cheloid is an hypertrophied scar ("die warzige Narbengeschwulst" of Dieburg). It occurs whenever a burn, ulcer, or other injury produces a scar which hypertrophies and becomes painful, and its structure is that of pure fibrous tissue.

Prognosis and treatment.—Trustworthy observers have recorded the spontaneous disappearance of cheloid tumours, but this must be extremely rare. They seldom or never ulcerate. They grow slowly, and appear not to menace life, but the pain they occasion is sometimes severe.

Unfortunately, if removed by the knife, by galvano-cautery, or by caustics, the tumours almost always return. Nor have any of the milder applications which have been tried produced absorption. Mr Hutchinson, however ('Medical Times,' May 23rd, 1885), recorded exceptional cases in which operation proved successful. Hyde and Montgomery recommend the application of oleate of mercury with morphia.

FIBROMA.†—This affection, named *molluscum* by Willan, differs altogether from so-called molluscum contagiosum, to be presently treated of

* Such cheloid growths originating in acne scars must not be confounded with acne-cheloid, which is a synonym of Hebra's *syccosis frambæsiiformis* or *syccosis capillitii* (p. 921).

† *Synonyms.*—Molluscum fibrosum, areolo-fibrosum, non-contagiosum, simplex, pen-

(p. 1001), except in the fact that they both consist of multiple pedunculated tumours. Those of fibroma are soft and painless, the skin over them is unaffected, they are more or less pedunculated, they vary in size from a pea to a marble or a fist, and when cut into they show œdematous, inelastic connective tissue. They resemble, both in appearance and structure, the firmer kinds of polypi of the nasal fossæ, the colon, rectum, uterus, and other parts of the mucous membranes. They might, in fact, be well termed "multiple cutaneous fibroma" or "multiple fibrous polypi of the skin."

The number of these tumours is sometimes almost innumerable, as is seen in the well-known case of Virchow, which forms the frontispiece to his work on morbid growths ('Kr. Geschw.,' Bd. i, S. 325). Their size varies from a pin's head to a foot or more in diameter.

The celebrated case of Tilesius, of Leipzig, published in 1793, was named molluscum by Willan from the soft fleshy character of the tumours (*corpus tectum est verrucis mollibus sive molluscis*).^{*} Bateman recognised that they were not glandular, and were quite distinct from the *molluscum contagiosum* described by himself.[†]

There appears to be little local predilection for these fibrous polypi. We sometimes see a single one on the face or elsewhere, or they may cover the face, the trunk, and the limbs. They also sometimes appear on the prolabium and the palate, as in Dr Fagge's case, which was figured in pl. xviii of the Sydenham Society's 'Atlas,' and modelled for the Guy's Hospital Museum (No. 497).

Cutaneous fibromata occasion no pain, and single ones may be met with in perfectly healthy persons, to whom they cause no inconvenience. Some of these are congenital, but many multiple fibromata are certainly not so. They usually appear in childhood. Multiple fibromata are certainly rare, and probably few cases have failed of being recorded; but one or two fibrous polypi are not infrequently seen if looked for.

When they have ceased growing they undergo no further change, and neither degenerate nor become absorbed. But, as Mr Hutchinson has pointed out, they sometimes lose their firm, fleshy feel, and become flaccid, so as to feel almost like empty cysts. (See the 16th of his Clinical Lectures "On Rare Diseases of the Skin.")

Large and numerous fibromata are sometimes associated with elephantiasis and œdema (p. 964). Such was the case in Dr Haen's and in Virchow's patients, and in the unfortunate person shown to the Pathological Society by Mr Treves (in 1885) as "the elephant man."

Fibromata are also occasionally found in large numbers associated with an unusual laxity of the skin of the trunk over the subcutaneous structures. In this curious condition, described as *cutis pendula*, *pachydermatocele*, or *dermatolysis*, the skin hangs in great folds like garments. A classical instance was recorded and figured by Meek'ren in 1657; the patient,

dulum—Fibroma molluscum (Virchow). Esmarch's 'Elephantiasis' (1885) records and figures several remarkable cases. See also Dr Sangster's case with drawings and references in the 'Clin. Trans.' for 1880, and seven cases in the 'Guy's Hosp. Rep.' for 1889, p. 388.

^{*} The skin of Rheinhard, the Mühlberg peasant who came under the notice of Tilesius, is still preserved in the museum of Leipzig.

[†] Dr Fagge believed that these tumours begin in the outer sheath of the hair-follicles and sebaceous glands; and in one case he found an enlarged sacculated gland occupying the interior of one of the growths ('Med.-Chir. Trans.,' 1870, vol. liii, pl. vi). This has, however, not been again observed, and the occurrence of similar tumours in the palm and sole seems to prove that the coincidence was accidental.

a young Spaniard, could bring the skin of his chest up to his eyes and down to his knees. Dr Valentine Mott, of New York, published in the 'Med.-Chir. Trans.' for 1854 five cases, with two portraits; in all of them the redundant masses of skin were successfully removed by operation, but in one the growth twice returned.

Virchow recorded a case in which the father, grandfather, and brother of a patient were all affected with multiple fibromata of the skin, and several similar cases have been since observed.

According to Hebra, when they are numerous the patient is usually ill-developed in mind and body. But this is certainly not always the case.

The only treatment is removal of the fibrous polypi by the scissors or the knife. They show no tendency to return.

Neuromata or neuro-fibromata are multiple, fibrous tumours of the nerve-trunks, scattered over both trunk and limbs. They have been long known, and formed the subject of a monograph by von Recklinghausen ('Ueber die multiplen Fibrome der Haut und ihre Beziehung zu den multiplen Neuromen,' 1882). Except by the pain which often accompanies them, these tumours are indistinguishable from ordinary fibromata. Dr Duhring has recorded some severe cases marked by paroxysms of neuralgia.

Myoma.—Tumours of unstriped muscular fibre (*leiomyomata*) have been described by Virchow ('Archiv,' vols. iii and vi), Klebs, Axel-Key, Rindfleisch, and Besnier ('Annales de Dermatologie,' 1880). They probably take their origin in the muscular bands connected with the hair-sacs. They are of no clinical significance.

Lipomata, or true fatty tumours, never affect the skin itself, but are always subcutaneous.

MOLLUSCUM.*—This somewhat rare disease was first described by Bateman, who added it to the small group of Tubercula as defined by Willan. The case figured by him in his 61st plate, on the face and neck of a young woman, was a typical example of the disease, and Bateman traced the contagion from a nursling of this patient and two other children in the same family back to a fourth patient with the same affection. He also mentions a second case in an infant apparently contracted from an older child. It was from these facts that the epithet *contagiosum* was applied by English physicians,† and also by von Bärensprung, Virchow, and Rindfleisch; and although the correctness of the epithet has often been doubted, it is now satisfactorily proved.

Carswell, Rayer, and other writers recognised Bateman's disease;

* *Synonyms*.—Molluscum contagiosum (Bateman)—Molluscum sebaceum—Epithelioma contagiosum (Virchow)—Acne varioliformis (Bazin)—Molluscum verrucosum.

† Wilson, 'Diseases of the Skin,' 1842, p. 302; Paterson, 'Edinburgh Medical Journal,' vol. lvi, 1841, pp. 213, 240. Cases were also recorded by Alibert, Biett, Cazenave, Schedel, Gibert, and Jacobovitz, 'Le Molluscum: Recherches critiques,' Paris, 1840. Most of the foreign cases, however, were examples, not of Bateman's disease, but of fibroma.

The term molluscum, there is no doubt, was taken by Bateman from the case of Tilesius, described by C. F. Ludwig, of Leipzig, in 1739. His words are, "Corpus tectum est verrucis mollibus sive molluscis." The word is obviously used as a synonym of mollis, just as mollusca was first applied to the mollusca nuda et testacea, the *soft-bodied* animals or malacozoa. Alibert, followed by Cazenave, misinterpreted the meaning of the term. Bazin unfortunately described the disease under the term *Acne varioliformis*, and this has led to additional confusion, especially since the same term has been employed for a singular variety of acne mentioned above (p. 914) before its misapplication to molluscum contagiosum had been forgotten.

Huguier, in 1846, had described it as a non-syphilitic affection of the vulva, and Caillaux, in his 'Treatise on Diseases of the Skin in Children,' named it "acne molluscum."

The disease occurs in the form of small rounded tumours of a pink colour, sometimes sessile, but more often pedunculated. They are scattered irregularly over the skin, which remains quite healthy between them. Their number varies from a single tumour to a countless multitude, and the size from that of a vetch, to use Bateman's comparison, or a large pin's head, to that of a marble; but occasionally reaches much larger dimensions. The colour also, though usually pink and waxy, is sometimes scarcely distinguishable from that of the skin, and at others it has a dead white or even yellowish tint. These last, however, are probably not uncomplicated examples of the disease. A minute dimple is to be found on each tumour, which was formerly supposed to be the orifice of a sebaceous duct. The growth of the little tumours is very slow. They may retain a size not exceeding a pea while increasing in number during many months, and perhaps longer. As they grow larger they become more rounded and the groove at their base becomes deeper, until they may hold to the skin by only a slender pedicle, a condition which was formerly described as *acorchordon*. The colour usually becomes paler and more translucent as they increase in size, but this change is not constant.

No *symptoms* are produced in the most marked and typical cases; as before stated, the skin between the little tumours is perfectly normal, and they themselves are no more than a disfigurement.

Anatomy.—On incising the tumour, a white opaque thick material can be usually squeezed out, and a hollow sac remains flaccid behind. Herein a molluscum tumour resembles an ordinary sebaceous cyst or steatoma (p. 918), but the contents are white instead of yellow, and to the naked eye have not the atheromatous appearance so characteristic of accumulated sebum. Moreover, tested chemically and microscopically, instead of fat, cholesterin, and earthy salts and epithelial scales, the white material seems to be made up almost entirely of characteristic oval transparent bodies with a hard capsule. These molluscum bodies have a characteristic pearly lustre, and are also recognised by the absence of a nucleus and by their not readily staining with logwood. These "molluscum corpuscles" were first recognised and well described by Erasmus Wilson in 1842; they were rediscovered at Saint Louis and described as cryptogamic spores, the source of the contagion. This view, however, is certainly incorrect; for their size, their aspect, their reaction to potash, and their inability to develop are conclusive against it. No doubt they are epidermic cells which have undergone a hyaline transformation. Along with these molluscum corpuscles there is often found a certain amount of fatty sebaceous material; but in some well-marked cases there is an entire absence of sebum.

A section horizontal to the surface shows that each little tumour is made up of loculi more or less separated from each other by septa, and in most cases a central cavity may be demonstrated, which was formerly supposed to be the duct of a sebaceous gland. That the whole tumour is a new growth, unconnected with the sebaceous apparatus and starting in the deeper epidermic cells, was the opinion held by Virchow, and supported by Dr Sangster's observations.* It may now be regarded as settled.

* See papers by Drs Morrison, Crocker, and Thin in the 'Pathological Transactions' for 1881, p. 245, and also a description by Mr Davies-Colley, 'Guy's Hospital Reports,'

Distribution.—Molluscum is most common on the face and neck, the eyelids and cheeks, and also on the mammæ of women; but when a multitude of small tumours occur, they may be found upon the arms, especially the thin skin of the flexor surface, as well as on the face.

Mr Hutchinson describes molluscum as not uncommon on the penis and scrotum of young adults, and refers to a case of Dr Paterson's where similar tumours appeared on the vulva of a woman whose husband was thus affected. He also refers to the molluscous growth occasionally suppurating, and points out that it may resemble an indurated chancre.

In the cases which the writer has seen of molluscum-like tumours occurring on the male genitals the tubercles have been yellow, not either pearly and translucent, or pink and waxy-looking; and on incision, or even on pressure without incision, have yielded opaque yellow oily material, so that they were no doubt steatomata. In the Guy's Hospital Museum a model shows, however, true molluscum upon the thigh ('Catalogue,' p. 240). Sometimes a large molluscum tumour will suppurate, burst, and thus cure itself. A case of this kind occurred under the late Dr Addison in a girl ten years old.

Molluscum contagiosum is most common in infants and children, less so in women, and decidedly rare in men. In children it almost always affects the face, in women the mammæ, and in men the genitals. But this is only true of the larger and fewer tumours. All the cases of numerous very small molluscum (simulating warts and sometimes described as *molluscum verrucosum*) which the writer has seen in adults, have been situated on the arms. Mr Hutchinson has observed similar cases in which the trunk or lower extremities have been so covered with little tumours as to resemble some papular eruption, as lichen. Others of these wart-like growths, however, may be multiple fibromata.

Molluscum appears to be much more common in England than abroad, though cases of the true disease are reported from France and Germany. It is well known in Scotland and also in America. The majority of cases occur in dirty neglected children, but it is often seen in those who are clean, rosy, plump, and in every respect healthy.

Pathology.—There can be no doubt that the epithet contagious is rightly applied to this disorder,* notwithstanding the frequent failure to reproduce it by inoculation, and the incredulity of Hebra and other dermatologists. The subject is well discussed by Duckworth in two interesting papers in the 'St Barth. Hospital Reports' for 1868 and 1872. No fungi and no bacteria have yet been observed. The molluscum corpuscles are now ascertained to be transformed epithelial cells, although Neisser and other good observers supposed them to be psorosperms or pseudo-navicellæ.

Treatment.—Molluscum tumours, beside, as above stated, sometimes suppurating, which appears particularly apt to occur when they are confluent, may also undergo passive involution by gradually shrinking and subsiding. This must be the case with many infants who have never

3rd series, vol. xviii, 1870, p. 350, and figs. 1 and 2, p. 364. He describes most of the characteristic oval cells as nucleated. Virchow's original paper was published in 1865 in the 33rd volume of his 'Archiv,' p. 144; Sangster's in the 'Med.-Chir. Trans.,' 1880, vol. lxiii; and Thin's in the 'Journ. of Anat. and Phys.,' vol. xvi, p. 202. The same conclusion was independently arrived at by Bizzozero, Retzius, Boeck, and other histologists. (See the figures in Dr Galloway's article in 'Allbutt's System,' vol. viii, p. 875, and the references on p. 879.)

* Mr Hutchinson has met with cases apparently contracted at Turkish baths.

come under medical treatment; but such spontaneous cures are rare or slow and uncertain, while treatment is rapid and efficient. Each tumour should be removed either by being snipped off with a pair of sharp scissors curved on the flat, or by being incised and emptied. In either case the whole of the diseased structure must be removed. Where the tumours are very numerous, it may be better to apply nitric acid or the acid nitrate of mercury to each one in the early stage. When the growths are not larger than pins' heads, Mr Hutchinson believes that white precipitate and sulphur ointment in equal parts will cure the affection; but it is rare for an opportunity for this treatment to occur.

Cases of "*giant molluscum*" have been recorded by Virchow, Laache, of Christiania, and Dr Walter Smith, of Dublin. In the last of these the tumour was more than three inches across, in Laache's it was still larger—as big as two fists. Dr Payne has described a still more anomalous case resembling Molluscum in the 'Brit. Journ. of Derm.' for 1891, iii, p. 250.

GLANDULAR TUMOURS.—*Colloid milium** was the name given by Ernst Wagner in 1866 to an exceedingly rare affection of the skin, which consists in the appearance of a multitude of raised, yellowish, glistening nodules like those of milium (p. 915). They occur in groups, and vary in size from a pin's head to a split pea. They do not contain sweat, but a thick gelatinous secretion.

These nodules are probably new growths, beginning in the sebaceous, not the sudoriparous glands. Their histology is described in the 'British Journ. of Derm.' (vol. iii, p. 36) by Dr Philipson, who considers them as being (benign) epitheliomata with colloid degeneration. They occur about the eyes, where they look like xanthelasma, but are also seen on other parts of the face. In one case of Dr Liveing's the arm was affected, and in two reported by French writers the whole of the front of the chest was covered. The few patients yet observed have been adults.

The disfigurement has been removed by scraping out the little tumours one by one.

Adenoma sudoriparum.†—Several cases have been reported of multiple minute tumours of the skin which are seated in the sweat-glands. One of the first observed in this country was described and figured by Dr Perry, and four by Mr Brooke, of Manchester, in the 'Brit. Journ. Derm.' for 1892 (vol. iv, p. 269). They are multiple papule-like cystic glandular growths, probably congenital, and affecting either the face and scalp alone, or trunk or limbs also. The affection has been identified with Colloid milium, and with Lymphangioma tuberosum multiplex (*infra*, p. 1006).

Adenoma sebaceum.—Under this title Dr Balzer published two cases of multiple tumours of the face ('Arch. de Physiologie,' 1885, No. 7, and 1886, No. 5), which were modelled for the museum of St Louis. Dr Pringle has added a third case, which, like the others, occurred on the face of a young woman ('Brit. Journ. of Derm.,' Jan., 1890). The figure he gives has a rough resemblance to acne, but there are no comedones and no suppurating points. The papules or nodules are firm, whitish, and like grains of sago in size and shape. Some look like milium, others more like multiple warts.

* *Synonyms*.—Colloid degeneration of the derma (Besnier)—Hydradenoma (Darier)—Syringo-cystadenoma (Török).

† *Synonyms*.—Hydradénome éruptif (Jacquet and Darier, 1887)—Épithéliomes kystiques bénins (Jacquet)—Cystadénomes épithéliaux bénins—Epithelioma adenoides cysticum.

When pricked, inspissated sebum can be squeezed out. Along with these sebaceous tumours are numerous dilated vessels, which, however, disappear under treatment. Microscopical observation shows hypertrophy of the sebaceous glands and of the deep layer of the cutis. The little tumours sometimes undergo spontaneous involution, but treatment is unsatisfactory.

The affection appears to be, as a rule, congenital, and is associated with other defects of the skin or of the nervous system.

VASCULAR TUMOUR.*—Excluding moles or pigment-spots, true or vascular nævi have always essentially the same structure. But they vary in appearance, from the smooth, flat, “port-wine stains,” as they are called, which sometimes cover a great part of the face, head, or even trunk, to the circumscribed pulsating tumour-like mass which can be removed by ligature, galvano-cautery, or other mechanical means.

Of similar structure though different pathology are the *stigmata* of gutta rosea and erythematous lupus, and the permanently injected patches which sometimes accompany the cicatrization of lupus, syphilis, or any other deep form of dermatitis. These acquired capillary and venous dilatations may be called *angiectases*.

There are, however, some rare and remarkable forms of disease of the skin which, though anatomically angioma, differ from true nævi not only in being acquired instead of congenital, but also in their course and event. Sometimes they will, as described by Hebra, while spreading in some directions, return to a normal condition in others; or, again, they may acquire a tumour-like and semi-malignant character, growing rapidly and forming large masses of erectile tissue. They are most often seen upon the extremities, though even here they are happily rare. They are sometimes complicated with fibrous growths, which have not only the pain of neuroma, but also its histological characters. Bruns recorded such cases on the lower extremities as *elephantiasis neuromatosa*.

Virchow and Kaposi described, under the somewhat similar title of *elephantiasis teleangiectodes*, multiple fibro-vascular growths, which begin as separate lobulated tumours, but afterwards form diffuse vascular thickenings of the skin.

Apparently identical with these is a case in a child which was examined by Dr Liveing and figured by the late Dr Tilbury Fox under the name of *fibroma fungoides* (pp. 352—354 of his work on ‘Skin Diseases’). He there described other cases of fibro-vascular ulcerating growths which he considered to be of the same nature. One of these, however, may probably have been syphilitic.

Angioma serpiginosum.—Mr Hutchinson described as “infective angioma” or “nævus-lupus,” a singular affection, better named as above by Crocker, which has also been observed by Mr Waren Tay and two or three others in this country and abroad. It is not congenital, but appears early in life. Minute capillary dilatations appear, like grains of cayenne pepper, forming groups and rings which slowly spread. They affect the limbs or occasionally the face. (See the account in Mr Hutchinson’s ‘Archives of Surgery,’ 1891, vol. iii, p. 161.)

Angiokeratoma.†—This curious and rare condition begins as minute

* *Synonyms.*—Nævus flammeus—Nævus vascularis—Angioma—Mother’s mark.

† *Synonyms.*—It has been called Lichen telangiectasique, a misleading term; and a combination of horny warts with telangiectasis, but there are no true papillomata present.

purple or brown spots on the dorsum of the fingers and toes, and consists in capillary dilatation from chronic venous congestion. The next stage is hypertrophy of the horny layer of epidermis, with disappearance of the *stratum lucidum*. The aspect then simulates a wart or a corn; but the papillæ are atrophied and flattened, not hypertrophied, and there is no clavus present. The disfigurement—for it is nothing more—is most apparent in winter. It is usually seen in children or young women, particularly if they have suffered from chilblains. It sometimes occurs in several members of a family. The name was given by Mibelli in 1889, and cases have been recorded by Sangster and several others since. Dr Pringle has treated it successfully by electrolysis ('Brit. Journ. Derm.,' 1891, vol. iii, pp. 237, 309, with two coloured plates).

LYMPHANGIOMA.—A curious affection of the skin, which has been described under this name, consists in what looks like a group of vesicles; but, on careful examination, they are found to be more deeply seated than usual, and in the event prove not to be inflammatory at all, but new formations, lasting unchanged for an indefinite period. In one case of the writer's they strikingly resembled the vesicles of zona, appearing in several groups, and arranged in a tolerably regular line. In this instance the affection was complicated by appearing upon a large congenital port-wine stain, and the result was that many of the lymph-cysts became pink by admixture of their contents with blood, and when accidentally ruptured, thick red or black scabs were formed. This coincidence with ordinary vascular nævi (which others have also noticed) as well as histological investigation, seems to prove that the disease is rightly regarded as analogous to acquired vascular nævi. But Mr Hutchinson described the affection under the unfortunate name of "lupus lymphaticus" ('Path. Trans.,' 1880, with figure). Several cases have been brought before the Dermatological Societies within the last few years. A careful histological description with figures by Mr Stewart will also be found in the 'Path. Trans.' for 1875, in the volume for 1879 (xxx, 474) by Drs T. and T. C. Fox, and in that for the following year (xxxi, p. 346) by Dr Sangster.

Lymphangioma may affect the vulva, where its symptoms vary considerably from those which attend it in other parts of the body (Roberts, 'Brit. Journ. Derm.,' August, 1896, and J. C. and C. J. White, 'Journ. Cut. and Gen.-Ur. Dis.,' February, 1898).

Kaposi described a remarkable case of lymphangioma in a woman twenty-two years old who had several hundred violet-red pimples, round or oval in shape, some of them as small as a lentil, situated in the cutis and somewhat resembling certain forms of syphilodermia. A minute portion, being excised, showed that the cutis was filled with dilated lymph-spaces lined with endothelium. He named it *lymphangioma tuberosum multiplex*. A few similar cases have been since recorded, and a collection of cases was published by Dr F. A. Noyes, of Melbourne, in the 'Brit. Journ. of Derm.,' December, 1890, and Dr Török added a critical and pathological account of the condition (*ibid.*, January, 1891).

MYCOSIS FUNGOIDES.*—Under this name Alibert, in 1814, figured in his

* *Synonyms*.—Pian fongoïde (Alibert, 1814)—Eczema tuberculatum (E. Wilson)—Lichen hypertrophicus (Hardy; who, however, now recognises its character as a new growth)—Lymphadénie cutanée—Papilloma areo-elevatum (Beigel)—Granuloma fungoides—Sarcoma lymphadenoides (Auspitz)—Papillome étalé ou en plaque (Charpy).

It must be understood that the terms *mycosis fungoides* and *mycoides* merely refer to

Atlas certain mulberry-masses of ulceration which he called Pian, *i. e.* yaws (framboesia). In 1832 he supposed it to be syphilitic, and named it *Mycosis fungoides*. Bazin in 1851 met with a case which resembled Alibert's account, and invented a *diathèse fongoïde* to explain it. In 1869 Ranvier showed that these curious soft tumours, readily ulcerating into fungous papillary masses, showed histologically a series of lymphatic spaces with cytogenic tissue between (Gilot, 'Thèse de Paris,' 1862). In a case under Auspitz, Hochsinger discovered a micrococcus in these tumours which he thought might be characteristic. Rindfleisch and also Stellwagon found streptococci.

Dr Payne described and figured a remarkable case of granuloma fungoides ('Path. Trans.,' vol. xxxviii, and 'Rare Diseases of the Skin,' p. 9); and, after discussing the lymphatic and the bacillary theories of the disease, concludes that neither of them is adequately supported by facts. In this case, as in the writer's mentioned below, cultivation failed to demonstrate the presence of a specific microbe. So Köbner and others have also found.

Mycosis fungoides is a rare disease, but not difficult to recognise from description or from drawings.

It has been observed by Landouzy in an infant, but most cases recorded have been in adults, both men and women. The position of the tumours is usually on the trunk, less often on the face or limbs.

It begins like patches of dermatitis, which usually resemble chronic eczema, but are sometimes more like pityriasis or lichen, erythema or urticaria, with considerable itching. After a variable period there follows deep induration and thickening of the cutis, with formation of separate raised swellings. These continue long stationary, and sometimes shrivel again; but more often ulcerate and form the fungating tumours which are characteristic of the disease. Occasionally the granulomatous growths appear without a preceding stage of dermatitis; but their appearance is often immediately preceded by a gradual thickening of the skin, showing that the dermatitis which was at first superficial like eczema or erythema is now become deep like lupus or elephantiasis. They exude a great quantity of clear, watery, colourless serum. In genuine cases the lymph-glands are probably always unaffected. The progress is very slow, but with one or two exceptions the result is fatal. No treatment is known to be of service.

The disease resembles both syphilis and leprosy, and, in its early stages, eczema and some forms of lichen. It has a remarkable likeness, in its developed stage, to the worst form of iodide eruption (p. 905; see Mr Hutchinson's figure in his 'Archives of Surgery,' pls. iii and iv); but it is probably most like yaws in aspect.

A well-marked example of this disease in Philip Ward was described and figured in the 'Clinical Transactions' for 1892, vol. xxv. The patient was a man of 66, who first developed what looked like ordinary dry eczema; and then fungating masses of granulations formed on the various parts of the body, the neck, shoulder, loins, and leg. Some of these sloughed, suppurated, and healed completely, but others formed, and at last one enormous mass on the back. He died from pulmonary gangrene. Sections of the tumour looked like those of a round-celled sarcoma; and there was a similar new growth in one adrenal—an unusual condition.

Another case was that of a gentleman whom the writer saw in consultation with Mr Bowlby in 1894. He was a tall, powerful man of 45, who developed characteristic tumours in several parts of the body, and was repeatedly in a very serious condition, but under quinine and opium internally, and local antiseptic and astringent treatment, he entirely recovered, and nothing remained of the disease but pigmented patches on the chest and

the "cauliflower" masses or "fungous" vegetations of the disease as seen by the naked eye, and carry no implication as to a fungous or bacterial origin.

back, the buttocks, thighs, and perinæum. Whether the tumours would have recurred is uncertain, for after their final disappearance and his restoration to health he was attacked by double pneumonia and died.

Whether this disease should be regarded as a sarcoma, a lymphoma, or a granuloma has been much disputed. It is clinically and anatomically distinct from the multiple cutaneous sarcomata described in the first volume (p. 83), and also from the multiple lymphadenoma of Hodgkin's disease (p. 768). If it is a granuloma, some specific microbe should be discovered.

XERODERMIA MALIGNA.*—Perhaps the most remarkable of all cutaneous diseases, allied to nævi in its early stages and markedly malignant in its later development, is a rare affection first described by Kaposi in 1870 under the borrowed title of *xeroderma*, a name which had been previously applied to a totally different condition by Wilson (p. 917).

It begins with spots of erythematous appearance not unlike those of measles. Then they fade and form pigment spots like freckles, with dilated venules. There is also a diffused pigmentation which affects chiefly the uncovered parts—the face and neck, the arms and hands, and only slightly the legs below the knee. The next stage may be months or years in appearing. When it arrives, the apparent ephelides become atrophic, the skin white, dry, thin, and wrinkled. Then it gradually contracts, so as to form a smooth, tightly drawn surface, which may evert the eyelids or the lips or contract one of the joints. At the same time fresh brown pigment spots and stigmata appear on the affected surface. The former undergo the same atrophic changes, the latter may increase until they resemble congenital vascular nævi. Pigmented moles also frequently appear.

The disease is not accompanied with itching or pain, yet after continuing in this comparatively innocent form for months, or sometimes years, the last stage comes on. The vascular spots become warty and ulcerate; fungoid growths of a most malignant character appear, not only in the maculæ, but also in distant places; and death ensues by hæmorrhage or exhaustion.

Hebra and Kaposi together observed only four cases of this remarkable affection. Erasmus Wilson described another under the name "general atrophy of the skin." Kaposi, in 1885 ('Wiener med. Wochenschrift,' No. 44), could tabulate only thirty-eight published cases; the youngest patient was five months, the oldest forty; eighteen were males and twenty females. Rüder saw seven brothers affected! A remarkable case was shown at the Clinical Society as one of lupus, and was recognised as identical with Kaposi's disease by Dr T. C. Fox. This same case, with the others in the same family, will be found fully described by Dr Crocker in the 67th volume of the 'Medico-Chirurgical Transactions,' p. 169, with coloured lithographs, and a table of thirty-four cases; most of these were recorded by Rüder in a monograph on the subject, the rest by R. W. Taylor, of New York, by Neisser, and by Vidal.

From this table it appeared that the disease had never been observed above the age of puberty; but one case has been since recorded in a man of twenty-five, and three others by Falcao, of Lisbon, in adults (1896). One case occurred in an infant four months old, most under two years, one at nine, and one as late as sixteen.

* *Synonyms.*—Kaposi's disease. It was called "*xeroderma pigmentosum*" by Kaposi. Other suggested names (all better than this) are "*angioma pigmentosum et atrophicum*," "*liodermia cum melanosi et telangiectasia*" (Neisser), "*leptodermia maligna*" (Cavafy), and "*atrophoderma pigmentosum*" (Crocker). For bibliography see the paper by Dr Funk, of Warsaw ('Brit. Journ. of Derm.,' vol. i, p. 182).

As a rule it occurs in boys and girls indifferently. More than one case is found in a family; twenty-six of the thirty-four cases in Dr Crocker's table belonged to nine families. The disease is never congenital.

The histological characters of the spots are those of vascular dilatation, of pigmentation, and of atrophy. The final tumours appear to be always true epithelial carcinoma, not sarcoma.

Treatment has at present been unavailing. (See Dr Pringle's paper in the 'Clinical Transactions' for 1898, vol. xxxi, p. 296.)

The ordinary malignant growths of the skin are happily infrequent, nor have they many special points of interest; for their pathology is essentially the same as that of the corresponding growths upon mucous membranes; moreover, their recognition is not difficult and their treatment purely surgical, so that but little need be said of them in this place.

Carcinoma fibrosum, or scirrhus cancer, the most typical of all the forms of cancer, rarely affects the skin primarily, though it frequently infiltrates it as the result of primary carcinoma of deeper parts, as, for instance, of the mamma. The writer has seen three examples of the remarkable form of hard, indurating, and widely spread cancer of the skin described by Velpeau as *squirrhe en cuirasse*. One was a patient under Velpeau himself, in whom the disease had spread from a cancerous breast; another was a patient of Dr Humphry's, of Cambridge. The remarkable and wide-spread induration, before ulceration begins and before implication of deeper organs occurs, renders it peculiar, and causes a superficial resemblance to scleroderma or to certain forms of lupus. It is usually secondary to mammary cancer.

Paget's disease.*—This remarkable form of dermatitis has been referred to above (p. 846). It closely resembles eczema in appearance, and yet invariably precedes, if it does not carry with it, true alveolar cancer. It was first described by Sir James Paget in the 'St Barth. Hosp. Rep.' for 1874.

The distinctions from eczema are the locality, the absence of severe itching—at least in the earlier stage, the induration of the skin—sometimes felt from the first, and the age of the patient, who is usually a woman about fifty. The presence of so-called psorosperms is also characteristic of its cancerous nature. The diagnosis is most important, for if recognised, prompt removal will probably cure the disease.

Epithelioma,† or keratoid cancer (see vol. i, p. 91), is the most common form of malignant disease in the skin. Even this is rare, compared with its frequency in the œsophagus and large intestine, and at the labial, anal, and urogenital orifices. Its formerly most frequent seat, the scrotum, is happily no longer so, and "chimney-sweep's cancer" has become a rare curiosity in this country. This form of cancer sometimes appears in patches of arsenical keratosis (p. 907).

Rodent ulcer.—This affection, originally described by Jacob, of Dublin, in 1827, is now ascertained to be histologically carcinoma. (See Mr Hulke's paper in the 'Path. Trans.,' vol. xxii.) The presence of epithelial cells in the cutis vera, and of the nest-cells characteristic of the horny form of cancer, leaves no doubt of its real pathology. It is, however, the least

* *Synonyms*.—Cancerous dermatitis of the nipple—Malignant papillary dermatitis—Duct cancer of the breast—Sporospermal dermatitis.

† The term *epithelioma*, applied by Hannover, of Copenhagen, to this disease, of which he was the first to describe the histology, was discarded by Virchow for "epithelial cancer." Unfortunately "epithelioma" is also applied to molluscum contagiosum.

malignant of cancerous growths, for it spreads slowly, there is little new growth, and it does not affect the neighbouring lymph-glands. It is usually seen near the eye, upon the side of the nose, on the cheek, or the temple. Like other kinds of carcinoma, it is a disease of mature life or old age. Its early stages are those of a small, smooth, pale growth not unlike a wart. If, as is sometimes the case, it has begun in a congenital mole, it retains the pigment of that structure. It often has a pearly aspect, so as to look somewhat like a molluscum tumour, or even like the cysts not unfrequently found about the eyelids. When ulceration begins, it is covered by a rather thin, dark, and adherent crust. It produces little or no pain, and advances so slowly that when it first comes under the surgeon's eye it presents the appearance of a chronic, indolent, indurated ulcer, with sharp, well-defined nodular edges, and no granulations. In its later stages it resembles more nearly its pathological allies, epithelial cancer of the lip, the scrotum, the glans, and the vulva.

The diagnosis from tertiary syphilis lies in the ulcer being single, in its not invading the bones or other tissues, and in there being no other sign of syphilitic disease. From lupus it is distinguished by the scab being thin and dark, by its beginning at a much later period of life, and by microscopical examination of the material obtained by scraping the edges of the ulcer.

Under the title of *crateriform ulcer*, Mr Hutchinson has described a sore of the face or head, which is much more malignant than the rodent ulcer just described, since it grows far more rapidly. Ulceration begins at the summit of a rounded nodule, and forms a deep crater-like hollow with indurated walls. It is most common in elderly people ('Path. Trans.,' 1889, and 'Arch. of Surgery,' vol. i, pl. xi).

Both this and the more common "rodent" ulcer can only be treated as a cancerous growth, by early excision.

Sarcoma.—Beside carcinoma in the histological sense of the word, the skin is occasionally liable to multiple sarcomata. These are almost always secondary to some internal growth; by their large number, small size, and hæmorrhagic or sometimes melanotic character they may resemble certain forms of purpura or pigmentation. In a marked case under the writer's care the primary disease was found after death to be in the cæcum; it was a round-celled sarcoma. Cases of primary, single, and multiple sarcoma of the skin, however, also occur, chiefly in young subjects. Prof. C. Boeck has described a case of multiple benign sarcoid of the skin (New Syd. Soc., vol. clxx, p. 295).

PIGMENTAL AFFECTIONS OF THE SKIN

“The freckles, blotches, and parched skins,
The worms, that, like black-headed pins,
Peep thro’ the damask cheek or rise
On noses bloated out of size.”

WHITEHEAD.

Albinism—Leucoderma, congenital and acquired—Vitiligo—its relation to leprosy—Canities—Melanoderma, secondary to dermatitis, syphilis, adrenal disease, malaria, etc.—Ephelis—Lentigo—Acanthosis nigricans—Chloasma—Relation of melanoderma to leucoderma.

WE are familiar with degrees of pigmentation of the skin, not only in the several races of mankind, but also in the wide difference between individuals belonging to the same stock, and even to the same family.

Albinism, or complete absence of pigment, not only from the skin and its appendages, but from the iris and choroid, is always a *congenital* defect in the human race, as in rabbits, mice, horses, and other animals. The so-called “white” elephants are either albinos or piebald.

Albinos occur occasionally among the dark races. The “white” negroes have a dirty, pale skin, colourless hair, and pink irides with dark red pupils.

The albinism which affects certain species in winter, as ptarmigan, hares, foxes, and stoats, involves a remarkably rapid change of colour. Increased pigment or melanism is also a frequent variety, as in the leopard.

Leucoderma.*—This may also be a congenital variety of coloration, a kind of “malformation.” Piebald horses may be called “abnormal,” but we should scarcely say so of cattle, dogs, swine, or guinea-pigs. This condition is, however, far more common in domesticated races than in a state of nature.

A similar congenital “piebald” state of the skin is occasionally seen in human beings. In negroes and in the natives of India it appears to be not uncommon. See a drawing of a remarkable case in a Hindoo given by Mr Hutchinson in his ‘Archives of Surgery’ (vol. i, pls. i and ii).† We sometimes see it in this country as white locks of hair occurring in the midst of brown. When acquired after birth, leucoderma has been, and still is, con-

* *Synonyms*.—Λεύκη—Vitiligo—White leprosy—Partial albinismus. The term vitiligo has been also applied to a circumscribed smooth white indurated spot level with or slightly sunk below the surface. This would make it identical with morphea, *i. e.* with circumscribed scleroderma (*v. supra*, p. 861). The word may well be abandoned.

† Dr Lesser figured a case of perfectly symmetrical leuco- and melano-dermia (‘Ziemssen’s Handbuch,’ Bd. xiv, 2te Hälft., p. 186, fig. 11).

founded with leprosy. In fact, "white leprosy," when it does not apply to psoriasis, seems generally to mean leucoderma occurring in patches.

Celsus (lib. iii, c. 25) distinguishes elephantiasis (*i. e.* leprosy) from *vitiligo* (calf's skin, parchment skin), which he divides into three species (lib. v, c. 16):—*V. alphas*, scattered, colourless, slightly rough patches (*psoriasis?*); *V. melas*, pigment-spots, to be presently mentioned under *melanoderma*; and *V. leuce*, still whiter than *alphas*, with white hairs growing on the patches (*leucoderma*). But later writers speak of vitiligo, and more particularly of leuce, as varieties of leprosy. The same ambiguity of meaning applies to the Arabic term "Baras," the equivalent of Leuce or Alphas in Greek, and Vitiligo in Latin, which was also used to denote a white patch or white leprosy. The confusion is due to patches of skin occurring in true leprosy, which are either deeper or paler in tint than the surrounding surface. The ambiguity appears still to exist, not only among the natives of Southern India and Ceylon, but among some physicians, judging by their reports in the Blue-book which was published in 1867. Dr Vandyke Carter stated expressly that in India leucoderma was still commonly confounded with anæsthetic leprosy.

The skin in true leucoderma is perfectly normal except for the loss of pigment. The Malpighian layer and also the hair are affected. There is no anæsthesia. The border is convex, and often a pigmented line separates it from the normal skin around; this was the case in the specimen which Gustav Simon first examined histologically. The patches are usually multiple, sometimes very numerous. They are occasionally symmetrical, more often irregular, with no predilection for one surface of a limb or the other. They may occur anywhere, but are most frequent on the trunk, especially the abdomen and genitals, where natural pigmentation is deepest.

Leucoderma is more common in hot countries and in the south of Europe than in England, but here cases are readily overlooked, since they are often inconspicuous, and give rise to no discomfort.

Removal of the white patches has been attempted by blisters and other irritants, and also by tattooing. The result is not often satisfactory.

Canities.—General blanching of the hair is a well-known senile change. But, like baldness, it often occurs in early adult life, especially when the hair is very dark. Beside losing its natural pigment, and so acquiring a dull, yellowish, "milk-white" appearance, the hair is apt to become dry and admit air-bubbles, which increase its refractive power, and produce the glistening steel-grey or "silvery" aspect.

Many instances are on record of rapid blanching of the hair of head or face in consequence of mental anxiety or grief. The cases of Sir Thomas More, of Henry the Fourth of France, and of Marie Antoinette have become historical, and it seems impossible to deny the fact that this premature senile change may come on in the course of a few hours. Bichat and Alibert record cases which they actually saw, and Brown-Séquard noticed blanching of his own beard ('Arch. de Phys.,' 1869, p. 442). A grey patch sometimes follows neuralgia (*cf.* vol. i, p. 557). The late Dr Laycock quoted an instance in which a sepoy was seen to turn grey in half an hour ('Med. Times and Gaz.,' 1862).

A young man who once consulted the writer for some slight ailment had perfectly white hair. In answer to inquiry he stated that a few years before he had fallen asleep after a debauch, and on waking in a cold room in the morning found that his hair had turned white. The objection that his beard was brown was answered by the explanation

that when the change of colour occurred it had not yet grown. This sudden change was probably due to development of air-bubbles in the shaft of the hair.

Melanoderma (melasma cutis, nigrities).—Increased pigmentation of the skin, like its diminution, may occur either universally or in patches.

A dark skin at birth is always hereditary. In after years it may occur as the result of exposure to the heat of the sun or to other irritants, or as the result of certain internal diseases.

As the result of hyperæmia or slight superficial inflammation, one sees increased pigmentation produced not only by the sun (*eczema solare*), but also by the wind in cold weather, or in driving, or by the light of snow-fields, which, as Alpine climbers know, will scorch the face without sunshine. Among Professor Hebra's patients the writer once saw a youth appear, who had wandered over Hungary in rags during the depth of winter. The exposed parts of the skin had become almost the colour of a mulatto, yet there had been little or no sunshine. Dr Bowles has made some careful experiments on this subject which confirm the result of experience, and show that the violet and ultra-violet (actinic) rays are the efficient bronzing agent ('Journal of the Alpine Club,' 1888, 'Trans. Derm. Soc. Gt. Brit. and Irel.,' vol. iii, p. 125).

Although all hyperæmia produces more or less increased pigment, there is considerable difference in the effect of different inflammatory diseases. The deeper and more chronic forms of dermatitis have very little pigmentary influence, as we should anticipate from their seat lying below the Malpighian layer and in the *cutis vera*. Long-standing *eczema* and chronic traumatic inflammation produce much pigmentation, as seen in the brown, almost black patches which surround indurated varicose ulcers in old people. Ordinary *eczema*, however, has little effect, and *impetigo* and *scabies* none at all.

Of the superficial inflammations, chronic inveterate *prurigo* produces the greatest pigmentation, and *prurigo pedicularis* almost as much, aided probably by the scratching which it occasions and also by the age of the patient; for all pigmentation is slow in childhood and rapid in old age.

Certain forms of erythema are marked by increase of pigment, particularly *roseola* (*pityriasis*) *maculata*, *pellagra* (*acrodynia*), and *urticaria pigmentosa*.

Psoriasis very early and readily causes pigmentation, and the colour is sometimes quite indistinguishable from the coppery hue of a syphilitic eruption. Indeed, we may say that next to syphilis, *psoriasis* will produce pigmentation in the shortest time. *Lichen planus* resembles *psoriasis* in this as in other particulars.

Besides the well-known brownish pigment which gives its characteristic colour to even early forms of specific eruption, a somewhat rare form of syphilide has been described by French authors as the "*café au lait*" form (*syphilide pigmentaire*). The writer has seen cases, both in Paris and in London, of ill-defined brownish maculæ occurring on the neck of women who were the subjects of secondary syphilis (p. 972); they are sometimes associated with patches of leucoderma.

Pigment has already been mentioned as occurring in some cases of *scleroderma*, of *osteo-arthritis*, and of *exophthalmic goitre*, and in the malignant kind of atrophic *nævi* of the skin called "*xeroderma*" by Kaposi (p. 1008). The remarkable increase of pigment in the course of Addison's

disease has been fully described in the chapter on that subject. Similar pigmentation, though far less intense, is observed as the result of malaria (vol. i, p. 393), and occasionally in the cachexia of cancer.

A source of cutaneous pigmentation which has only been recognised during the last twelve or fifteen years is the long continued absorption of arsenic, whether as medicinal or toxic. Numerous cases by R. Foerster (1890), Haffter (1889), Manssurow (1888), and in this country by Hutchinson (1891) and Gowers (1894), and many others, are referred to by Dr Nielsen, of Copenhagen, in a paper reprinted in the *New Syd. Soc. vol., No. 170*. The pigmentation is sometimes preceded by patches of slight dermatitis; it increases very gradually, and is most marked on the trunk and the flexor surfaces, less on the face and the hands and feet. It may simulate Addison's disease by its colour and also by the vomiting due to arsenic, but there appear to be no patches on the buccal mucous membrane (*cf. supra*, p. 726). The widespread epidemic of arsenical poisoning from beer which prevailed in and about Manchester in the spring of 1901, was accompanied by marked melanosis, often precisely resembling disease of the adrenals.

Maculæ—Ephelides—Lentigo.—It remains to mention circumscribed pigment patches, which occur without inflammation and independently of any other morbid sign. The most familiar are the small, dark brown or yellowish spots which, when they occur on the face, are named freckles (*ephelides*). They are no doubt, as their name implies, the result of exposure to the sun. They occur most frequently on the face, but also upon the hands and arms when these are bare. They are almost confined to xanthochroic complexions, and are particularly common in persons with red hair, blue eyes, and the delicate pink and white skin which often goes with them. These freckles, like the diffuse pigmentation of sunburn, disappear in time, though much more slowly.

Precisely similar minute dark spots appear in covered parts of the skin, and in mucous membranes, sometimes along with the melasma of Addison's disease or with pigmentation from malaria, and sometimes in conditions of health. Others are congenital, and may then be described as pigmentary *nævi* or "mothers' marks." When combined with a congenital papilloma, often covered with a strong growth of hair, they are called "moles" (*nævus pigmentosus*). In some cases these moles are accumulated over the gluteal region, genitals, and upper part of the thighs, so as to form a continuous hairy coat.

Acanthosis nigricans.—A rare and striking combination of melanoderma with papillomatous growths was described by Pollitzer in 1889 under this name. The following case occurred in Guy's Hospital, and was shown at the London meeting of the British Medical Association in 1895 ('*Lancet*,' vol. ii, p. 501).

A woman about 52 years old was admitted under the writer's care, July, 1895, with pigmentation of the skin, chiefly of the trunk, which had lasted five or six years. The colour was a deep mulatto-bronze deepening into black in the flexures. On the neck and also on the abdomen were numerous characteristic pigmented warts. There was no internal disease and no affection of the lips, tongue, or umbilicus, as observed in other cases. The aspect was not unlike that of congenital ichthyosis, and some of the dark colour was removed by soap. Excessive pruritus and scratching had also added to the pigmentation, but the comparatively short duration and the presence of warts decided the diagnosis.

A similar case was recorded by Janovsky and two by Darier under the superfluous synonym, *dystrophie papillaire et pigmentaire*. A section of

the skin in one of the latter cases, that of an elderly woman, showed that the chief thickening was in the horny layer of epidermis. Dr Crocker recorded a case in a young woman in 1881 ('Clin. Trans.,' vol. iv, p. 152); and Mr Malcolm Morris published another well-marked case in the 'Med.-Chir. Trans.' for 1894.

One or two cases are said to have recovered, but most have proved fatal, either from intercurrent affections or from cancer of the stomach or uterus. This last fact suggests a comparison between this remarkable disease and the equally remarkable pigmentation ending in cancer described by Kaposi (p. 1008).

Chloasma.—Diffused patches of yellowish-brown pigment are sometimes seen on the forehead of pregnant women, and have long been known under the name *chloasma uterinum*. In some cases they appear during each pregnancy and disappear after delivery. The word *chloasma* was at one time extended to the pigmented patches on the trunk which we now know to be due to a fungus, and call *tinea* (or *pityriasis*) *versicolor* (p. 940). But there seems no reason why at present the term should not be reapplied in its original signification.

Similar diffused pigmentation on the forehead and about the eyes is symptomatic of ovarian irritation, and appears in some cases of dysmenorrhœa with each menstrual period. (See eight cases reported by Dr Champneys with valuable comments in the 'St Barth. Hosp. Rep.,' vol. xv.)

Such pigmentation may also be the result of sexual excesses in male subjects, but this cannot be distinguished from the dark circles round the eyes which often accompany severe attacks of headache, especially migraine or prolonged fatigue, anxiety, and want of sleep.

These cases may be grouped together by their clearly neurotic origin. They must be carefully diagnosed from not unfrequent instances in which lamp-black or other pigment has been designedly applied to the face, forehead, and eyes by hysterical or otherwise deceitful women.

Lastly, there are certain cases in which patches of pigmentation occur in various parts of the body, unconnected with local irritation and without any internal disease.

These cases of idiopathic circumscribed melanoderma are decidedly rare, as rare as leucoderma without pigmentation. The rule is for diffused melanoderma to be associated with leucoderma; that is to say, white patches occur in the pigmented surface. The pale spots are sharply defined and have convex borders, the dark colour is most marked close to the white (apart from the effect of contrast) and gradually shades away into the normal skin. Most cases may be called either melanoderma or leucoderma, or both at once; and apparently consist in an irregular distribution of pigment. The white patches usually come first.

Treatment of pigmentation.—Solutions of corrosive sublimate, such as "virgins' milk" and "Gowland's cosmetic," are believed to have the power of removing freckles. The mingled patches of white and dark skin just described are best left alone, but circumscribed pigmentary nævi which cause disfigurement on the face may, if small, be removed by excision or galvano-causis.

ZONA

AND OTHER TROPHONEUROSES OF THE SKIN

“Ignis sacri plura sunt genera, inter quæ medium hominem ambiens, qui Zoster appellatur, et enecat si cinxerit.”—PLINY.

ZONA or Herpes Zoster—Anatomy—distribution—symptoms—sequelæ—Pathology—Traumatic zona—Cases—Treatment.

Trophic nerves, vaso-motor or secretory—characters of neurodermatoses—bullæ, gangrene, distribution, anæsthesia, origin in the ganglia or in the trunks of nerves—Atrophy, pigmentation, and other supposed nervous lesions.

THE names given to this remarkable disease, Zona,* zoster, or shingles, refer to the fact that it passes round the trunk of the body like a girdle. The brief notice of it given by the elder Pliny also implies the knowledge of another striking feature, namely, that it is limited to one lateral half of the body. He says that it kills if it encircles; and a popular tradition to the same effect still exists in England. Nevertheless zoster is never fatal, and does not pass over to the other side of the body from that first affected.

The distribution of the eruption in a case of shingles corresponds more or less with that of the peripheral distribution of sensory nerves, as Bärensprung pointed out; but typical Herpes zoster does not accurately follow the intercostal nerves. As Dr Head has shown, its distribution is that of the nerve-roots which come off regularly from each segment of the spinal cord. These individual nerve-fibres lose their connection when they are collected into trunks after forming the plexus to supply the limbs.

When the supra-orbital branch of the fifth nerve or some twig of the superficial cervical plexus is affected, the affected area has, of course, no longer the form of a belt. The term zoster, or zona,† is now generally applied to this well-marked disease, and is every way preferable to the cumbersome double name, herpes zoster. Herpes is an ambiguous term which should never be used, except for herpes labialis or herpes præputialis (*cf. supra*, pp. 896, 897).

Anatomy.—The eruption of shingles is “herpetiform,” *i. e.* it consists of vesicles in groups on an inflamed patch. They are of flattened form, and larger than those of eczema, sometimes as large as split peas; they are

* *Synonyms.*—Herpes zoster—Zona ignea—Ignis sacer (Celsus)—Exedens præcordiorum herpes (Tulpius)—Erysipelas phlyctænodes (Cullen)—Shingles; a corruption of Cingulum = zona and zoster, a girdle.

† Which is used is of no consequence, whether *zoster* the soldier's belt, or *zona* the lady's scarf.

arranged in clusters of from a dozen to twenty or thirty, each cluster lying on a reddened and slightly swollen patch of skin; when the vesicles are thickly set they often run together, and form flat bullæ of irregular shapes.

Many years ago Dr Haight, of New York, first found an opportunity in Vienna of investigating the histology of Zona. His observations showed that the roofs of the vesicles consist of the horny layer of the cuticle, with some of the superficial elements of the rete mucosum adherent to the under surface; their floors are formed by the bare summits of the papillæ, with the deepest elements of the rete occupying the depressions between them; their cavities are traversed by numerous bands, consisting of masses of the intermediate elements of the rete, drawn out into long spindle-cells and cells with several tapering processes.

Dr Head's recent observations confirm this description. The vesicle is unilocular (not divided like that of smallpox, vol. i, p. 192), though partial septa may be formed by epithelium. The contents are altered cells which have lost their prickles, and immigrated leucocytes with serum.

The fluid which the vesicles contain is at first transparent, but after a time the presence of floating leucocytes renders it opalescent, and ultimately it may become purulent, or acquire a purple colour from the escape of blood through the softened tissues beneath. The cutis itself seems always to take some share in the inflammation, leucocytes being scattered in the spaces between its fibrous bundles, and along the vessels and nerves. When pus is formed, if the roofs of the vesicles have been removed by the friction of the clothes, ash-coloured surfaces are exposed, looking like layers of false membrane. In severe cases this necrotic process causes a permanent, sometimes anæsthetic scar.

The number of clusters is very variable, from a single one to ten or even more. They are generally developed, not all at the same time, but in quick succession; those come out first which lie nearest the nerve-roots involved; and after a few days fresh ones cease to make their appearance. There is a short papular stage; and some of the last groups not infrequently abort, without going beyond it. In certain very mild cases, when only one or two patches are formed, none of them pass into a vesicular condition.

In ordinary cases the eruption begins to dry up from the fifth to the eighth day; the centres of the vesicles become depressed, yellowish or brownish crusts form, and in the course of the third week these fall off, leaving reddish or purple stains. But when the cutis is thickly infiltrated with pus-cells, its superficial layer undergoes destruction, and an eschar has then to be thrown off: thus the process of healing is retarded, and an indelible cicatrix results. The distribution of such cicatrices in the course of a particular nerve shows at once the nature of the disease from which they arose. When zona attacks the forehead, it is particularly likely to leave permanent scars.

Distribution.—Bärensprung and other writers have given local names to zoster, according to the nerves affected; but the refinement is needless, and, as above noticed, the names do not properly apply to the anatomical nerve-trunks of the limbs, but to the nerve-fibres of the spinal nerve-roots.

When the disease attacks the face, the nerve which it follows is the fifth, the greater part of which answers to the sensory portion of an ordinary spinal nerve. Indeed, it is remarkable how exactly the clusters of vesicles sometimes map out the points of emergence of the several twigs of the tri-

facial nerve from their bony canals. When the first division of the nerve is affected, the loose tissue of the upper eyelid becomes extremely oedematous and swollen, so that the affection may be mistaken for erysipelas by a careless observer. Another peculiarity of this form (supra-orbital zona) is that it is often attended with ulceration of the cornea and iritis, by which the sight may be seriously damaged. Mr Hutchinson has remarked that the ocular affection never arises unless the eruption occupies the distribution of the nasal twig. When the two lower divisions of the trifacial nerve are involved, a few vesicles often appear on the mucous membrane of the mouth and palate. Paget has recorded an instance in which necrosis of the alveoli followed infra-maxillary zona, so that some of the teeth fell out.

Cervical zoster, and that which affects the upper limb, follow the distribution of the sensory nerve-fibres. In some instances of brachial zoster the vesicles reach down to the fingers, but this is very exceptional; in the great majority of cases they do not extend below the elbow.

When the second and third intercostal nerves are affected, the intercosto-humeral branch produces a very characteristic eruption down the inner side of the arm as far as the elbow or rather lower.

On the trunk, which is by far the most frequent seat of the disease, the area occupied by the eruption of course slants more and more downwards as it approaches the pubes. It often happens that one or two vesicles lie slightly beyond the meridian plane, both at the linea alba and at the spine; this probably depends upon the fact that the nerves of the opposite sides overlap in their distribution, just as in the Siamese twins there was a part of the connecting band which received nervous filaments from each of them.

In the lower limb zoster is almost invariably confined to the buttock and thigh. Mr Hutchinson says that it never extends below the knee, but an instance to the contrary is figured by von Bärensprung in which there were a few small papules as low as the middle of the calf. Usually the eruption follows the distribution of the first and second lumbar and third sacral nerves on the buttock, of the second lumbar root and second sacral on the thigh, and of the third lumbar on the knee.

Side affected.—True zona is always strictly unilateral; and there is probably no difference in the liability to it of the right or left side. In ninety-four consecutive cases the writer found that forty-one were on the right and fifty-three on the left side.

Mr Hutchinson once saw a zoster in the course of the fourth dorsal nerve on the *right* side associated with a frontal zoster on the *left* side. In one of von Bärensprung's cases an ordinary zoster, limited to the *right* half of the thorax, was accompanied by a single vesicle in the *left* axilla, the patient having been suffering from severe burning pains on both sides. The writer has twice seen Zona of both sides, but not at the same level.

Symptoms.—Zona runs an acute course of a week or two, and is seldom attended with more than slight fever or disorder of the general health. In children, who are liable to it at all periods after the first year, it commonly runs its course without any unpleasant sensation, or is merely accompanied by a little numbness and tingling. Von Bärensprung tested the cutaneous sensibility with a pair of compasses, and found that in two cases it was considerably increased, while in a third it was diminished. Sir Thomas Watson relates a curious case in which zona affected the scalp, and in which the patient, who had for seven years been plagued with continual noises in the head, became free from this symptom, and remained so for eighteen months

afterwards. He also mentions a patient in whom the eruption came out in February, and who suddenly lost a cough which had teased him all the winter. Bärensprung met with two cases of zoster affecting the distribution of the fourth cervical nerve, in each of which vomiting occurred at the commencement.

By far the most important subjective symptom of zoster is pain of a neuralgic character, and referred to the same regions as are affected by the eruption. This is entirely absent in young patients, but in adults it is generally present, and in old people it is apt to be exceedingly severe.

Von Bärensprung cites a case in which there was only a single patch, of the breadth of two or three fingers, but in which the tenderness was such that the patient kept the part covered with the palm of his hand night and day, lest his shirt should come into contact with the vesicles. When the eruption fades the pain commonly subsides; but in some instances it continues long afterwards, for months, or even for years, with scarcely any abatement.

The persons in whom shingles leaves behind it this terrible neuralgia are always advanced in age. The two worst cases the writer has seen were in men over eighty, and in them it lasted until their death.

Sometimes the pain precedes the development of the eruption by several days. This led Anstie to regard zoster as a mere complication of neuralgia, analogous to other curious "trophic" changes which are met with in that disease (vol. i, p. 557). But in most cases of shingles (in two out of three, according to von Bärensprung) pain is altogether absent, and when it is the earliest symptom the rash appears within a fortnight at latest.

Cases of zona do not relapse, and the disorder seldom occurs more than once in the same patient.

Traumatic and secondary zona.—Some instances have been recorded of zoster following pressure upon the corresponding nerve-trunks, or occurring in the course of ordinary neuralgia. Thus Charcot and Cotard published a case in which one half of the neck and one shoulder were covered with the vesicles, the cervical nerves of that side being compressed by cancerous disease of the vertebræ. Charcot met with another patient, who, during a second attack of sciatica, presented herpetic vesicles on the lower part of the thigh. In a third case a man suffered from pain in the leg and back of the foot after a gunshot wound of the thigh, and an herpetic affection repeatedly developed itself upon those parts.

The writer has seen more than one case of recurrent bullous or vesicular eruptions following injuries to nerves, and one of typical zona in a patient suffering from cervical pachymeningitis, but it is very rare for any organic disease of the cord or nerves to be present when zona appears.

Pathology.—There can, however, be no doubt that zona is a true tropho-neurosis. This seems proved by its distribution; and, moreover, it is associated with anatomical changes in the ganglia of the posterior nerve-roots. That these ganglia are the starting-points of the disease was asserted by von Bärensprung in 1861. Charcot and Cotard found (in the case already referred to) that whereas the nerve-roots were healthy, the ganglia and the nerve-trunks to a little distance outside the intervertebral notches were much reddened and slightly swollen, their stroma being also crowded with nuclei. Precisely similar appearances were afterwards discovered by von Bärensprung himself in a child which died soon after an

attack of shingles; and much additional evidence to the same effect has since been obtained. The ganglia on the posterior roots are found swollen and infiltrated with leucocytes, sometimes with blood discs also.

Kaposi, Pfeiffer, and Landouzy abroad, with Mr Hutchinson and Dr Head among English dermatologists, regard zona as resembling an exanthem, particularly in its power of protecting against itself. But shingles is of infrequent occurrence; and in 100 cases under the writer's care no fewer than four of the patients gave a (more or less credible) history of a previous attack of the same disorder, and two of them showed scars which confirmed the statement.

With regard to the *causes* of zoster almost nothing is known. It occurs equally in both sexes, and at almost every age. There is no reason to suppose that it is caused by contagion, and no bacterial origin has yet been discovered. Zona has repeatedly been observed in persons who were taking arsenic, and it looks as if some chemical poison were the immediate cause of the affection of the sensory ganglia in other cases. In one instance, a patient of the writer's assured him that his father and a younger brother had suffered from a similar unilateral and painful acute eruption before.*

Cases.—Among 100 consecutive cases of zona which came under the writer's care in private or hospital practice, there were 61 in male and 39 in female patients. The time of life varied from infancy to old age; 15 patients were between one and nine years old, 31 between ten and twenty, 10 between twenty and thirty, 11 between thirty and forty, 8 between forty and fifty, 6 between fifty and sixty, 9 between sixty and sixty-five, one was seventy-seven, and one eighty-five years old. Dr Head, dealing with 378 cases at the London Hospital, found that no less than 283 occurred in patients under twenty-five years old, and the majority of these were between four and thirteen.

Among the writer's cases there were nine of supra-orbital and one of infra-orbital zona. In sixty-six cases the intercostal nerves were affected, in eight the great auricular, lesser occipital, or descending branches of the cervical plexus, in two the external cutaneous branch of the brachial plexus (second dorsal root), in eight the lumbar or sacral gluteal nerves, in nine the small sciatic (third sacral root), or external cutaneous of the thigh (second lumbar root).

Treatment.—There is but little to be done. The vesicles must be protected by a soft linen rag, with a pad of cotton wool or a flannel bandage. Some writers recommend that flexible collodion should be painted over them to facilitate their drying up; or a little starch powder may be dusted over the affected part as soon as any discharge appears. Children require no medicine whatever.

The treatment of the neuralgia which sometimes follows shingles is urgent, but often unsatisfactory. Bazin is said to have used arsenic with success; but as a rule it utterly fails. Dr Fagge used to prescribe *vinum colchici*, and in several instances the pain has quickly subsided while the patient has been taking this medicine. Subcutaneous injection of morphia gives at least temporary relief, and in old patients some form of opiate is almost always needful. Local anodynes are also applied, but without much

* Zona was found by Head and Campbell to be frequent among patients in asylums suffering from general paralysis.

benefit. Cocaine is perhaps the most efficient. Blisters have been applied over the roots of the affected nerves with good success in relieving the neuralgia.

OTHER NEURODERMATOSES.—It has been disputed whether disorders of nerves produce cutaneous diseases other than zona. The existence of trophic nerves is a physiological fact, but they give so easy an explanation that we must beware of admitting it without adequate proof.

The earlier experiments by Majendie and Donders, Legallois, Schiff, the elder Waller, Cl. Bernard, and many other physiologists, down to Langley and Sherrington, have proved that division of the nerves of a limb produces not only paralysis but wasting of muscles and of bones; that section of the inner fibres of the first division of the sixth cranial nerve causes sloughing of the cornea; and that section of the vagus leads to gangrenous inflammation of the lung. But after long controversy most physiologists seem now to be agreed that these trophic effects can be ascribed either to withdrawal of the protection of sensation, or to interference with secretion, or to vaso-motor paralysis.

In pathology, we find examples of all these effects of abolished innervation on the tissues, and also some evidence of the direct action of the nervous system on the growth and action of living bioplasm. One striking example is that of the atrophic changes in joints observed by Charcot in cases of tabes (vol. i, p. 696); another is atrophy of one half of the face; a third the effects of tabes on the bones, observed by Weir Mitchell in America, and by Page, Targett, and others in this country. Again, syringomyelia is followed by trophic changes in the skin (*ibid.*, p. 632), and general paralysis of the insane by effects on the osseous system. The so-called perforating ulcer (*ibid.*, p. 696) and painless whitlow are two other examples. Lastly, the series of atrophic paralyses which are due to anterior cornual lesions or to peripheral neuritis are each and all examples of disturbance of the trophic influence of a nerve-centre.

In the case of the skin, beside the effect of inflammation of the ganglia of the posterior spinal roots and of the Gasserian ganglion, in causing zona—which seems scarcely explicable by any vaso-motor action—we must refer to nervous disturbance “glossy fingers,” “quiet whitlows,” and the more acute forms of sloughing bedsores (*ibid.*, pp. 611, 612), with some other cases of gangrene of the skin; also perhaps the petular form of ichthyosis which follows the nerve-trunks down the limbs (p. 959), but this is more open to criticism.

Traumatic lesions of the nerves also produce glossy fingers and causalgia (vol. i, p. 574). Hilton recorded a classical case of ulceration after severance of the ulnar nerve, and many similar observations will be found in Mr Bowlby's ‘Injuries and Diseases of Nerves’ (p. 41 *seqq.*), where he justly decides that the evidence points not to irritation but inhibition of the trophic nerves. Other cases have been recorded by Leloir, Schwimmer, and Weir Mitchell.

Apart from distribution in the region supplied by a paralysed nerve-trunk or in the area connected with a definite nerve-root and ganglion, we may recognise the nervous origin of dermatoses by their being combined either with anæsthesia or causalgia (not with pruritus), by the nails and the hair being particularly apt to suffer, by secretion of sweat being affected,

and by the frequency of either bullæ and vesicles or of ulceration and gangrene. The sphacelus which sometimes attends diabetes is probably due to peripheral neuritis; the bullæ of syringomyelia are striking examples of a trophic nerve-lesion, and in the case of leprosy we found in the bullæ and ulcers, and slow, "quiet" mortification with anæsthesia, the cutaneous lesions of a special kind of peripheral neuritis.

Area has been referred to a neurotic cause, but there is no proof of it. Little if any anæsthesia is to be detected, and the patches do not follow the course of cutaneous nerves. Moreover, the atrophy of the skin would probably dull sensation by destroying the papillæ.

Leucodermia has been referred to the same cause. But here again there does not seem to be any reason for ascribing the disease to nerves, except the difficulty of finding a better explanation. *Melanodermia* may, perhaps, in some cases have a nervous origin, as in the dark eyelids of sleeplessness and exhaustion, the chloasma of gestation, and the bronze colour of Addison's disease.

The association of pigmentation with disturbance of innervation is, however, in most cases explained by the scratching to which pruritus gives rise and the subsequent darkening of the skin, as in chronic eczema.

Much that can be said in favour of the neurotic origin of *morphœa* and *sclerodermia* will be found in Mr Hutchinson's 'Clinical Lectures,' vol. i, p. 313.

The formation of the wheals of *urticaria* may be ascribed to vaso-motor nerves, and it sometimes follows mental emotion with great rapidity.

It has been held by some authors that symmetry points to a nervous origin of a cutaneous disease; while others hold symmetry to be the mark of "blood diseases," and asymmetry that of neuroses. Neither belief seems to be well supported. All general diseases are symmetrical because the human body is so; a one-armed man would be unsymmetrically affected by scarlatina or psoriasis. Again, psoriasis and eczema are symmetrical because they affect the skin of a certain structure and surroundings which is found on the corresponding parts of the limbs or trunk. The disease of the skin which is most certainly of nervous origin—*zona*—is, like neuralgia, markedly unsymmetrical.

See on this subject the monograph by Weir Mitchell, 'Injuries of Nerves and their Consequences' (1872); that by Leloir, 'Recherches sur les affections cutanées d'origine nerveuse' (1882); Mr Bowlby's Astley Cooper Essay on 'Injuries and Diseases of Nerves' (1889); also a valuable paper by Dr Crocker, with numerous references ('Brain,' October, 1884, p. 343); and one by Dr Savill, published in the 'Clinical Journal' (1897-8). Dr Head's important observations were first published in 'Brain' (1893), and will be found with fresh valuable matter in his article in the seventh volume of 'Allbutt's System' (pp. 624-9), 1901.

REMARKS ON THE PRACTICAL CLASSIFICATION AND DIAGNOSIS OF CUTANEOUS DISEASES

“Mais ce qu’il y a de fâcheux auprès des grands, c’est que, quand ils viennent à être malades, ils veulent absolument que leurs médecins les guérissent.”—MOLIÈRE.

Diagnosis and nomenclature—Rare and exotic diseases—Factitious and traumatic lesions—Animal and vegetable parasites—Bacteria—Symptomatic rashes—Syphilitic dermatoses—Eczematous, psoriatic, and acneiform dermatitis—Deep dermatitis; ulcers, lupus, and late syphilis—Treatment as the result of diagnosis.

IN the preceding chapters the diagnosis between two diseases which may be mistaken one for the other has only occasionally been stated in a formal manner. If the characteristic symptoms and circumstances of a malady are ascertained, they form the only and sufficient bases for its diagnosis; and although it is a useful exercise for a student to make lists of the distinctive characters of two or more diseases, the attempt to fix them in a tabular form is of little service to others, and tends to artificial memory of words rather than to familiarity with things. Symptoms differ endlessly at the bedside, and none of them is really “pathognomonic.” Moreover, the diagnosis of cutaneous diseases in particular often turns upon very slight differences in the form of lesion or in the distribution, which it is impossible to put into words; while much of what is called diagnosis in dermatology is not distinction between one pathological condition and another, but only between certain more or less arbitrary forms which have been fitted with still more arbitrary names.

In the present chapter it is proposed to treat briefly of this question of diagnosis, on which, in its true meaning, all successful treatment must rest; and after all, our patients, great or small, desire to be cured.

Complete diagnosis is recognition not only of the appearances of the affected skin, of its distribution, anatomy, and symptoms, but also of its origin and nature. Such complete diagnosis we can make in cases of measles or smallpox, of syphilis, scabies, ringworm, lupus, leprosy, and zona. Each of these names denotes a single pathological and clinical condition; and it is conclusive for prognosis and for treatment—the touchstone of all true diagnosis. It is no doubt desirable to recognise the exact drug which has caused the rash, the breed or race of fungus which is present in the

particular case of tinea, the prevalence of ulceration, nodulation, or pigmentation in the case of lepra, or the probable stage in the case of syphilis. But these are comparatively unimportant additions.

We ought to aim, then, first at a sufficient diagnosis to give the disease before us a distinctive name, which will connote for us the probable progress of the case and the efficient mode of treatment.

Unfortunately many of the names used in dermatology are mere descriptions of anatomical lesions or of symptoms or course or incidence, and many more are rendered cumbersome or useless or misleading by the vicious binomial classification into genera and species which once hindered the progress of every branch of medicine. In the hands of Erasmus Wilson and of Bazin this had become intolerable; and by a healthy reform such names as Psoriasis, Scabies, Ringworm, Zona, were allowed for a while to stand alone; but of late not only binomial but trinomial designations have multiplied, and in some cases a proposed name is an inadequate description a line long.

For practical purposes we must recognise that many names are useless, such as *keratolysis* or *angioneurosis*, or even *seborrhœa*, for they only express some eminent writer's theory of the origin or essential nature of the lesion, and include under the same term such diverse conditions as psoriasis and ichthyosis, or as erythema, sudamina, varicella, and pemphigus, or as what used to be called dry eczema and pityriasis capillitii.

Double names are very rarely needful. In most cases the "specific" name merely denotes some trivial variety of appearance often due to the stage of the disease: *e. g.* *guttata*, *nummularis*, *gyrata* applied to psoriasis, or the local varieties of zona. Some adjectives may be allowed as convenient distinctions added to the true diagnostic name, as *Variola corymbosa*, or *V. confluens*, *Pemphigus foliaceus*, *Erythema nodosum*, *Tinea corporis*, *Syphilis squamosa*; but these would be improved by translation of the adjective into the vernacular.

A small number of double names must, however, be retained for the opposite reason. They are not trivial or useless, but essential; they do not qualify the "generic term," but determine it. Such are *Pityriasis-rubra*, *Lichen-planus*, *Gutta-rosea*, *Lupus-erythematosus*, *Mycosis-fungoides*. The names are ill-chosen and meaningless, but it would be impossible to change them, and they at least connote an unambiguous diagnosis.

Many names no doubt represent only a provisional diagnosis, which does not go deeper than the anatomical changes and the natural history of the disease. Frequently we must wait until the general characters of an early dermatitis have passed and the special characteristics appear. Nor must we forget that most of the pigmentation and induration may be secondary changes due to the patient's hands or to over treatment.

Histological investigation has done much in the last twenty years for the pathology of the skin, particularly since staining reached its present perfection; but so many dermatoses differ less in their minute than in their external structure, that we are often disappointed.

The important share taken by bacteria, as distinct from fungi, in cutaneous pathology has thrown most valuable light on many diseases; but the skin, from soon after birth to the day of death, sustains an abundant bacterial flora, so that it is difficult to distinguish the several members, and more difficult to decide which are harmless and "natural," which pathogenic.

There are not a few diseases which are so rare that they rank as little

more than as curiosities. Such, for instance, are Pemphigus foliaceus, Urticaria pigmentosa, Favus, Cheloid, Acanthosis nigricans, and Xerodermia maligna.

Many important diseases of the skin again are exotic, as Leprosy and Lichen agrius, and only of practical importance for English readers who may practise their profession in India or the colonies.

Neglecting these, there are certain common affections which, differing by more or less important characters in appearance and in histology, nevertheless agree very closely in their pathology, in their causes so far as they are known, and, what is most important, in the kind of treatment which is generally successful. These form natural groups.

From a practical point of view, then, looking chiefly to questions of prognosis and treatment, we may arrange diseases of the skin as follows.

I. *Factitious eruptions*.—We must never forget the possibility of the affection before us being artificial. All kinds of dermatitis—eczema, erysipelas, pemphigus, impetigo—may be simulated by the application of various irritants. Pigmentation also has been often imitated with success. Such artificial diseases will generally be found on the arms, particularly the left, rarely on the face, and scarcely ever beyond reach of the patient's hands. The persons who are guilty of such attempts at imposition are usually either deliberate malingerers, like prisoners in gaol, or else they are hysterical young women or neurotic girls and boys. When one's suspicions are once awakened, it is seldom difficult to detect the imposture. Mustard, cantharides, and some other irritants can be distinguished by help of the microscope.

II. *Traumatic eruptions*.—In all cases of dermatitis we should seek for the irritant, and sometimes it is so directly the cause of the disease that the eczema or impetigo in question may be considered purely traumatic, and efficient treatment immediately follows accurate diagnosis: *sublata causa tollitur effectus*.

Pediculi in the hair should be carefully looked for in all cases of impetigo in children, pediculi vestimentorum in all cases of prurigo in old people. Scabies itself is but an extremely definite and well-characterised dermatitis resulting from the presence of a living source of irritation. But beside these well-known cases of parasitic dermatitis, it will be found that some supposed cases of purpura in children are nothing but fleabites, resemblance to which originated the name petechia. Moreover, many cases of infantile prurigo, urticaria, and ecthyma are due to the presence of bugs or gnats. In adults pediculi pubis may sometimes be found in the axillæ as well as in their proper region, and when they have been destroyed by white precipitate ointment the patient is at once relieved from pruritus.

In many trades an irritant must be sought in the objects which the patient habitually handles. The coarser kinds of brown sugar are a frequent cause of eczema of the hands (grocers' itch). So with many of the "chemicals" used in a variety of modern handicrafts. Constant wetness of the hands in washerwomen, in scrubbers, in potmen, and many others, produces eczema rimosum. The heat of the sun is the cause of eczema solare and ephelides, the heat of the fire of the pigment-spots on the shins of elderly people. Sweat, again, is a very common irritant, producing the dermatitis which usually accompanies sudamina and also intertrigo of op-

posed surfaces. Scratching as a cause of traumatic dermatitis has been repeatedly referred to in the preceding chapters, and also the danger of keeping up irritation by such applications as sulphur or mercury or tar when they have already done their work.

III. *Fungous diseases*.—The next great group of skin diseases includes those which are due to vegetable parasites—*tinea versicolor* of the trunk, *eczema marginatum* of the perinæum and thighs, *tinea circinata* of the neck and other parts, *tinea sycosis* of the chin, and *tinea tonsurans* of the scalp. Here the general characters detailed in the chapter on the *tineæ* are sufficient to show the nature of the affection to a practised eye, but in all cases the microscope should be employed.

Tinea of the scalp is rare in adults, and *tinea circinata* still more so; *tinea marginata* occurs only in adult males.

IV. *Bacterial and infective disorders*.—It is important to recognise the presence of bacteria as distinct from fungi, particularly those of so-called *seborrhœa capitis*, which enable it to spread to the body and set up dermatitis (*Seborrhœa corporis* or *Lichen circinatus*), which is cured by mercury, sulphur, or other antiseptic agents.

Still more important are the infections with staphylococci and other pyogenic microbes, which lead to pustular dermatitis and complicate other disorders. To this group belong boils, pustular sycosis and acne, and impetigo. The practical experience that suppurative dermatitis is best treated with yellow or red oxide or ammoniated mercury thus receives its justification.

V. *Toxic dermatoses*.—The rashes caused by drugs and poisons are often mistaken. They may resemble very closely the exanthem of scarlatina or of measles, acne, eczema or syphilis, or even malignant disease. The most severe and misleading are those caused by the iodides. The commonest are due to bromides. The ordinary cause of error is what prevents recognition of factitious eruptions—the possibility not entering our minds.

VI. *Febrile rashes*.—We must take care never to forget the possibility of a cutaneous eruption being part of an acute exanthem. The clinical thermometer is a great help in this respect, but the writer has seen a man with typhus (and the rash fully out) appear as an out-patient for a skin disease, and varicella and even modified variola are not infrequently mistaken for acne or impetigo.

VII. *Syphilodermia*.—When we have satisfied ourselves that the eruption before us is not factitious, nor directly traumatic, nor parasitic, nor an infective or symptomatic eruption, we may next consider whether or not it is due to syphilis. In this inquiry it is undesirable to ask questions, the answers to which are as apt to mislead as to guide aright.

(1) We should first consider the *colour* of the affected skin, remembering, however, that the pigmentation which gives the so-called coppery or raw ham tint to a syphilitic eruption is sooner or later imitated by all forms of chronic dermatitis. Psoriasis, chronic eczema, lichen planus, and prurigo may all produce shades which bear the closest resemblance to syphilodermia.

(2) The lesions of syphilis are *multiform* or polymorphic. It is rare in any but syphilitic affections to find mere hyperæmia in one part and associated pustules, papules, scales, or ulcers in others; and it is not often that a syphilitic eruption exhibits only a single elementary lesion.

A pustular eruption in an adult should always suggest the question of syphilis when that of scabies has been answered in the negative and there is no local source of pyogenic infection.

(3) Syphilitic eruptions for some unknown reason *do not itch*, and the exceptions to this rule are remarkably few; they occasionally occur during the stage of scabbing of pustular rashes or during the healing of tertiary ulcers. An ordinary secondary syphilide may, however, as a rare exception, be so irritable that wheals and scratch-marks are produced. On the other hand, psoriasis is often free from irritation, while the degree of itching of eczema, and even of scabies and prurigo, varies greatly.

(4) The local *distribution* of syphilitic diseases is a great aid in diagnosis. Specific eruptions are certainly not, as is often stated, symmetrical; the early roseolous rash is only so because it is general, and therefore, upon a symmetrical surface like the human body, more or less symmetrical. Moreover, as it chiefly affects the face, chest, and trunk generally, it is near the middle line. But we do not see symmetrical patches of syphilide in corresponding parts of both sides of the face, both sides of the trunk, or the right and left limbs. In all but the earliest syphilides the affected patches are very decidedly and constantly *unsymmetrical*, irregularly scattered over head, trunk, and limbs, and chiefly remarkable for having no well-marked seats of predilection.

The forehead, especially about the roots of the hair, is, however, very frequently the seat both of the early and middle erythematous, scaly, and pustular syphilides, and the palms of the hands and soles of the feet are frequently symmetrically affected with the later scaly eruption.

Practically, when we find a disease of the skin occupying some unusual position, we should at least consider the question of syphilitic origin.

(5) These signs alone or in combination serve to distinguish early specific roseola from erythema, eczema, scarlatina, and measles,—the later eruptions from eczema, lichen, scabies, impetigo, and psoriasis,—and the tertiary stage from lupus, leprosy, and frambœsia.

The eruptions of *congenital syphilis* which are most liable to be mistaken are—the so-called pemphigus of infants, which is known by its affecting the palms and soles; rupia, which, by the form of the crusts and the ulcerated surface beneath, may always be distinguished from impetigo; an erythematous rash of the nates and genitals of infants, which is distinguished from eczema of the same parts, also common at that age, by its coppery colour, its blotchy distribution, and more defined margin.

The *tertiary ulcers* of syphilis are distinguished by their appearing on unusual places, by their punched-out edges, circular or so-called horseshoe shape, and by their usually producing little pain or discomfort. Tertiary ulcers have no predilection for the outer side of the leg, but inasmuch as the part above the inner ankle is for anatomical causes the chosen seat of varicose ulcers, most ulcers in the former position will be syphilitic, and in the latter not. For the same reason most ulcers on the arms are found to be tertiary.

VIII. *Primary superficial inflammation*.—To distinguish the superficial from the deeper kinds of dermatitis we should notice whether the cutis alone is infiltrated and thickened, or whether it is bound down by adhesions to the subcutaneous tissues. The presence of scars, however slight, is a proof that the process has gone deeper than the papillæ and has more or less extensively destroyed the papillary layer. Superficial inflammations, excluding those due to the acarus, to pediculi, and to other direct irritants, and excluding those which are the result of vegetable parasites and of syphilis, fall with respect to their treatment into three large groups:

(1) The first, represented by most forms of Eczema, are subacute, and accompanied with burning, itching, and pain, sometimes with a slight degree of fever. They sooner or later yield to local remedies designed to reduce the hyperæmia, diminish the exudation, and calm the irritation, aided by light diet, free diluents, laxatives, and diuretics. In short, they are to be treated as inflammations of the skin.

(2) The second group of superficial inflammations of the skin is typically represented by Psoriasis, but includes Lichen-planus and the more chronic, dry, and obstinate forms of Eczema (often seborrhœic). They are chronic, with little irritation, exudation, pain, or active signs. They are best treated locally by tarry preparations, internally by arsenic.

(3) The third group is that of Erythemata. Here the indication is to correct some internal disorder of which the eruption is the symptom. They may denote an infectious fever, or rheumatism, or the effect of drugs, or gastric disturbance with toxic effects.

IX. *The acne group*.—Acne, both in its pathology and ætiology, differs from other forms of dermatitis. The age of the patient and its distribution are sufficient for diagnosis. It is at once a superficial and a deep dermatitis, and is often followed by scars. With acne may be classed Sycosis and Furunculus. They all depend on purulent infection, and must be treated from that point of view by local parasitocides and disinfectants.

X. *Deep affections*.—When we have ascertained that the affection of the skin is deep, that is to say, that it goes below the papillary layer, the field for diagnosis is limited.

Excluding erysipelas, which is distinguished by its acute character and febrile symptoms, excluding the pustular affections which penetrate the skin deeply and produce scars only at isolated points, such as acne, variola, and zona; and excluding, thirdly, leprosy and other exotic diseases, we have to distinguish in the great majority of cases of deep dermatitis—first, traumatic and varicose ulcers; secondly, gummata and syphilitic ulcers; thirdly, lupus; fourthly, rodent ulcer and carcinoma of the skin.

With regard to the first of these, we must not assume, because a sore upon the skin is said to be the result of a blow or a kick, that it is purely traumatic, for syphilitic ulcers often arise in this way. Malignant ulcers are rare, and usually obvious from the age of the patient, the pain they occasion, their tumid margins, and their blood-stained secretions. Moreover they are, with few exceptions, confined to the neighbourhood of the orifices of the body, especially the lower lip, the urethra, the vulva, and the anus. Rodent ulcer, however, is very difficult to be sure of. Its locality, its slow and painless progress, and its belonging to the latter half of life, usually serve to distinguish it from lupus; and its being single, excessively chronic, and

unaccompanied by nodes or other syphilitic lesions, are the best characters for diagnosis from a tertiary ulcer.

Between lupus and syphilis the difficulty of diagnosis is occasionally extreme. Lupus, however, is rarely more than single, syphilis is usually multiple; both are commonly free from pain and itching, but in syphilis the colour tends from red to rusty brown, in lupus from red to violet-blue; the scars of syphilis are depressed and pigmented, those of lupus hypertrophic and white; the edges of a lupous ulcer are beset with nodules, those of syphilis are either thin and smooth or indurated by chronic inflammation; lupus is in the majority of cases a disease of the face, syphilitic ulcers are quite as frequently on the limbs or trunk; lupus is a disease of the skin alone, syphilis affects the subjacent tissues also.

Methods of resulting treatment.—When by a careful study of the anatomical lesions—secondary as well as primary, microscopical as well as visible to the naked eye—by consideration of their regional distribution, the symptoms which accompany them, the course and natural history of the disease, and the condition of the patient, we have arrived at a diagnosis, we must next determine whether the affection is innocent and needs only patience and protection from injurious complications to pass away, or whether it demands active treatment. If so, we have to decide on either internal or external remedies or on both. We must choose the local drug to be applied and also the method of applying it—by baths or by lotions, by powders, paints, or ointments, by bandages or plasters, continuously or at intervals (*cf. supra*, p. 835 *seqq.*).

When we fail with one method we must try another, but still keeping the same end in view. Only when the case is clearly not benefited should we go back upon our diagnosis, and thus perhaps find that not so much our methods as our object was in fault, that with a more accurate and complete diagnosis, the treatment becomes efficient and successful.

57 12

INDEX OF SUBJECTS

The Roman numerals refer to the volume, the Arabic numbers to the page.

A.

- Abrin, i, 16
- Abscess of brain, i, 788
- of heart, ii, 215
- of kidney, ii, 698
- of liver, ii, 525
- of mediastinum, ii, 150
- subphrenic, ii, 483
- of tonsil, ii, 310
- Acanthosis nigricans, ii, 1014
- Acarus scabiei, ii, 850
- Acephalocysts, ii, 458
- Acetonæmia, i, 447
- Achondroplasia, i, 538
- Achorion, ii, 939
- Acne, ii, 909
- atrophica, ii, 980
- cornea, ii, 915
- frontalis, *see* A. varioliformis, ii, 914
- kéloidique, ii, 922
- rosacea, ii, 950
- varioliformis, ii, 914
- Acrochordon, ii, 918
- Acrodynia, ii, 996
- Acromegaly, i, 547
- Actinomycosis, i, 431
- Acute delirious mania, i, 982
- yellow atrophy of liver, ii, 543
- Addison's anæmia, ii, 773
- disease, ii, 723-732
- Adenoid growths, ii, 305
- Adenoma, i, 91
- sebaceum, ii, 918, 1004
- sudoriparum, ii, 1004
- Adrenals, ii, 733
- Ægophony, i, 1038, 1090
- Aërobic, i, 20
- Ætiology, i, 6
- Agraphia, i, 737, 739
- Ague, i, 335
- Alalia, *see* Aphasia, i, 735
- Albinism, ii, 1011
- Albumen, tests for, ii, 606-9
- Albuminuria, i, 118, 162, 172, 177, 185, 198,
238, 275, 292, 311, 327; ii, 606-615
- Albuminuric retinitis, ii, 623
- Alcoholic cirrhosis, ii, 533, 536
- Alcoholic dyspepsia, i, 948
- insanity, i, 996
- neuritis, i, 595
- neuroses, i, 942, 947
- paralysis, i, 595, 639
- Aleppo boil, ii, 996
- Alexia, i, 737
- Alkaptonuria, ii, 578
- Allorhythmia, i, 175
- Alopecia, ii, 942, 945
- Ammoniacal urine, ii, 589
- Amnesia, i, 738
- Amœba of malaria, i, 397
- Amphoric breathing, i, 1031, 1111
- percussion-note, ii, 1025
- vocal resonance, ii, 1111
- Amyloid disease, *see* Lardaceous, ii, 614
- Anæmia, ii, 749
- idiopathica, ii, 773
- lymphatica, ii, 767
- of cord, i, 633
- parasitica, ii, 466, 475
- perniciosa, ii, 773
- splenica, ii, 766
- Anæmic murmurs, ii, 753
- Anæsthesia, i, 573
- hysterical, i, 958
- Aneurysm, ii, 279-283
- abdominal, ii, 291
- cardiac, ii, 213
- cerebral, i, 726
- dissecting, ii, 293
- thoracic, ii, 283
- Angina, *see* Cynanche, ii, 302
- pectoris, ii, 183-193
- vaso-motoria, ii, 188
- Angiokeratoma, ii, 1005
- Angioma, ii, 1005
- Anidrosis, ii, 925
- Ankle-clonus, i, 608
- Ankylostomum, ii, 466
- Anopheles, i, 398
- Anorexia nervosa, i, 971
- Anosmia, i, 591
- Anthraxis of lungs, ii, 71
- Anthrax, i, 415-423
- Aortic aneurysm, ii, 280

- Aortic (sigmoid) incompetence, ii, 250
 — — stenosis, ii, 249
 Apex in phthisis, ii, 39
 Aphasia, i, 735-745
 Aphonia, ii, 113, 121
 Aphthæ, ii, 298
 — epizooticæ, i, 428
 Apoplexy, i, 746-761
 Appendix, inflammation of, ii, 402
 Area, ii, 943
Argyll Robertson pupil, i, 695
 Arsenical keratosis, ii, 953
 — paralysis, i, 851
 — pigmentation, ii, 1014
 Arteries, cerebral, disease of, i, 717
 — — embolism, i, 718
 — — in *Bright's* disease, ii, 628
 — — rupture, i, 723
 — — thrombosis, i, 719
 Arterio-capillary fibrosis, ii, 627
 Arthritis, i, 465
 — atrophica, i, 696
 — deformans, i, 518
 — gonorrhœal, i, 511
 — rheumatoid, *see* Osteo-arthritis, i, 518
 Arthropathy in tabes, i, 696
 Ascaris lumbricoides, ii, 461
 Ascending paralysis, i, 637
 Ascites, ii, 493-500
 Asthma, ii, 94-102
 Ataxia, i, 692
 — cerebellar, i, 707
 — *Friedreich's*, i, 705
 Ataxic spastic paraplegia, i, 705
 Atelectasis, i, 1055
 Atheroma, ii, 281, 282
 Athetosis, i, 737
 Atrophic paralysis, i, 661, 676
 — — acute, i, 662, 667
 — — chronic, i, 668
 — — — with spasm, i, 672
 — — of adults, i, 667
 — — of children, i, 662
 — — primary, i, 684
 — — subacute, of *Duchenne*, i, 675
 Atrophy, linear, of skin, ii, 964
 — of brain, i, 847
 — of kidney, ii, 687
 — of liver, ii, 542
 — myopathic, i, 684
 — progressive muscular, i, 668
 Auditory vertigo, i, 932
 Aura, i, 914
 Auscultation, i, 1028
 — of breathing, i, 1030
 — of heart, ii, 164, 243-249
 — of œsophagus, i, 311
 — of the voice, i, 1036
 Azoturia, ii, 570
- B.
- Bacillus anthracis, i, 416
 — diphtheriæ, ii, 321
 — febris flavæ, i, 296
 — (*Spirillum*) febris recurrentis, i, 257
 Bacillus influenzae, i, 229
 — lepræ, ii, 993
 — lupi, ii, 977
 — mallei, i, 425
 — (streptococcus) of erysipelas, i, 307
 — tetani, i, 374
 — tubercularis, i, 367
 — typhoides, i, 138
 — (*Vibrio*) cholerae, i, 282
 Bacteria, i, 19, *see* Microphytes
 Baruria, ii, 570
Basedow's disease, ii, 743
 Baths in treatment of fever, i, 151
Bazin's disease, ii, 986
 Beaded hair, ii, 946
 Beading of ribs, i, 532
 Bedsores, i, 156, 611
 Bell-sound, i, 1112
Bell's palsy, i, 577
 Beri-beri, i, 303
 Bile acids in urine, ii, 507
 Bilharzia hæmatobia, ii, 475, 717
 Biliary calculi, ii, 517
 — cirrhosis, ii, 534, 540
 Bilious cachexia, ii, 502
 Bilirubin in urine, ii, 506
 Birth-palsy, i, 896
 Black-water fever, i, 391
 Bladder in diabetes, i, 449
 — in paraplegia, i, 609
 Blood, diseases of, ii, 747-807
 — in *Addison's* anæmia, ii, 769
 — in anæmia, ii, 749-753
 — in chlorosis, ii, 785
 — in enteric fever, ii, 132
 — in *Hodgkin's* disease, ii, 769
 — in leuchæmia, ii, 760, 766
 — in pneumonia, ii, 5
 — in purpura, ii, 800
 — in scurvy, ii, 990
 Blue line, ii, 370
 Bothryocephalus, ii, 454
 Bovine tuberculosis, ii, 80
 Brachial neuralgia, i, 558
 — paralysis, i, 567
 Bradycardia, i, 229; ii, 170, 508
 Brain, disease of, *see* Cerebral
 Brassfounders' ague, i, 953
Bright's disease, ii, 601-671
 Bromidrosis, ii, 926
 Bronchial breathing, i, 1032
 Bronchiectasis, i, 1069; ii, 29
 Bronchitis, i, 1049-1078
 — plastic, i, 1078-1082
 Bronchocele, ii, 743
 Bronchophony, i, 1037
 Broncho-pneumonia, ii, 23-26
 Bruit d'airain, i, 1112
 — de diable, ii, 753
 — de pot fêlé, i, 1026; ii, 53
 — de souffle, ii, 244
 — d'oboe, ii, 244
 Bubonic plague, i, 261
 Bucnemia, ii, 964
 Bulbar paralysis, i, 677
 — — acute, i, 682
 Bulimia, ii, 332
 Burmese ringworm, ii, 996

C.

Cachexia strumipriva, i, 1019
Caisson-palsy, i, 534
Calcic carbonate as a calculus, ii, 675
 — oxalate as a calculus, ii, 673
 — as a urinary deposit, ii, 885
 — phosphate as a calculus, ii, 675
 — as a urinary deposit, ii, 585
 — sulphate as a urinary deposit, ii, 589
Calculi, biliary, ii, 517
 — phthisical, ii, 43, 51
 — urinary, ii, 672-703
Callosities, ii, 953
Cancer, i, 74, 91
 — bodies, i, 92
 — of bile-duct and gall-bladder, ii, 554
 — of bowel, ii, 416
 — of kidney, ii, 709
 — of larynx, ii, 139
 — of liver, ii, 551
 — of lung, ii, 159
 — of œsophagus, ii, 308
 — of pancreas, ii, 737
 — of peritoneum, ii, 492
 — of skin, ii, 1008-9
 — of stomach, ii, 353-364
 — of vertebræ, i, 614
 — seat of, i, 78, 95
Cancroid, i, 93
Capillary bronchitis, i, 1053
Carbuncle, ii, 924
 — in diabetes, i, 446
 — in plague, i, 263
Carcinoma, i, 91-103, *see* *Cancer*
Cardiac disease in children, ii, 278
 — in old age, ii, 279
 — dropsy, ii, 240
 — hypertrophy and dilatation, ii, 195-209
 — murmurs, ii, 244-249
 — rupture, ii, 27
Cardialgia, ii, 177
Cardio-vascular changes in Bright's disease, ii, 625
Carnification, i, 1056
Caseation, i, 365
Casts (cylinders or tube casts), ii, 615
Catalepsy, i, 971
Cataract, diabetic, i, 446
Catarrhal pneumonia, ii, 23
Causalgia, i, 574
Causation of disease, i, 6
Cavernous, *see* *Amphoric*, i, 1031
Cavities, *see* *Vomicæ*, ii, 1012
Cerebellar ataxia, i, 707
 — tumour, i, 777
Cerebral abscess, i, 788
 — atrophy, i, 847
 — diseases, i, 716 *seqq.*
 — embolism, i, 718
 — hæmorrhage, i, 723-749
 — sclerosis, i, 848
 — softening, i, 761
 — tumours, i, 764-788
Cerebro-spinal fever, i, 815
Certificates of insanity, i, 1002 *seqq.*
Cestoid worms, ii, 447
Chancre, i, 339

Charbon, *see* *Anthrax*, i, 415
Charcot's crystals, ii, 101
 — joint disease, i, 528
Cheirpompholyx, ii, 886
Cheloid, ii, 697
Chemiotaxis, i, 23, 51
Cheyne-Stokes breathing, i, 10-13
Chicken-pox, i, 205
Chilblain, ii, 952
Child-crowing, ii, 115
Children, cardiac disease, ii, 278
 — cirrhosis of liver, with large spleen, ii, 537
 — constipation, ii, 376
 — diabetes, i, 458
 — dyspepsia, ii, 327
 — enteric fever, i, 121, 148
 — osteo-arthritis, i, 524
 — phthisis, ii, 62
 — pneumonia, ii, 17
 — psoriasis, ii, 872, 875
 — rheumatism, i, 492
 — scabies, ii, 852
Chiragra, i, 465
Chloasma, ii, 1015
Chlorosis, ii, 783
Cholecystitis, ii, 522
Cholelithotomy, ii, 522
Cholera, i, 270-291
Cholesteatoma, i, 766; ii, 918
Cholesterine, ii, 518
Chondroma, i, 81
Chorea, i, 880-895
 — *Huntington's*, i, 895
 — major, i, 859, 973
 — post-hemiplegic, i, 735
Choroid, tubercles of, ii, 92
Chromidrosis, ii, 926
Chylous ascites, ii, 497
Chyluria, ii, 714
Cirrhosis of kidneys, ii, 653
 — of liver, ii, 533
 — of lung, ii, 27
 — of pancreas, ii, 736
 — of stomach, ii, 365
Classification, i, 11, 104, 435
 — of diseases of skin, ii, 810
Clavus, ii, 953
Clergyman's sore throat, ii, 122
Clonus, i, 608
Cnidosis, *see* *Urticaria*, ii, 900
Cœnurus, ii, 449
Co-existence of specific diseases, i, 726
Cog-wheel respiration, ii, 52
Colic, biliary, ii, 518
 — intestinal, ii, 367
 — plumbic, ii, 368
 — renal, ii, 682
Colitis, catarrhal, ii, 383
 — in *Bright's* disease, ii, 622
 — ulcerative, ii, 384
Collapse of lung, i, 1088
Colloid cancer, i, 94
 — milium, ii, 1004
Colotomy in membranous colitis, ii, 385
 — in obstruction, ii, 439, 440
Coma in apoplexy, i, 744
 — in diabetes, i, 447
 — in uræmia, ii, 631, 632

Coma-vigil, i, 119
 Comedo, ii, 914
 Composite portraits in phthisis, ii, 68
 Compression of intestine, ii, 413
 — paraplegia due to, i, 612
 Concretions, gastric, ii, 365
 — phthisical, ii, 50
 Condyloma, i, 884; ii, 957
 Confluent smallpox, i, 195
 Congenital cardiac disease, ii, 274
 — ichthyosis, ii, 960
 — idiocy, i, 1010
 — syphilis, i, 354
 — — of larynx, ii, 131
 Conjugate deviation, i, 730
 Consecutive Bright's disease, ii, 662
 Consonance, i, 1037
 Constipation, ii, 373-377
 Constitution, i, 9
 Contagion, i, 15, 16, *note*, 17
 Contractures, i, 734, 960
 Copaiba rash, i, 582
 Copper test for sugar, ii, 437
 Corns, ii, 953
 Cornu cutaneum, ii, 960
 Coronary arteries, ii, 212
 — thrombosis, ii, 212
 Corpus callosum, i, 783
 Corpuscular richness of blood, ii, 750
Corrigan's pulse, ii, 251
 Cortex of brain, i, 778-782
 Cortical centres, i, 779 *seqq.*
 Corymbose smallpox, i, 197
Coster's paste, ii, 935
 Cough, i, 1044
 Cow-pox, i, 201
 Cracked-pot sound, i, 1027
 Craniotabes, i, 534
 Crateriform ulcer, ii, 1010
 Crepitation, i, 1036; ii, 7
 Cretinism, i, 1015
 Crisis, i, 35
 Criteria of specific contagia, i, 18
 Croup, i, 318
 Crutch-palsy, i, 567
Cruveilhier's paralysis, i, 668
Curschmann's spirals, ii, 101
 Cutis anserina in ague, i, 386
 — pendula, ii, 1000
 Cyanosis, i, 1050; ii, 241
 Cyclic albuminuria, ii, 612
 Cyliodroma, i, 93
 Cynanche, ii, 302
 Cyrtometer, i, 1040
 Cystic disease of brain, ii, 847
 — — of kidney, ii, 664
 — — of liver, ii, 665
 — — of ovary, ii, 496
 — — of pancreas, ii, 736
 Cysticercus, i, 767; ii, 449, 450, 452
 Cystine, ii, 584, 674
 Cystitis, i, 609

D.

Darier's disease, ii, 925
 Deafness and otorrhœa, i, 165, 177

Deafness in epidemic meningitis, i, 817
 — in paroxysmal vertigo, i, 923
 — in typhus, i, 236
 Degeneration, fatty, of heart, ii, 215
 — — of liver, ii, 557
 — — of kidney, ii, 704
 — fibrous, of heart, ii, 210
 — lardaceous, ii, 56, 644, 648
 Delhi boil, ii, 996
 Delirium tremens, i, 942
 Deltoid paralysis, i, 568
 Delusions in insanity, i, 991
 Dementia paralytica, i, 838-847
 Demodex, ii, 909
 Dengue, i, 299
 Dental neuralgia, i, 558
 Dermatitis herpetiformis, ii, 888
 — in Bright's disease, ii, 622
 — papillomatosa capillitii, ii, 921
 Dermatolysis, ii, 1000
 Dextrosigmoid affections, ii, 264
 Diabetes, i, 435-464
 — insipidus, ii, 570
 — with bronzing of skin, ii, 737
 Diabetic coma, i, 447
 Diagnosis, i, 4
 Diapedesis, i, 49
 Diarrhœa, ii, 377-383
 — in enteric fever, i, 117
 — in phthisis, ii, 46
 — lardaceous, ii, 651
 Diazo-reaction, i, 118
 Dicrotism, ii, 168
 Dilatation, acute, of stomach, ii, 318
 — chronic, of stomach, ii, 328, 360
 — of bronchi, i, 1069
 — of heart, ii, 196, 207
 Diphtheria, i, 316
 Diplegia facialis, i, 579
 Diplococcus of pneumonia, ii, 14
 Disease and health, i, 3
 Disinfection, i, 28
 Dislocation of abdominal viscera, ii, 557
 — of thoracic viscera, i, 1113
 Dissecting aneurysm, ii, 293
 Disseminated sclerosis, i, 708
 Distribution of cancer, i, 100
Dittrich's plugs, i, 1072
 Divers' palsy, i, 634
Dochmius duodenalis, *see* *Ankylostomum*.
 Dothientérie, i, 112
 Dracunculus medinensis, ii, 474-5
 Drug-eruptions, ii, 904
Dubini's disease, i, 896
 Duct-cancer, i, 93
 Dulness and resonance, i, 1023-4
 Duodenal ulcer, ii, 352
 Dura mater, hæmatoma of, i, 835
 — — inflammation of, i, 836
 — — thrombosis of, i, 833
 Dyscrasia, ii, 505, *note*
 Dysentery, ii, 386-401
 Dysidrosis, ii, 927
 Dyspepsia, acute, ii, 314
 — chronic, ii, 320
 Dyspnœa, i, 1040
 — phrenic, i, 1041

E.

Echinococcus, ii, 449, 458, 736
 Echinorhynchus, ii, 475
 Eclampsia, i, 925-7
 — nutans, i, 859
 Ecstasy, i, 973
 Eczema, ii, 821-845
 — local forms and treatment, ii, 843
 — marginatum, ii, 937-8
 — seborrhœic, ii, 830
Ehrlich's test, i, 118
 Elephantiasis Arabum, ii, 965
 — Græcorum, ii, 991
 Embolism, i, 64, 718
 — in chorea, i, 893
 — of cerebral arteries, i, 718
 — of pulmonary artery, ii, 161
 Emphysema, i, 1059-1068
 Empyema, i, 1005, 1086, 1094
 Enchondroma, i, 81
 Endarteritis obliterans, ii, 627
 Endocarditis, acute, ii, 232
 — chronic, ii, 234
 — in chorea, i, 882, 885
 — in rheumatism, i, 487
 — in scarlatina, i, 176
 — ulcerative, ii, 234
 Enteric fever, i, 111-157
 Enteritis, ii, 383
 Enteroptosis, ii, 556
 Entozoa, ii, 446
 Ephelides, ii, 1014
 Epidemic fevers, i, 106
 — meningitis, i, 815
 Epidermolysis bullosa, ii, 888
 Epilepsy, i, 913-931
 Epileptic idiocy, i, 1010
 — mania, i, 936
 Epistaxis, i, 116, 165
 Epithelioma, i, 90, 93
 Equinia, i, 424
Erb's juvenile muscular atrophy, i, 685
 — paralysis, i, 568, 569
 Ergotism, i, 870
 Erysipelas, i, 305-314
 Erythema, ii, 892
 — bullosum, ii, 896
 — elevatum diutinum, ii, 895
 — induratum, ii, 986
 — multiforme, ii, 895
 — nodosum, ii, 899
 Erythrasma, ii, 939
Esbach's fluid, ii, 609
 Eustrongylus gigas, ii, 468
 Exanthema, i, 116, 162, 172, 184, 190, 192,
 206, 235, 275, 301, 302
 Exfoliative dermatitis, ii, 876
 Exudation, i, 45

F.

Facial neuralgia, i, 556
 — paralysis, i, 577
 — spasm, i, 853
 Farcy, i, 426

Fasciola hepatica, ii, 475
 Fastigium, i, 35
 Fatigue-spasms, i, 860
 Fatty stools, ii, 513
 Favus, ii, 939
 Febricula, i, 157
 Festination, i, 876
 Fever, i, 37
 Fevers, i, 105-110
 Fibroma, i, 81
 — cutis, ii, 999
 Filaria sanguinis, ii, 472, 474
 Flatness, *see* Dulness, i, 1023
 Floating kidney, ii, 720
 — spleen, ii, 740
 Fluid vein, i, 1033-1035
 Fœtid bronchitis, i, 1071
 Fomites, i, 15
 Foot-and-mouth disease, i, 428
 Fortification-forms in migraine, i, 899
 Framboesia, ii, 995
Friedreich's ataxia, i, 705
 — spasms, i, 895
 Fungi, i, 19
 Furunculi, ii, 922
 Fusible calculus, ii, 675

G.

Gall-stones, ii, 517-523
 Gangrene, i, 54
 Gastralgia, ii, 329
 Gastric carcinoma, ii, 353
 — concretions, ii, 365
 — dilatation, acute, ii, 318, 360
 — — chronic, ii, 328
 — — from pyloric obstruction, ii, 357
 — induration, ii, 365
 — ulcer, ii, 341
 Gastritis, acute catarrhal, ii, 314
 — chronic, ii, 320-322
 — phlegmonous, ii, 319
 Gastro-colic fistula, ii, 361
 Gastro-cutaneous fistula, ii, 343
 Gastrodynia, ii, 329
 Gastrojejunostomy, ii, 364
 Gastrostomy, ii, 312
 General paralysis of the insane, i, 838
 German measles, i, 182
 Giant-cells, i, 57, 364
Gilles de la Tourette, maladie de, i, 860
 Glanders, i, 424
 Glandular fever, ii, 741
Glénard's disease, ii, 557
 Glioma of brain, i, 767
 Globus hystericus, i, 961
 Glossy skin, i, 574
 Glycosuria, i, 437, 442, 451
Gmelin's test, ii, 506
 Goitre, endemic and sporadic, ii, 743
 — exophthalmic, ii, 744
 Gonorrhœal scleritis, i, 515
 — synovitis, i, 511
 Gout, i, 465-482
 — and insanity, i, 998
 Gouty kidney, i, 478
 Granuloma, i, 60; ii, 983

Granuloma fungoides, ii, 1006, *note*
Graves's disease, ii, 744
 Grey granulations, i, 363
 Grippe, *see* Influenza, i, 222
 Ground-water, i, 286
 Gummata, i, 347
 Gutta rosea, ii, 950

H.

Hæmamœba malarie, i, 298
 Hæmatemesis, ii, 337, 349
 Hæmatoma of dura mater, i, 835
 Hæmatomyelia, i, 629
 Hæmaturia, ii, 678, 682, 717
 Hæmoglobinuria, ii, 593-600
 Hæmophilia, ii, 803
 Hæmoptysis, ii, 48, 242
 Hæmorrhage, cerebral, i, 723
 — from bowels, i, 122, 155
 Handicraft-spasms, i, 861
 Hay-fever, ii, 102
 Headache, i, 910
 Heart, functional disorders of, ii, 166
 Heat-stroke, i, 938
 Hemisrania, i, 899
 Hemiparaplegia, i, 647
 Hemiplegia, i, 728
 Hepatic abscess, ii, 525-533
 — atrophy, ii, 498, 542
 — cirrhosis, ii, 533
 — dropsy, ii, 494
 — syphilis, ii, 498, 542
 Hepatisation, ii, 9, 10
Henoch's purpura, ii, 803
 Herpes circinatus, ii, 896, 937
 — in pneumonia, ii, 5
 — iris, ii, 897
 — labialis *v.* faciei, ii, 896
 — præputialis, ii, 897
 — zoster, ii, 1016
 Hiccough, ii, 337
 Histrionic spasm, i, 853
Hodgkin's disease, ii, 767
 Hooping-cough, i, 214
 Humours, ii, 505, *note*
Huntington's chorea, i, 895
Hutchinson's triad, i, 358
 Hydatid, ii, 457
 — fluid and hooklets, ii, 459
 — in liver, ii, 561
 — kidney, ii, 713
 — pelvis and other organs, ii, 460, 461
 Hydroa, ii, 898
 — æstivalis, ii, 890
 — gestationis, ii, 898
 Hydrocephalic idiocy, i, 1010
 Hydrocephalus, i, 823
 Hydronephrosis, ii, 688-695
 Hydrophobia, *see* Rabies, i, 403
 Hyperidrosis, ii, 925
 Hyperkeratosis, ii, 952-3
 — centrifuga, ii, 961
 Hyperpyrexia, i, 37, 491, 505
 Hypertrophic cirrhosis of liver, ii, 540
 Hypertrophy of brain, i, 850
 — of lungs, i, 1068
 Hypnotism, i, 973
 Hypochondriacal melancholy, i, 987

Hypochondriasis, i, 976, 988
 Hypopyrexia, i, 42
 Hysteria, i, 956
 Hysterical paralysis, i, 642

I.

Ichthyosis, ii, 957
 Icterus, ii, 504
 — gravis, ii, 543
 Idiocy, i, 1009, 1010, 1015
 Idiosyncrasy, i, 7
 Imbecility, i, 1010
 Immunity, i, 23
 Impetigo, ii, 847, 854
 — herpetiformis, ii, 890
 Indican, ii, 576
 Indigo, ii, 674
 Infantile paralysis, i, 662
 Infection, i, 15, 67
 Inflammation, i, 43-60
 Influenza, i, 222-231
 Inosite, ii, 570, *note*
 Insanity, i, 979-1011
 Inspection of chest, i, 1040
 Insular sclerosis, i, 708
 Intercostal neuralgia, i, 559
 Intermittent pulse, ii, 173
 Interstitial form of Bright's disease, ii, 652
 Intertrigo, ii, 823
 Intestinal casts, ii, 385
 — contraction, ii, 414
 — impaction, ii, 412
 — obstruction, ii, 409-445
 — strangulation, ii, 425
 Intra-venous injection in cholera, i, 290
 — — in diabetes, i, 463
 Intussusception, ii, 416
 Invagination, ii, 416
 Irregular pulse, ii, 174
 Irritable heart, ii, 172

J.

Jacksonian epilepsy, i, 775
 Jaundice, ii, 504-517
 — as a symptom, ii, 509
 — epidemic, ii, 511
 — recurrent, ii, 512

K.

Kaposi's disease, ii, 1008
 Keloid, ii, 997
 Keratoid cancer, i, 93
 Keratosis (leucokeratosis) buccalis, ii, 955
 — pilaris, ii, 858
 Knee-jerk, i, 608
 — in diabetes, i, 445
 — in insular sclerosis, i, 710
 — in tabes, i, 695
 Kyestine, ii, 592

L.

Labio-glosso-laryngeal paralysis, i, 677
Landry's paralysis, i, 637

Lardaceous degeneration, ii, 614
 — form of *Bright's* disease, ii, 628
 Laryngeal affections, ii, 106-141
 — catarrh, ii, 118
 — leprosy, ii, 128
 — lupus, ii, 128
 — malformations, ii, 140
 — paralysis, ii, 108-113
 — perichondritis, ii, 134
 — spasm, ii, 115
 — syphilis, ii, 129
 — tuberculosis, ii, 124
 — tumours, ii, 136
 Laryngismus stridulus, ii, 115
 Laryngitis, acute phlegmonous, ii, 131
 — in diphtheria, i, 325
 — in enteric fever, i, 136
 — membranous, i, 316, 318; ii, 121
 Laryngoscope, ii, 106
 Lead-colic, ii, 369
 Lead-palsy, i, 597
 Legal aspects of insanity, i, 1000-1010
 Leprosy, ii, 991
 Leuchæmia, ii, 758
 Leucin in urine, ii, 545
 Leucocytes, i, 50
 Leucoderma, ii, 1011
 Leucoplakia buccalis, ii, 955
 Leucotrichia annularis, ii, 946
 Lichen, ii, 857
 — circumscriptus, ii, 857, 916
 — pilaris, ii, 858
 — planus, ii, 861
 — ruber, ii, 861
 — scrofulosorum, ii, 859
 — tropicus, ii, 857
 Lingual paralysis, i, 588
 Lipæmia, i, 449
 Lipoma, i, 81
 Lipothymia, ii, 179, *note*
 Lithæmia, ii, 503
 Lithates, *see* Urates
 Localisation in the brain, i, 775-785
 — in the cord, i, 602
 Locomotor ataxia, i, 690
 Lues, i, 335
 Lumbago, i, 509
 — renal, ii, 678
 Lupus, ii, 975
 — disseminatus, ii, 989
 — erythematosis, ii, 987
 Lymphadenoma, i, 83; ii, 741, 768
 Lymphangioma, ii, 1006
 Lymph-glands, abscess, etc., ii, 741
 Lymphoma, i, 83
 Lympho-sarcoma, i, 83
 Lymph-scrotum, ii, 715
 Lysis, i, 35, 119

M.

Malacosteon, i, 543
 Malaria, i, 384, 392
 Malformation of kidney, ii, 719
 Malignancy, i, 74-79
 Malignant pustule, i, 418
 Malleation, i, 859
 Mallein, i, 425

Malta fever, i, 302
 Mania, i, 983
 — acute delirious, i, 983
 — paroxysmal, i, 936
 Measles, i, 160-168
 Mediastinal growths, ii, 152
 — induration, ii, 151
 — suppuration, ii, 149
 Medicinal rashes, ii, 904
 Mediterranean fever, i, 302
 Melancholia, i, 987, 988
 Melanodermia, ii, 1013
 — arsenical, ii, 1014
 Melasma suprarenale, ii, 723, 725
Ménière's disease, i, 932
 Meningeal hæmorrhage, i, 157, 835, 896
 — tumour, i, 615
 Meningitis, basal, i, 812
 — cerebral, i, 197
 — epidemic, i, 813
 — spinal, i, 657
 Mentagra, ii, 919
 Mental disorders, i, 979
 Meralgia paræsthetica, i, 560
 Mercurial tremors, i, 952
 Metallic tinkling, i, 1112
 Miasm, i, 16
 Microbe, microphyte, or micro-organism, i, 20
 Micrococcus, i, 19
 Microphytes of anthrax, i, 418
 — of cholera, i, 282
 — of diphtheria, i, 320
 — of enterica, i, 138
 — of erysipelas, i, 307
 — of glanders, i, 425
 — of gonorrhœa, i, 517
 — of influenza, i, 229
 — of leprosy, ii, 993
 — of Malta or Mediterranean fever, i, 303
 — of relapsing fever, i, 257
 — of tetanus, i, 374
 — of tubercle, i, 367
 — of yellow fever, i, 296
 — specific, i, 19
 Migraine, i, 898
 Miliary fever, i, 158, 269
 — tubercles, i, 363, 366
 — tuberculosis, ii, 89
 Milium, ii, 914
 Miners' nystagmus, i, 866
 Mitral incompetence, ii, 259
 — stenosis, ii, 254
 Mollities ossium, i, 543
 Molluscum, ii, 1001
 Moral insanity, i, 997
 Morbilli, i, 160
 Morphia habit, i, 934
 Morphœa, ii, 962
Morton's neuralgia, i, 560
 Mosquitoes and ague, i, 398
 Moulds, i, 19
 Moveable kidney, ii, 720
 Mumps, i, 211-213
 Muscular hypertrophy, i, 689
 Myalgia, i, 509
 Myasthenia gravis, i, 683
 Mycosis fungoides, ii, 1006-7
 Myelitis, acute, i, 621

Myocarditis, acute, ii, 215
 Myoclonus multiplex, i, 895
 Myopathic paralysis, i, 684-689
 Myotonia congenita, i, 872
 Myxœdema, i, 1017, 1027
 Myxoma, i, 83

N.

Nails, diseases of, ii, 946
 Nematoid worms, ii, 461
 Neoplasms, i, 73
 Nephralgia, ii, 682
 Nephrorrhaphy, ii, 722
 Neuralgia, i, 553
 Neurasthenia, i, 643
 Neuritis migrans, i, 569
 — peripheral, i, 591-599
 Neurodermatoses, ii, 1021
 Neuroses from alcohol, i, 942
 — from morphia, i, 950
 New growths, i, 73
 Nodes, i, 347
 Nodi digitorum, i, 521
 Nomenclature, i, 12
 Nosology, i, 3, 104
 Notification of infectious diseases, i, 110
 Nyctalopia, ii, 791
 Nystagmus, i, 709
 — miners', i, 866

O.

Obesity, ii, 559
 Obstruction of the bowels, ii, 409-445
 Ocular paralysis, i, 582-587
 Œsophagus, dilatation of, ii, 308
 — stricture of, ii, 308, 311
 Ophthalmoplegia, i, 587
 Opium-eating, i, 950
 Optic neuritis, i, 771
 Orthopnœa, i, 1042
 Osteal resonance, i, 1026
 Osteitis deformans, i, 546
 Osteo-arthritis, i, 518, 528
 — in children, i, 524
 Osteo-arthropathy, i, 549
 Osteoid tumour, i, 82
 Osteoma, i, 82
 Osteomalacia, i, 543
 Otorrhœa, i, 65, 117, 788
 Ovaries and testes, ii, 742
 Oxalates, ii, 585
 Oxyuris, ii, 464

P.

Pachydermia, ii, 965
 Pachymeningitis cervicalis, i, 674
 — hæmorrhagica, i, 836
Paget's disease of the nipple, ii, 846
 Palpation, i, 1039
 Palpitation, ii, 176
 Pancreas in diabetes, i, 449, 455
 Pancreatic hæmorrhage, ii, 734
 Pancreatitis, ii, 735
 Papilloma, ii, 957
 Paracentesis abdominis, ii, 499

Paracentesis thoracis, i, 1103
 Parakeratosis variegata, ii, 864
 Paralysis agitans, i, 874
 — cerebral, of children, i, 896
 — multiple peripheral, i, 591-599
 — peripheral, i, 567-591
 Paralytic insanity, i, 999
 Paramyotonus multiplex, i, 895
 Parangi, ii, 995
 Paraplegia, i, 600-656, 959
 — dolorosa, i, 617
 — spastica, i, 651
 Paroxysmal mania, i, 936
 — vertigo, i, 931
 Pea-soup stools, i, 117
 Pectoriloquy, i, 1037
 Pediculi, ii, 854
 Pelvic symptoms, i, 612
 Pemphigus, ii, 881
 — acutus, ii, 885
 — foliaceus, ii, 885
 — vegetans, ii, 886
 Pentastoma, ii, 475
 Percussion, i, 1022
 Perforating ulcer, i, 696
 — — of bowels, i, 123
 — — of stomach, ii, 343, 352
 Pericardial adhesions, ii, 225
 — effusion, ii, 227-8
 Pericarditis, ii, 218-230
 — in rheumatism, i, 489
 Pericolitis sinistra, ii, 385
 Perihepatitis, ii, 497, 541
 Peripheral neuritis, i, 591
 Peritonitis, cancerous, ii, 492
 — chronic, ii, 487
 — circumscribed purulent, ii, 483-4
 — septic, ii, 478
 — tuberculous, ii, 489
 Perlsucht, i, 369
 Pernio, ii, 952
 Pertussis, i, 214
 Pestis, i, 261
 Petit mal, i, 916
Pettenkofer's test, ii, 507
 Phagocytes, i, 24, 51
 Pharyngitis, granular, ii, 303
 — hypertrophic, ii, 305
 Phlorizin-diabetes, i, 455
 Phosphates in urine, ii, 585
 Phosphorus-poisoning, ii, 548
 Phthiriasis, ii, 854
 Phthisis, ii, 37-89
 — ab hæmoptoë, ii, 49
 — and insanity, i, 999
 — fibrous or fibroid, ii, 43
 — in diabetes, i, 446
 — recovery from, ii, 59-62
 Pigeon-chest, i, 532
 Pituitarium in acromegaly, i, 528
 — tumour of, i, 783
 Pityriasis capitis, ii, 915
 — rosea, ii, 876
 — rubra, ii, 871
 Plague, i, 261
 Plastic bronchitis, i, 1078
 Pleomorphism, i, 20
 Plethora, ii, 748

Pleurisy, i, 1081
 Pleuritic effusion, i, 1085
 — rub, i, 1084
 Pleurodynia, i, 1046, 1091
 Plumbic gout, i, 475
 — paralysis, i, 597, 951
 Plumbism, i, 924; ii, 24, 369, 654, 757
 — and insanity, i, 998
 Pneumococcus, ii, 14
 Pneumoconiosis, ii, 70
 Pneumonia, ii, 1–21
 — chronic, ii, 22
 — — — interstitial, ii, 27
 — epidemic, ii, 13
 — in diabetes, i, 447
 — secondary, ii, 21
 — septic, ii, 26
 Pneumonic casts, ii, 4
 Pneumothorax, i, 1107
 Podagra, i, 465
 Polyorromentitis, i, 1084
 Polyuria, ii, 569
 Porokeratosis, ii, 961
Pott's disease, i, 612
 Priapism in paraplegia, i, 611
 Prickly heat, ii, 857
 Proglottis, ii, 451
 Prognosis, i, 8
 Progressive muscular atrophy, i, 668
 — — — of children, i, 684
 Prurigo, ii, 866
 — æstivalis, ii, 865
 — hyemalis, ii, 865
 — pedicularis, ii, 854
 — senilis, ii, 854
 Pseudo-hypertrophic paralysis, i, 685
 Psilosis, ii, 382
 Psoriasis, ii, 868–875
 Psorospermia, ii, 475–6
 Ptomaines, i, 16
 Ptyalism, ii, 299
 Puerperal insanity, i, 994
 Pulmonary artery, embolism of, ii, 161
 — auscultation, i, 1031
 — cancer, ii, 159
 — catarrh, ii, 23
 — collapse, i, 1055
 — (dextrosigmoid) incompetence, ii, 265
 — — stenosis, ii, 264
 — gangrene, ii, 33
 — syphilis, ii, 30
 — tuberculosis (acute miliary), ii, 89
 — — chronic, ii, 37
 — vomicae, ii, 41
 Pulse, allorhythmic, ii, 175
 — disorders of, ii, 166 *seqq.*
 — intermittent, ii, 173
 — irregular, ii, 174
 — tension of, ii, 178
 Pulsus paradoxus, ii, 179
 Pupils in fever, i, 119, 236
 Purpura, i, 164, 175, 191, 197; ii, 798
 Pyæmia, i, 63–72
 Pylephlebitis adhæsiva, ii, 498
 — septica, ii, 522
 Pyrexia, i, 31, 37
 Pyrosis, ii, 333
 Pyuria and pyelitis, ii, 695

Q.

Quinsy, ii, 301

R.

Rabies, i, 404–414
 Radesyge, ii, 995
 Râles or rattles, i, 1035, 1051
 Ray fungus, i, 431
Raynaud's disease, ii, 600
 Reaction of degeneration, i, 570
 Reflex paralysis, i, 641
 — spasms, i, 858
 — symptoms in paraplegia, i, 606–612
 Relapses in enterica, i, 127
 — in rheumatism, i, 491
 Relapsing fever, i, 252–260
 Renal atrophy, unilateral, ii, 687
 — dropsy, ii, 617
 Resonance, i, 1025
 Retinitis in Bright's disease, ii, 623
 Rhachialgia, i, 644
 Rhachitis, i, 530
 Rheumatic fever, i, 483
 — gout, *see* Osteo-arthritis, i, 518
 — nodules, i, 489
 Rheumatism, i, 483
 — chronic, i, 508, 518
 — gonorrhœal, i, 511
 — in children, i, 493
 — muscular, i, 509
 Rheumatoid arthritis, i, 518
 Rhinoscleroma, ii, 989
 Rhonchus, i, 1035, 1050
 Rice-water stools, i, 272
 Rickets, i, 530
 Ringworm, ii, 928–939
 — of body, ii, 937
 — of nails, ii, 938
 — of scalp, ii, 929
 Risus sardonicus, i, 375
 Rodent ulcer, ii, 1009, 1010
 Rôtheln, i, 182
 Rubella, i, 182
 Rubeola, i, 160, 182

S.

Sabre-shaped tibia, i, 534
 Salaam-palsy, i, 859
 Salivation, ii, 299
 Saltatorial spasms, i, 858
 Sapræmia, i, 62
 Sarcinæ in urine, ii, 592
 Sarcoma, i, 84–87
 — multiple, i, 88–90
 Saturnine gout, i, 475
 Scabies, ii, 847
 Scarlatina, i, 169–181
 Sciatica, i, 563
 Scirrhus, i, 92
 Sclerema, ii, 963
 Scleroderma, ii, 961
 Sclerosis, disseminated, i, 703
 — insular, i, 708
 — of brain, i, 848
 — of cord, i, 649

Sclerosis of lateral columns, i, 653
 — of posterior columns, i, 698
 Sclerostomum duodenale, ii, 466
 Sclerotitis in gonorrhœa, i, 515
 Scorbutus, ii, 788
 Scrivener's palsy, i, 861
 Scrofula, ii, 980, 981
 Scurvy, ii, 788
 — and rickets, i, 536
 Seborrhœa capitis, ii, 915
 — corporis, ii, 916
 — oleosa, ii, 915
 — sicca, ii, 915
 Senile phthisis, ii, 62
 Septicæmia, i, 62
 Serratus magnus, paralysis of, i, 567
 Shingles, ii, 1016
 Sibbens, ii, 995
 Sibilus, i, 1035, 1050
 Sigmoid (aortic) valvular affections, ii, 249
 Signs and symptoms, i, 6
 Singultus, ii, 337
 Skin, diseases of, ii, 817-1029
Skodaic resonance, i, 1026
 Smallpox, i, 187
 Soil-water, i, 286
 Somnambulism, i, 972
 Souffle voilé, ii, 53
 Spasmodic neuroses, i, 853-872
 Spastic paraplegia, i, 651, 896
 Sphygmograph, ii, 167
 Sphygmometer, ii, 169
 Spinal meningitis, i, 657
 Spirillum, i, 19
 Spirochæta, i, 19
 Spirometer, i, 1041
 Spleen in ague, i, 392
 — in anthrax, i, 419, 421
 — in enterica, i, 116, 136
 — in leukæmia, ii, 760
 — in rickets, i, 535
 Splenic abscess, ii, 738
 — diseases, ii, 738-9
 — enlargement, signs of, ii, 740
 Spondylitis deformans, i, 515
 Sprue, ii, 382
 Sputa, i, 1045
 Squamous cancer, i, 93
 Staphylococci, i, 19
 Statistics of cerebral embolism, i, 719
 — of cerebral tumours, i, 768
 — of chorea, i, 888-9, 892
 — of diseases of the cord, i, 715
 — of idiopathic anæmia, ii, 778
 — of pityriasis rubra, ii, 879
 — of pleurisy, right or left side, i, 1092
 — of pneumonia, ii, 3-6, 12, 17
 — of psoriasis, ii, 872
 — of rheumatism, i, 488, 491, 500
 — of xanthelasma, ii, 966
 Status lymphaticus, ii, 721
 Steatoma, ii, 917
 Stenosis, pyloric, ii, 355
 — rectal, ii, 416
 — tracheal, ii, 142
 — valvular, ii, 237
 Stethoscope, i, 1029
 Stomatitis, ii, 297

Strabismus, i, 583
 Streptococci, i, 19
 Striæ atrophicæ, ii, 964
 Stricture of intestine, ii, 415
 Strobila, ii, 450
 Strophulus, ii, 859
 Stupor, active and passive, i, 990
 Subdiaphragmatic (subphrenic) abscess, ii, 483
 Succussion-splash, i, 1112
 Suction-sound, ii, 53
 Sudamina, ii, 927
 Sunstroke, i, 938
 Suppression of urine, ii, 684
 Sweating sickness, i, 268
 Sycosis, ii, 919
 — frambœsiformis capillitii, ii, 921
 — parasitic, ii, 921
 Symptoms and signs, i, 5
 Syncope, ii, 179
 Synocha, i, 158
 Syphilides of the skin, ii, 969
 Syphilis, i, 335-360
 Syphilitic arteritis, ii, 283
 — cirrhosis of lung, ii, 30
 — disease of liver, ii, 542
 — — of myocardium, ii, 211
 — insanity, i, 998
 — laryngitis, ii, 129
 — meningitis, i, 814
 — paraplegia, i, 624
 — stricture of rectum, ii, 416
 Syringomyelia, i, 631

T.

Tabes dorsalis, i, 690
 Tachycardia, ii, 171
 Tænia echinococcus, ii, 457
 — mediocanellata, ii, 452
 — solium, ii, 450
 Tapeworms, ii, 447, 455
 Temperament, i, 9, 10, 505
 Temperature, i, 37
 — subnormal, i, 42
 Tender points, i, 555
 Tetanilla (tetany), i, 866
 Tetanus, i, 373
 Therapeutics, i, 10
 Thermometry, i, 33
 Thermotaxy, i, 32
Thomsen's disease, i, 872
 Thrombosis of cerebral arteries, i, 719
 — — sinuses, i, 833
 — of veins, i, 126
 Thrush, ii, 298
 Thyroid compressing trachea, ii, 143
 — diseases of, ii, 742
 Tic convulsif, i, 853
 — douloureux, i, 556
 — mental, i, 860
 Tinea capitis, ii, 929
 — corporis, ii, 937
 — marginata, ii, 937
 — versicolor, ii, 940
 Tonsillitis, acute, ii, 310
 — follicular, ii, 300
 — hypertrophic, ii, 302

Tonsils, growths of, ii, 307
 Tophi, i, 468
 Tracheal obstruction, ii, 145
 — stenosis, ii, 142-145
 — ulceration, ii, 144
 Trance, i, 792
Traube's plugs, i, 1072
 Trematoda, ii, 475
 Tremors, i, 709, 874, 952, 954
Trichina spiralis and trichiniasis, ii, 468
Trichocephalus dispar, ii, 465
 Trichoclasia, ii, 946
 Tricuspid incompetence, ii, 265
 — stenosis, ii, 265
 Tubal nephritis, ii, 636
 Tubercle of adrenals, ii, 725
 — of brain, i, 764, 797
 — of intestines, ii, 381
 — of kidney, ii, 704
 — of larynx, ii, 124
 — of liver, ii, 560
 — of lungs, ii, 37
 — of skin, ii, 975, 986
 Tuberculosis, i, 362
 Tubular breathing, i, 1032
 Tumours, i, 73
 Tylosis, ii, 953
 Tympanic dulness and resonance, i, 1026
 Tympanites, i, 118
 Typhlitis, ii, 403-408
 Typhoid fever, i, 111-157
 Typhus, i, 232-250
 — abdominalis, i, 111
 Tyrosin in urine, ii, 545

U.

Ulnar paralysis, i, 569
 Uræmia, ii, 630-636
 Uric acid as calculus, ii, 673
 — — as deposit, ii, 579
 — — in gout, i, 468
 — — supposed diathesis, ii, 503
 Urobilin, ii, 575
 Uro-erythrin, ii, 577
 Uro-stealith, ii, 674
 Urticaria, ii, 900
 — pigmentosa, ii, 902

V.

Vaccination, i, 201, 204

Vaccinia, i, 201, 203
 Valvular disease, ii, 237
 — — effects of, ii, 238-240
 Varicella, i, 205-210
 — prurigo, i, 209
 Variola, i, 187-201
 Veins, arborescent girdle of, i, 1057
 Venesection, ii, 19, 20
 Verrucæ, ii, 956
 Vertebral disease, i, 612, 614, 615
 Vertigo, i, 770, 931, 932, 934
 Vibrio, i, 19, 282
 Vitiligo, ii, 1012
 Vitiligoidea, *see* Xanthoma, ii, 966
 Vocal fremitus, i, 1039, 1040
 Volvulus, ii, 427
 Vomitus, ii, 334

W.

Warts, ii, 956
 Weak-minded patients, i, 992
Weil's disease, ii, 511
 Whooping-cough, i, 214
Woillez, maladie de, ii, 22
 Worms, ii, 446
 Writer's cramp, i, 861
 Wryneck, i, 854

X.

Xanthelasma, ii, 966
 Xanthin as a calculus, ii, 674
 — as a urinary deposit, ii, 585
 Xanthoma, ii, 966, *note*
 — diabetorum, ii, 968
 Xerodermia, ii, 917, 959
 — maligna, ii, 1008
 — pigmentosa, ii, 1008, *note*

Y.

Yaws, ii, 995
 Yellow atrophy of liver, ii, 542
 — fever, i, 291-298

Z.

Zinc, toxic effects of, i, 953
 Zona, ii, 1016
 Zoster, ii, 1016

INDEX OF AUTHORS

** * The Roman numerals refer to the volume, the Arabic numbers to the page, and those enclosed in brackets to the date of the Author, or to that of the work quoted.*

A.

ABERCROMBIE, JOHN, of Edinburgh (1781-1844), i, 747, 806, 975
 ACLAND, THEODORE, i, 204, 431; ii, 87
 ADAMI, of Montreal, ii, 207, 537, 543, *note*, 743
 ADDISON, THOS. (1793-1860), i, 36; ii, 3, 9, 22, 38, 402, 558, 723, 775, 961, 966, 998
 AIRY, HUBERT, i, 899
 ALBRECHT, ii, 425, *note*
 ALDERSMITH, ii, 931, 934
 ALIBERT (*ob.* 1837), ii, 997, 1006
 ALLBUTT, i, 771, 773, 803, 881; ii, 200, 206
 ANDERSON, MCCALL, of Glasgow, ii, 836
 ANDRAL (*ob.* 1876), ii, 533
 ANSTIE, F. E. (1874), i, 555, 883, 901, 945, 946, 996
 ARBUTHNOT (1735), ii, 14, 793
 ARETÆUS, of Cappadocia (*fl.* 70), i, 271, 436, *note*; ii, 2
 ASHBY, HENRY, of Manchester (Ashby and Wright), i, 176; ii, 358
 AUENBRUGGER (1761), i, 1021
 AUSPITZ, of Vienna (*ob.* 1886), i, 869, 874, *note*, 878, 882

B.

BABES, of Bucharest, i, 414, 428
 BABINGTON, BENJ. GUY (son, *ob.* 1866), ii, 106
 BABINGTON, WILLIAM (*ob.* 1833), i, 487, 891
 BADCOCK (1845), i, 203
 BAGINSKY (1896), i, 333, 652
 BAGLIVI, of Rome (*ob.* 1707), i, 149
 BAILLIE, MATTHEW (1761-1828), ii, 533, 537
 BAKER, SIR GEORGE (*ob.* 1809), i, 223; ii, 369, 389, *note*
 BAKER, MORRANT, i, 99; ii, 903, 922, 935
 BALFOUR, GEO., of Edinburgh, ii, 187, 290
 BALLARD, i, 144; ii, 378
 BALZER, ii, 918, 962, 1004 (*see also* Besnier)
 VON BAMBERGER (*ob.* 1888), i, 858
 BANCROFT, of Brisbane, ii, 472
 VON BÄRENSPRUNG (*ob.* 1864), ii, 1001, 1016, 1018, 1019

BARKER, ii, 364
 BARLOW, SIR THOMAS, i, 536, 812
 BARR, JAMES, of Liverpool, i, 153
 BARRS, of Leeds, i, 1101, *note*
 BARTELS, of Kiel (*ob.* 1878), i, 1093; ii, 582, 636
 BARTHOLINUS, THOMAS, of Copenhagen (1654), i, 315
 BASSEREAU, i, 339
 BASTIAN, CHARLTON, i, 618, *note*, 622, 761; ii, 28, 461
 BATEMAN (*ob.* 1831), ii, 859, 919, 939-1001
 BÄUMLER, of Freiburg, i, 34, 158, 328; ii, 49, 109, 218, 425
 BAXTER, BUCHANAN (*ob.* 1885), i, 29, 348; ii, 878
 BAYLE (1810), i, 362; ii, 9
 BAZIN (*ob.* 1878), ii, 888, 980, 986
 BEACH, FLETCHER, i, 850, 1014
 BEARD and ROCKWELL (1883), i, 643
 BEATTY, WALLACE, of Dublin, ii, 888
 BÉCHAMP, i, 20
 BECK, MARCUS (*ob.* 1893), ii, 662
 BEHRING, E. A., i, 333
 BENNETT, ALEX. HUGHES (son, 1885), i, 698
 BENNETT, J. HUGHES, of Edinburgh (*ob.* 1875), ii, 18, 87, 758, 836
 BENNETT, SIR J. RISDON (*ob.* 1891), ii, 154
 BERNARD, CLAUDE (*ob.* 1878), i, 450
 BEEVOR, C. E., i, 680
 BEEVOR, SIR HUGH, ii, 79, 82, *note*
 BESNIER, ii, 968, 982, 988, 1001
 BIDDER, of Dorpat (*ob.* 1894), i, 93
 BIERMER, of Zurich (*ob.* 1892), i, 1079; ii, 774
 BIETT (*ob.* 1840), ii, 970, 987-999
 BILHARZ (1852), ii, 454, 466, 717
 BILLROTH, of Vienna (*ob.* 1894), i, 19, *note*, 311, 364
 BLACKLEY, of Manchester, ii, 103
 BLANDFORD, i, 840, 845
 BOECK, CÆSAR, ii, 875, 925
 BOERHAAVE (*ob.* 1738), i, 63
 BOGHURST, i, 263, *note*
 BOLLINGER, i, 403, 425, 431; ii, 206
 BOTKIN, of St. Petersburg, ii, 19
 BOUCHARD (1889), i, 726, 734
 BOUILLAUD (*ob.* 1881), i, 487, 739; ii, 18, 753

BOWDITCH, H. I., of Boston (*ob.* 1892), ii, 74
 BOWLBY, ANTHONY, i, 154, *note*, 642
 BOWLES, ROBERT, i, 200, 745; ii, 830
 BOX, ii, 732
 BRADFORD, J. R., ii, 633, 635
 BRADSHAW (1898), i, 544
 BRAMWELL, BYROM, i, 658, *note*; ii, 782
 BRANDE, of Stettin, i, 704
 BREHMER, ii, 85
 BRETONNEAU, of Tours (1862), i, 112, 315
 BRIEGER (1885), i, 66, 139
 BRIGHT, RICHARD (*ob.* 1858), i, 393, 1055;
 ii, 543, 601
 BRINTON (*ob.* 1867), ii, 345-8, 358, 417, 434
 BRISTOWE, J. S. (*ob.* 1895), i, 79, 217, 783,
 1026
 BROADBENT, Sir WM., i, 724, 729; ii, 172
 BROCA (*ob.* 1880), i, 738
 BROCKBANK, ii, 518
 BROCCQ, ii, 925
 BRODIE, Sir BENJ. (*ob.* 1862), ii, 147, 172
 BRODIE, T. G., i, 97, 334
 BROOKE, of Manchester, ii, 875, 1004
 BROUSSAIS (*ob.* 1838), i, 112
 BROWN, THOS., of Glasgow, i, 646
 BROWNE, Sir CRICHTON, i, 918, 928
 BROWN-SÉQUARD (*ob.* 1894), i, 641, 647
 BRUCE, i, 303
 BRUNTON, Sir LAUDER, ii, 188, 192
 BRUNTON and TUNNICLIFFE, ii, 272, *note*
 BRYANT, THOS., ii, 439
 BUCHANAN, Sir GEORGE (*ob.* 1895), i, 238,
 295; ii, 378
 BÜCHNER, i, 20, 417, *note*
 BUDD, GEORGE (*ob.* 1882), ii, 526
 BUDD, WILLIAM (*ob.* 1880), i, 140, 656
 BUHL, of Munich, i, 145, 320
 BULKLEY, of New York, ii, 898, 904, 910, 940
 BULLOCK, i, 333
 BUNGE (1889), ii, 787
 BURDACH, i, 660
 BURTON, ii, 370
 BURY, JUDSON, of Manchester, i, 592
 BUSK, GEO. (*ob.* 1886), i, 309
 BUZZARD, THOS., i, 226, 329, 592, 683, 701,
 714, 789

C.

CABOT, of Boston, i, 118; ii, 6, *note*
 CADGE, of Norwich, ii, 676
 CALMEIL (*ob.* 1895), i, 926
 CALMETTE, of Lille (1896), i, 267
 CAMPBELL, H. J., of Bradford, i, 449; ii,
 40, 80, 525
 CANTANI, of Naples, ii, 87
 CANTLIE, ii, 483
 CARSWELL (*ob.* 1857), i, 708
 CARTER, VANDYKE, of Bombay, i, 253, 258
 CATON, RICHARD, of Liverpool, i, 507
 CAUTLEY, i, 265; ii, 786
 CAVAFY, JOHN (*ob.* 1901), i, 704; ii, 623,
 593, 968, 989
 CAYLEY, i, 121, 140, 151, 1106; ii, 316
 CAZENAVE, i, 312; ii, 857, 987, *note*
 CEELEY, i, 203
 CELSUS (*fl.* c. 10 A.D.), i, 74, *note*, 271, 378,
 388, 463, 915; ii, 38, 362, *note*, 387, *note*

CHAMBERS, T. K., ii, 323, 334, 361
 CHAPMAN, of Hereford, ii, 253
 CHARCOT (*ob.* 1893), i, 528, 608, 611, 673,
 674, 694, 696, 709, 723, 733, 957, 960, 970;
 ii, 540, 685
 CHAUVEAU, i, 17
 CHÉADLE, i, 186, 536, 540, 870; ii, 798, 540
 CHEVERS, NORMAN, ii, 395
 CHEYNE, GEORGE, of Bath (*ob.* 1743), i, 406
 CHEYNE, JOHN, of Dublin (*ob.* 1836), i, 249,
 318, 1043
 CHEYNE, WATSON, i, 29; ii, 802
 CHILDE, L. F., i, 264
 CHOMEL, i, 496, 591
 CHOSSAT, i, 40, 540
 CHRISTISON, Sir ROBERT (*ob.* 1882), i, 255
 CHURCH, Sir WM., i, 488, 491, 503; ii, 154,
 709, 968
 CLARK, Sir ANDREW (*ob.* 1893), ii, 55, 100
 CLARKE, LOCKHART (*ob.* 1808), i, 373, 660,
 671; ii, 109
 CLEVELAND, ii, 744, *note*
 CLOUSTON, ii, 395
 COATS, of Glasgow (*ob.* 1901), ii, 27
 COBBOLD (*ob.* 1886), ii, 453, 457
 COHN, i, 19, 20
 COHNHEIM, of Leipzig (*ob.* 4884), i, 31, 42,
 45, 46, 98-9, 278, 362, 367, 686; ii, 591,
 619, 692
 COLLES, of Dublin, i, 348
 COLLIE, ALEXANDER, i, 140, 147, 152, 179,
 200, 255, 260
 COOPER, Sir ASTLEY (*ob.* 1841), i, 511
 COPEMAN, i, 188, 204; ii, 600
 CORNIL, i, 310
 CORNIL and RANVIER, i, 468
 CORRIGAN, Sir DOMINIC, of Dublin (*ob.*
 1880), ii, 27, 248
 CORVISART, J. N. (*ob.* 1821), i, 1021; ii, 198
 COUPLAND, ii, 18, 778
 CREIGHTON, CHARLES, ii, 80
 CROCKER, RADCLIFFE, ii, 846, 874, *note*,
 953, 1008
 CROOKSHANK, i, 204, 397, 432
 CROSSE, i, 391, 401; ii, 600
 CRUVEILHIER (1874), i, 63, 519, 615, 668,
 708; ii, 344, 351
 CULLEN, of Edinburgh (1790), i, 12; ii, 330,
 333
 CURLING, THOS. B., i, 1013; ii, 353
 CURRIE, JAMES, of Liverpool (*ob.* 1805), i,
 151
 CURSCHMANN, i, 191, 192, 197, 1072; ii,
 101
 CYON, i, 694
 CZERMAK (*ob.* 1873), ii, 105
 CZERNY, ii, 695, 713

D.

DA COSTA, of Philadelphia, ii, 772, 205
 DARIER, ii, 846, *note*, 925
 DAVAINÉ, i, 416; ii, 460, 462
 DAVIES, HERBERT, i, 498
 DAVIES-COLLEY (*ob.* 1900), i, 420, 516
 DAVY, JOHN (1839), i, 33
 DÉJÉRINE, i, 684
 DELÉPINE, of Manchester, i, 132; ii, 476

DE MORGAN, i, 306, 314
 DEVERGIE, ii, 877, 946, 978, 980
 DICKINSON, W. H., i, 444, 535, 823; ii, 257, 336, 384, 595
 DIDAY (*ob.* 1894), i, 357
 DITTRICH, of Erlangen, i, 1072; ii, 31, 134
 DOLEŪS (1684), ii, 789
 DONKIN, H. B., i, 37
 DRESSLER, ii, 595
 DUBINI, of Milan, ii, 466
 DUCHENNE, of Boulogne (*ob.* 1875), i, 668, 675, 685, 690, 1041
 DUCKWORTH, Sir DYCE, i, 117, 475, 521, 1066; ii, 559, 560, 885, 929, 1003
 DUHRING, of Philadelphia, ii, 865, 888
 DUKES, CLEMENT, of Rugby, i, 186, 212; ii, 612
 DUMÉNIL, of Rouen, i, 593
 DURET, i, 720
 DURHAM, A. E. (*ob.* 1895), i, 543
 DURHAM, H. E. (son), i, 132, 303; ii, 379

E.

EBERTH, i, 138, 368, *note*
 EBSTEIN, i, 471; ii, 709, 720
 EDDISON, of Leeds, i, 1092
 EHRENBURG, i, 20, *note*
 EHRLICH, i, 118; ii, 6, *note*, 598, 752, 765
 EICHHORST, i, 127; ii, 751, 778
 EICHSTADT, ii, 940
 ELSÄSSER (1843), i, 534, 536
 ERB, of Heidelberg, i, 569, 579, 605, 608, 671, 685, 700, 703
 ESBACH, ii, 609
 ESMARCH and KULENKAMPE, ii, 965
 ESQUIROL, i, 926
 ESTLANDER, of Helsingfors, i, 1107
 EULENBURG, i, 555, 700, 704
 EWART, ii, 42, 43, 222
 EYRE, ii, 406, 745

F.

FAGGE, CHARLES HILTON (*ob.* 1883), i, 72, 76, 77, *note*, 85, 88-9, 128, *note*, 128, 134, *note*, 218, 228, 280, *note*, 309, 318, 326, 341, 363, *note*, 448, *note*, 453, 463, 488, 500, 689, 712, 724, 768, 770, *note*, 933, 1013, 1070; ii, 4, *note*, 38, *note*, 42, *note*, 132, 143, 158, 174, 181, 211, 212, 256, 318, 320, 322, 358, 371, 414, 483, 596, 705, 858, 961
 FAUVEL (1843), ii, 256, 791
 FAWCETT, i, 482; ii, 408
 FAYRER, Sir JOSEPH, i, 402, 940; ii, 401
 FEHLEISEN, i, 307
 FENWICK, BEDFORD (son), ii, 265
 FENWICK, SAMUEL, ii, 48, 316, 322, 506
 FERRIER, DAVID, i, 707, 779, 920
 FINNEY, of Baltimore, i, 155, *note*
 FINSER, of Copenhagen, i, 201; ii, 985
 FISHER, THEODORE, of Bristol, ii, 206, 226
 FITZ, of Boston, ii, 735
 FLEISCHER, ii, 633
 FLINT, AUSTIN, of Philadelphia, ii, 19, 252, 398
 FLÜGGE, i, 20; ii, 802

FOOT, of Dublin, ii, 926
 FORSTER, COOPER (*ob.* 1886), i, 404
 FOTHERGILL (1748), i, 315, 557
 FOURNIER, i, 338, 347, 701, 839; ii, 32
 FOWLER, J. K., i, 1101; ii, 53, 89
 FOX, COLCOTT, ii, 864, *note*, 902, 986
 FOX, E. LONG (*ob.* 1902), i, 889
 FOX, TILBURY (*ob.* 1879), ii, 858, 888, 902, 927, 937
 FOX, WILSON (*ob.* 1887), i, 367, 492, 505; ii, 14
 FOXWELL, ii, 264
 FRACASTORIUS (1530), i, 337, 350
 FRÄNKEL, of Breslau, i, 355, *note*; ii, 14, 764
 FRASER, THOMAS, of Edinburgh, ii, 273, 351
 FRAZIER, ii, 406, *note*
 FRENTZEL, i, 1093; ii, 205
 FRERICHS (*ob.* 1881), ii, 321, *note*, 328, 361, 546, 634
 FRIEDLÄNDER (1882), ii, 14
 FRIEDREICH, of Heidelberg, i, 670, 688, 689, 705
 FRITSCH and HITZIG, i, 779

G.

GAFFKY, i, 138
 GAIRDNER, Sir WILLIAM, of Glasgow, i, 1056, 1066; ii, 256, 994
 GALEN, CLAUDIUS (A.D. 200), i, 30, 73, 74, *note*, 84; ii, 505, *note*
 GALLOWAY, ii, 848
 GAMALEIA, ii, 15
 GAMGEE, A., ii, 574-5, 593
 GARCIA, MANUEL, ii, 106
 GARROD, Sir ALFRED, i, 469, 473, 524
 GARROD, ARCHIBALD E. (son), i, 495, 521, 538; ii, 576, 578
 GEE, SAMUEL J., i, 172, 206, 429, 533, 535, 653, 1026; ii, 116, 676
 GEISSLER (1861), ii, 5
 GENDRIN, i, 723, 958; ii, 8
 GERHARDT, ii, 110
 GIBERT, ii, 876
 GILBERT and FOURNIER, ii, 537
 GILCHRIST, of Baltimore, ii, 848, 860, 909
 GLISSON, FR. (*ob.* 1677), i, 530
 GODLEE, J. R., i, 1105, 1109
 GOLGI, of Pavia, i, 388
 GOLL, i, 660
 GOLTDAMMER (1876), i, 147
 GOLTZ, of Strassburg, i, 609
 GOOCH, i, 806
 GOODALL, E. W. (*see also* Washbourn), i, 122, 123, 177, 179, 404
 GOODALL, EDWIN, i, 844
 GOODEVE, i, 272, 274
 GOODHART, JAMES, i, 166, 185, 213, 217, 221, 491, 664, 831, 1109; ii, 17, 33, 207, 386, 720
 GOODSIR, of Edinburgh (*ob.* 1867), ii, 361
 GOWERS, Sir WILLIAM, i, 350, *note*, 608, 660, 677, 687, 870; ii, 31, 624, 750
 VON GRAEFE (*ob.* 1870), i, 585, 587, 771
 GRAVES, of Dublin (*ob.* 1853), i, 123, 227, 236, 592; ii, 544

GREEN, T. H., ii, 31
 GREENFIELD, ii, 31, 33, 638, 640, 785
 GREENHOW, E. H., i, 501, *note*, 503, 953;
 ii, 72, 73, 728
 GRIESINGER (*ob.* 1868), i, 252, 842; ii, 467,
 718
 GRUBER, MAX, of Vienna
 GRUBY, ii, 921, 943
 GUILLEMARD, ii, 719
 GULL, Sir WILLIAM (*ob.* 1890), i, 499, 611,
 616, 641, 788, 966, 971, 1016
 GUY, ii, 66

H.

DE HAEN, of Vienna (1777), i, 33, 386
 HAFKINE, i, 267, 289
 HAHN, ii, 722
 HAIGHT, of New York, i, 311; ii, 1017
 HALL, MARSHALL (*ob.* 1857), i, 221, 708,
 806
 HALLIBURTON, i, 829, 1018
 HAMBURGER, ii, 179, 311
 HAMILTON, of Aberdeen, i, 364, 1052; ii,
 39, 629
 HAMMOND, of New York, i, 635, 859
 HANKIN, i, 423
 HANNOVER, of Copenhagen, i, 93
 HANSEN, of Bergen, ii, 993
 HARDY, A., of St. Louis, ii, 980
 HARLEY, GEORGE, ii, 595
 HARLEY, JOHN, i, 117, 855; ii, 717
 HARLEY, VAUGHAN (son), ii, 795
 VAN HARLINGEN, ii, 907, 962, 986, *note*
 HARRIS, VINCENT, i, 368; ii, 43
 HASLUND, ii, 875
 HAUSHALTEN, i, 383
 HAUST, ii, 540
 HAVILAND, i, 100
 HAYEM, i, 629; ii, 750
 HAYGARTH, i, 519
 HEAD, HENRY, i, 554; ii, 1016, 1020
 HEBERDEN (1710-1801), i, 205, 332, 479,
 518, 735; ii, 173, 183, 682
 HEBRA, FERDINAND, of Vienna (*ob.* 1880),
 ii, 821, 860, 861, 866, 887, 890, 892, 919,
 953, 989, 1008
 HEBRA, HANS VON (son), ii, 860, 921, 960
 HEIBERG, of Copenhagen, i, 67
 HEIDENREICH, i, 257
 HEINTZE, ii, 125
 HEITLER, of Vienna, ii, 42
 HENDERSON, of Edinburgh, i, 252
 HENOCH, ii, 10, 116
 HÉRICOURT, i, 353
 HERTZ, of Amsterdam, i, 386
 HESSE, ii, 157
 HEUBNER, of Leipzig, i, 719, 720
 HEWITT, PRESCOTT, i, 835
 HICKS, J. BRAXTON, i, 171
 HILDENBRAND, of Vienna (*ob.* 1818), i, 12,
 158
 HILLIER, i, 888
 HILTON, ii, 426, 439
 HINDENBURG, ii, 801
 HIPPOCRATES, of Cos (*fl.* 400 B.C.), i, 231,
 251, 473, 758, 1113; ii, 362, *note*
 HIRSCH, i, 227

HITZIG, i, 581, 777 (*see also* Fritsch)
 HODGKIN, THOS. (*ob.* 1866), ii, 767
 HOME, of Edinburgh, i, 318
 HOOD, DONALD, ii, 406, 599
 HOPKINS, F. G., ii, 582, 779, 781
 HORSLEY, VICTOR, i, 621, 787, 1018
 HUETER, i, 307, 320; ii, 202
 HUGUENIN, i, 145, 837
 HUMPHRY, Sir GEORGE, i, 377; ii, 680
 HUNTER, WILLIAM, of Glasgow (*ob.* 1783),
 ii, 277, 336, 518
 HUNTER, WILLIAM (1901), ii, 515, 777,
 781
 HUSS, of Stockholm, ii, 18, 70
 HUTCHINSON, JONATHAN, i, 209, 273, 344,
 251, 358, 510, 580, 587, 732; ii, 300, 385,
 413, 863, 886, 906, 940, 944, 946, 953,
note, 971, 995, 1003, 1005, 1010
 HUXHAM (*ob.* 1768), i, 113
 HYDE, J. N., ii, 886
 HYDE and MONTGOMERY, ii, 907

I.

IMMERMAN, ii, 774, 793
 IRVINE, i, 127
 ISRAEL, JAMES, i, 431

J.

JACCOUD (1864), i, 881
 JACKSON, HUGHLINGS, i, 696, 730, 732, 920,
 936
 JACOBSON, of Königsberg, i, 48
 JADASSOHN, of Breslau, ii, 907
 JAFFÉ, ii, 575-577
 VON JAKSCH, i, 440
 JAMIESON, of Edinburgh, ii, 854, 890, *note*
 JARUNTOWSKI, ii, 86
 JENNER, EDWARD (1749-1823), i, 201
 JENNER, Sir WILLIAM (*ob.* 1893), i, 113,
 119, 236, 535, 1062; ii, 68, 120, 493
 JOHNSON, Sir GEORGE (*ob.* 1896), i, 280; ii,
 109, 611, 615
 JOLLY, i, 683, 956
 JONES, BENICE (*ob.* 1873), ii, 574, 606
 JONES, THOS. WHARTON, i, 46
 JÖRG, EDUARD (*ob.* 1878), i, 1055
 JÜRGENSEN, of Tübingen, i, 121, 151, 158,
 1056; ii, 10, 13, 19, 24, 25

K.

KANTHACK, of Cambridge (*ob.* 1898), i, 29,
note, 433; ii, 101, 736
 KAPOSI, of Vienna (*ob.* 1902), ii, 860, 887,
 921, 989, 1008
 KARSCH, ii, 946
 KELLY, ii, 76
 KING, WILKINSON (*ob.* 1847), ii, 144, 167
 KITISATO, i, 265, 374, 383
 KLEBS, of Breslau and Chicago, i, 320, 396;
 ii, 638
 KLEIN, E., i, 20, 29, *note*, 170, 321, 333;
 ii, 14, 638
 KLEMPERER, ii, 21
 KNOCH, ii, 455

KÖBNER, ii, 907, 921, 937
 KOCH, ROBERT, of Berlin (1882), i, 19, 282
 KOHLS, i, 1045
 KÖNIG, of Rostock, i, 307, 308
 KRISHABER, ii, 311
 KÜCHENMEISTER, ii, 450
 KÜHNE, of Heidelberg, i, 279; ii, 644
 KÜLZ, i, 498, 593
 KUSSMAUL, i, 448, 678, 682, 921; ii, 72, 151, 175, 227, 276, 329, 442

L.

LAACHE, of Christiania, ii, 206, 1004
 LAENNEC (1786-1821), i, 362, 1028, 1059, 1061; ii, 533
 LANCEREAUX, i, 336, 455; ii, 31, 290
 LANCISI (1716), i, 113, 385
 LANDERER, ii, 86
 LANDOUZY, i, 685; ii, 1007, 1020
 LANDREY, i, 637
 LANE, WM. A., i, 526, 533
 LANKESTER, E. R., i, 20, 397
 LATHAM, P. W., i, 902
 LATHAM, PETER MERE (1875), ii, 229
 LAVERAN, i, 396
 LEBERT, of Zurich and Breslau (*ob.* 1878), i, 273, 363, 768; ii, 774
 LEES, D. B., i, 539, 812; ii, 207, 277
 LEGG, WICKHAM, i, 293; ii, 213, 550, 803, 888
 LEGGATT, i, 293-3
 LEICHTENSTERN, ii, 417
 LÉPINE, ii, 776, *note*
 VON LEUBE, of Würzburg, i, 421, 710; ii, 361, 611, 696
 LEUCKART, ii, 449, 453, 470
 LEWIS, T. R., ii, 472
 VON LEYDEN, of Berlin, i, 31, 40, 594, 1072; ii, 101, 483
 LICHTHEIM, i, 1056; ii, 780
 VON LIEBERMEISTER, of Basel, i, 31, *note*, 40, 42, 121, 123, 127, 293
 VON LIMBECK, ii, 993
 LIND, ii, 789
 LISTER, Lord, i, 18, *note*, 21, 45, *note*, 65, *note*
 LITTEN, i, 259
 LIVEING, EDWARD, i, 902; ii, 101, 337
 LIVEING, ROBERT, ii, 860, 869, 901, 914, 919-999
 LÖFFLER, i, 320, 425
 LORETA, of Milan, ii, 364
 LOUIS (*ob.* 1872), i, 112, 362
 LUBBOCK, MONTAGU, i, 268, *note*
 LUCAS, R. C., i, 513
 LUDWIG, E., of Vienna, i, 332

M.

MACCARTHY, ii, 675
 MACCORMAC, of Belfast, ii, 65
 MACDONALD, GREVILLE, ii, 114, 122, *note*
 MACÉWEN, of Glasgow, i, 784, 787
 MACKENZIE, of Burnley, i, 556, *note*
 MACKENZIE, F. M., i, 273-5

MACKENZIE, HECTOR, i, 1019
 MACKENZIE, MOREL, ii, 113-115, 123, 129, 141, 301, 305
 MACKENZIE, STEPHEN, i, 393; ii, 798, 899, 968
 MACLAGAN, i, 501, 503
 MACLEAN, i, 386, 391, 939; ii, 392, 398, 399
 MACLEOD, ii, 864, *note*
 MACNAMARA, i, 270, 281
 MAGENDIE (*ob.* 1855), i, 407
 MAHMSTEN, ii, 921
 MALASSEZ, ii, 750
 MANSON, PATRICK, i, 300, 304, 396; ii, 396, 473, 475
 MARCET, ALEX. (1817), ii, 674
 MARIE, PIERRE, i, 547, 549, 707
 MARSON, i, 191, 193, 197, 200
 MARTIN, SIDNEY, i, 22, 321, 423; ii, 66, 333, 345
 MAYNEL, of Lyons, i, 489
 MENDELSSOHN, i, 1062
 MENIÈRE, i, 932
 MERCATUS, of Madrid (1614), i, 315
 VON MERING, of Halle, i, 455
 MERKEL, of Nürnberg, ii, 72
 MERYON, EDWARD, i, 685, 688
 METCHNIKOFF, i, 23, 44, 52, *note*, 364
 MEYER, of Copenhagen, ii, 305
 MEYERS, ii, 205
 MIBELLI, ii, 961, 1006
 MICHEL, of Cologne, ii, 305
 MILROY, i, 264
 MINKOWSKY, i, 455
 MITCHELL, WEIR, i, 569, 573, 861, 965
 MOLLENDORF, i, 903
 MOORE, Sir J. W., of Dublin, i, 127, 147; ii, 674
 MOORE, NORMAN, i, 337, *note*, 468, 540
 MOREAU, i, 279, 527
 MOREHEAD, i, 939; ii, 529
 MORGAGNI, J. B. (1682-1771), ii, 2, 217
 MORISON, ALEXANDER, ii, 75, 81, 171
 MORRIS, HENRY, ii, 690, 702
 MORRIS, MALCOLM, ii, 875, 931, 946, 968
 MORROW, ii, 906
 MORTON, R. (*ob.* 1698), i, 169, 191, 199, 385; ii, 49
 MOTT, i, 233, *note*, 571, 845; ii, 393
 MOXON (*ob.* 1886), i, 37, 78, 278, 622, 708, 738, 740; ii, 282, 288-9, 356, 382, 578, 611
 MUIR and REID, i, 29, *note*
 MUNRO, of Sydney, ii, 869
 MURCHISON (*ob.* 1879), i, 111, 117, 123, 127, 140, 233, 245, 254, 260; ii, 343, 361, 502
 MURPHY, SHIRLEY, i, 140
 MURRAY, of Newcastle, i, 1019
 MURRAY, MONTAGUE, ii, 454
 MURREL, ii, 192
 MUSSER, of Philadelphia, ii, 220, *note*, 554, 681, *note*, 734, 778

N.

NÄGELI, i, 19, 21, 22, 26
 NEELSEN, ii, 48
 NEISSER, ii, 1003
 NEUMANN, ii, 762, 868, 886, 939
 NEWSHOLME, i, 322; ii, 378, *note*

NICOLAÏER, i, 374
 NIELSEN, ii, 1014
 NIEMEYER, of Tübingen, i, 365; ii, 49
 NORMAN, C., ii, 394
 NORTH, i, 388, 396
 NOTHNAGEL, of Vienna, i, 712, 725, 783;
 ii, 188
 NOTTER, J. L., i, 303

O.

OBERMEIER (1873), i, 257
 O'DWYER, of New York, ii, 136
 OERTEL, i, 320, 327; ii, 274
 OERTMANN, i, 34
 OGATA, i, 597
 OGLE, WM., i, 591, 736, 742
 OGSTON, i, 20
 OLIVER, GEORGE, of Harrogate, ii, 731,
 751
 OLIVER, THOMAS, of Newcastle, i, 475
 OLLIVIER, AUGUSTE, ii, 217
 VON OPPOLZER, of Vienna (*ob.* 1871), ii, 15
 ORD, WM. M. (*ob.* 1902), i, 255, 1017, *note*;
 ii, 226, 579, 674
 ORMEROD, J. A., i, 707; ii, 257
 ORTH, of Bonn, i, 307
 OSLER, WILLIAM, of Toronto and Baltimore,
 i, 71, 397, 817; ii, 226, 235, 400, 901, *note*
 OSLER and MCCREA, ii, 358
 OWEN, ISAMBARD, i, 502, 539; ii, 69

P.

PAGET, Sir JAMES (*ob.* 1899), i, 546, 574; ii,
 46, 69, 79, 82, 468, 846
 PAGET, STEPHEN, i, 213
 PAKES, WALTER, i, 327
 PALTAEUF, of Vienna, ii, 741
 PANUM, of Copenhagen (*ob.* 1885), i, 66; ii,
 234
 PARÉ, AMBROSE (*ob.* 1590), i, 63
 PARKES, EDMUND (*ob.* 1876), i, 222, 243,
 294; ii, 6
 PARKINSON (1817), i, 875
 PARROT, i, 356, 539; ii, 278
 PARRY, CALEB, of Bath (*ob.* 1828), i, 928;
 ii, 744
 PASTEUR, i, 25, 65, 412, 423
 PATTERSON, of Leith (1840), i, 182
 PAUL, F. T., of Liverpool, i, 505, 506
 PAULUS, ÆGINETA, ii, 387, *note*
 PAVY, i, 442, 450, 462; ii, 344, 596, 612
 PAYNE, J. F., i, 78, 101, 268, *note*, 269; ii,
 744, 835, 865, 885, 888, 916-17, 939, 946,
 1007
 PEACOCK, T. B., i, 157, 488, 1079; ii, 199,
 205, 234, 275, 288, 294, 423
 PEKELHARING, of Utrecht, i, 304, 597
 PEREZ, i, 152
 PERNET, ii, 885
 PERRY, ii, 225, 962, 1004
 PERRY and SHAW, ii, 353, 356, 381, 492
 PETRESCO, of Bucharest, ii, 20
 VON PETTENKOFFER (*ob.* 1901), i, 27, *note*
 PFEIFFER, i, 132, 229; ii, 741, 1020
 PFLÜGER, i, 31
 PIORRY, i, 1021

PITCAIRN, ALEX., i, 487
 PITT, G. N., i, 707, 710, 719, 790; ii, 21,
 234, 265, 402, 408, 535, 735
 PLENCK, of Vienna, ii, 813
 PLIMMER, i, 97
 PLUMBE, ii, 899
 POLAND (*ob.* 1872), i, 374
 POLLITZER, ii, 967, 1014
 POLLOCK, JAMES, ii, 58, 59
 POORE, i, 860-64, 1076; ii, 86
 PORTER, W. H., of New York, ii, 32
 POTT, PERCIVAL (*ob.* 1788), i, 68
 POWELL, Sir R. DOUGLAS, i, 1111; ii, 50, 234
 POWER, W. H., i, 170, 321
 PRALL, S., i, 277
 PRICE, of Reading, ii, 535
 PRINGLE, J. J., ii, 925, 945, 962, 1004, 1006,
 1009
 PROUT (*ob.* 1850), i, 448; ii, 576, 681, 715
 PURSER, of Dublin, ii, 939, 940

Q.

QUAIN, Sir RICHARD (*ob.* 1898), ii, 210,
 216
 QUINCKE, of Bern, i, 114; ii, 749, 755, 774

R.

RAKE, BEAVEN, of Trinidad, ii, 412, 463
 RAMAZZINI, ii, 70, 369
 RANSOM, W. H., ii, 382, 463
 RANSOME, ii, 65, 77, 79
 RANVIER, *see* CORNIL
 RASMUSSEN, of Copenhagen, ii, 50
 RAYER, ii, 602
 RAYMOND, PAUL, ii, 902
 RAYNAUD (*ob.* 1881), i, 1039; ii, 600
 REES, GEO. OWEN (*ob.* 1889), i, 498; ii, 591,
 602, 608, 610
 REMAK, ii, 4
 REYNOLDS, Sir RUSSELL (*ob.* 1896), i, 217,
 311, 399, 760, 923, 956
 RHASES (*fl.* 850), i, 160
 RICORD (*ob.* 1889), i, 340, *note*
 RIEGEL, ii, 110
 RILLIER and BARTHEZ (1855), i, 662, 827
 RINDFLEISCH, i, 192, 316, 366
 RINGER, i, 40, 163, 389, 492
 VON RITTERSHAIN, i, 534
 ROBERTS, Sir WILLIAM (*ob.* 1899), i, 471,
 580, 668, 670; ii, 334, 579, 609, 685
 ROBERTSON, ARGYLL, of Edinburgh, i, 695
 ROBSON, MAYO, ii, 364, 523
 VON ROKITANSKY, of Vienna (*ob.* 1878), i,
 12, 767; ii, 344, 533, 644
 ROLLESTON, G. H., ii, 258, *note*, 732, 736
 ROMBERG (*ob.* 1873), i, 579, 690, 698
 ROSE, of Zürich, i, 375
 ROSENTHAL, i, 31
 ROSS, Surgeon-Major, i, 388
 ROSS, JAMES, of Manchester, i, 687, 689,
 701
 ROSTAN, i, 762
 ROUX, i, 321, 383
 RUFFER, i, 97
 RÜHLE, i, 56, 57
 RUSSELL, RISIEN, i, 871, 896

S.

- SABOURAUD, ii, 931, 939, 944
 SALKOWSKI and LEUBE (1882), ii, 576
 SALTER, HYDE, ii, 98, 102, 799
 SALTER, S. J. A. (Bro.), i, 558
 SAMWAYS, ii, 259, *note*, 268, 270
 SANARELLI, of Monte Video, i, 296
 SANDERSON, JOHN BURDON, i, 17, 20, 31, 46, 282, 367; ii, 168
 SANDWITH, i, 252; ii, 467
 SANGSTER, ii, 902, 922, 1002, 1006
 SANSOM, ii, 257, 258, *note*
 SAUNDBY, of Birmingham, ii, 346, 540
 SAUVAGES (*ob.* 1767), i, 231
 SAVAGE, GEO., i, 653, 840, 978-1011
 SCHEUBE, i, 596
 SCHLESINGER, i, 632
 SCHLIEP, ii, 329
 SCHÖNLEIN (*ob.* 1864), ii, 901, 939
 SCHRÖTTER, of Vienna, ii, 131, *note*
 SCHULEIN (1875), i, 36
 SCHULZEN, i, 481
 SCHUNCK, ii, 576
 SCHÜPPEL (1871), i, 364
 SCHWIMMER, of Buda Pesth, ii, 955, *note*
 SCUDAMORE (1816), i, 467, 473
 SEATON, i, 159, 204, 366
 SÉE, GERMAIN (*ob.* 1889), i, 889, 892
 SEITZ, ii, 53, 203
 SEMMOLA, of Naples, ii, 610
 SEMON, Sir FELIX, ii, 111, 441
 SENATOR, of Berlin, i, 130, *note*, 147, 332, *note*, 526, 543
 SEQUEIRA, ii, 225, *note*
 SHARKEY, i, 190; ii, 207
 SHATTOCK, ii, 674
 SHATTOCK and BALLANCE, i, 97, 100
 SHATTUCK, of Boston, i, 112
 SHATTUCK, F. C. (son), of Boston, i, 120, 302; ii, 363, *note*, 740
 SHAW, LAURISTON, i, 504, 683; ii, 350
 SHERRINGTON, i, 425
 SHOEMAKER, ii, 934
 SIBSON, FR., i, 488
 SIMON, of Hamburg, i, 190
 SIMON, GUSTAV (1848), ii, 953, 1012
 SIMON, Sir JOHN, i, 48, 204, 323
 SKODA, of Vienna (*ob.* 1881), i, 1023, *note*, 1026; ii, 18, 36, 226
 SMITH, ANDREW, of New York, i, 635
 SMITH, EUSTACE, i, 167, 217, 221, 540; ii, 90, 380, 798
 SMITH, FR., of Sheffield, i, 865; ii, 371, 550
 SMITH, JOHNSON, ii, 795
 SMITH, LORRAIN, ii, 749
 SMITH, WALTER G., of Dublin, ii, 879, 946, 1004
 SNOW, JOHN, i, 284
 SPENDER, of Bath, i, 522; ii, 375, *note*
 STARR, ALLAN, of New York, i, 665
 STEDMAN, i, 300
 STEFFEN, J. T. A., ii, 23
 STERNMAN, ii, 14
 STEVENSON, THOS., ii, 370, 781
 STEWART, D. D., of Philadelphia, ii, 369
 STEWART, Sir T. GRAINGER, of Edinburgh (*ob.* 1900), i, 592, 1076; ii, 611, 612, 622, 650
 VOL. II.

- STILL, GEO., i, 524, 813
 STILLING, i, 644
 STOCKER, RICHARD (*ob.* 1878), i, 237, 243, 806
 STOKES, of Dublin (*ob.* 1878), i, 1043
 STONE, i, 1038
 STÖRK, ii, 303
 STOWERS, i, 207
 STRICKER, i, 501
 VON STRÜMPPEL, of Erlangen, i, 712, 714
 STURGES, i, 887
 STURGES and COUPLAND, ii, 13
 SUTTON, C. R. A., ii, 624
 SUTTON, H. G., i, 500; ii, 29
 SUTTON, J. BLAND, i, 101, 114, 540; ii, 80
 SYDENHAM, THOMAS (*ob.* 1689), i, 160, 169, 466, 518, 880
 SYMONDS, C. J., ii, 105, *note*

T.

- TALAMON, ii, 14
 TAPPEINER, i, 369
 TATHAM, ii, 81
 TAYLOR, FREDK., i, 383, 631, 1038, 1084; ii, 263, 537, 765, 766, 774, 931
 TEALE, of Leeds, i, 37
 THAYER, of Baltimore, i, 549
 THEYER, i, 118
 THIERFELDER, ii, 547-9
 THIN, ii, 384, 801, 926, 930, 944
 THOMA, of Magdeburg-Sudenburg, i, 46; ii, 750
 THOMAS, of Leipzig, i, 162
 THOMSON, STCLAIR, i, 829
 THORBURN, of Manchester, i, 549
 THORINGTON, i, 298
 THORNE, Sir RICHARD (*ob.* 1900), i, 322
 TILESIIUS, ii, 1000
 TODD, R. B. (*ob.* 1860), i, 691, 732, 959; ii, 533
 TOOTH, i, 660, 685
 TOPINARD, i, 696
 TÖRÖK, ii, 1006
 TRAUBE, of Berlin (*ob.* 1876), i, 31, 33, 1095; ii, 56, 71, 175, 256
 TREVES, ii, 416, 1000
 TROUSSEAU (*ob.* 1867), i, 182, 208, 389, 480, 555, 682, 736, 737, 748, 803, 867, 1112; ii, 697
 TULP, NICHOLAS (1652), i, 1078
 TÜRCK, of Vienna, i, 659; ii, 105
 TURNER, F. C. (*ob.* 1890), i, 665; ii, 210, 257
 TURNER, GEORGE, i, 116, 143

U.

- UNNA, of Hamburg, ii, 837, 861, 864, 872, 890, 917, 939, 989

V.

- VALLEIX, i, 555
 VELPEAU, i, 92
 VILLEMIN, i, 366
 VIRCHOW, RUDOLF, of Berlin, i, 63, 75, 83, 84, 316, 363, 766, 835, 1012; ii, 71, 136, 390, 663, 758, 784, 993, 1000

VOGEL, JULIUS, of Giessen, ii, 297
 VOISIN, i, 922
 VOIT, i, 635
 VOLKMANN, of Halle, i, 307, 310, 664, 984
 VOLTOLINI, of Vienna, i, 813; ii, 103
 VULPIAN (1877), i, 701

W.

WALDEYER, of Strassburg, i, 76, 95; ii, 355
 WALKER, of Peterborough, ii, 514
 WALSHE (*ob.* 1892), i, 1068; ii, 54, 185
 WARD, ii, 401
 WARE, JOHN, of Boston, i, 942
 WARREN, of Boston, ii, 925, *note*
 WASHBOURN, i, 1100; ii, 27
 WASHBOURN and GOODALL, i, 146, 171, 174, 220, 247, 323, 328
 WASSERMANN, i, 383; ii, 111, 885
 WATSON, of Baltimore, i, 547
 WATSON, Sir THOS. (*ob.* 1882), i, 173, 217, 379
 WEBER, Sir HERMANN, i, 502; ii, 64
 WEICHSELBAUM, of Vienna, i, 820; ii, 14
 WEIL, ii, 511
 WELCH, of Baltimore, ii, 346, 358
 WERLHOF, ii, 798
 WERNICKE, i, 738
 WERTHEIM, ii, 919
 WEST, CHARLES, i, 664
 WEST, SAMUEL, i, 1079, 1109, 1111; ii, 230, 766
 WESTPHAL, i, 608, 695
 WHIPHAM, i, 116, *note*
 WHITE, of Boston, ii, 925, 989
 WHITE, WM. HALE, i, 32, *note*, 449, 768, 783, 788; ii, 385, 730, 778
 WIDAL, i, 132
 WIDERHOFER, ii, 144
 WILKS, Sir SAMUEL, i, 67-8, 79, 174, 371, 683, 719, 753, 933; ii, 16, 31, 151, 211, 267, 355, 543, 602-3, 724, 767, 774, 927

WILLAN, ROBERT, ii, 821, 857-8, 881, 892, 897, 964, 975
 WILLIAMS, C. J. B. (*ob.* 1889), i, 25, 45, *note*; ii, 7, 58, 79, 100
 WILLIAMS, CHAS. THEODORE (son), ii, 43, 67
 WILLIAMS, DAWSON, i, 161, 183, 367, 423; ii, 378, 742
 WILLIS, THOMAS (*ob.* 1675), i, 436
 WILSON, Sir ERASMUS (*ob.* 1884), ii, 861, 877, 887, 925, 1002, 1008
 VON WINCKEL, of Munich, ii, 595
 WINTERNITZ, i, 31, *note*
 WOILLEZ, ii, 22
 WOLLASTON, ii, 584
 WOOD, CARTWRIGHT, i, 333
 WOOD, H. C., of Philadelphia, i, 32
 WOODALL, ii, 794
 WOODHEAD, G. S., i, 333, 374, 414
 WOODWARD, ii, 389
 WOOLDRIDGE, L. C. (*ob.* 1889), i, 66, 423
 WRIGHT, A. E., of Netley, i, 139, 156, 303, 459; ii, 88, *note*, 290, 752, 803
 WUCHERER, ii, 467-472
 WUNDERLICH, of Leipzig, i, 33, 37, 197, 387

Y.

YERSIN, i, 265, 320
 YOUATT, i, 403, 407, 410

Z.

ZAMBACO, of Constantinople, i, 633
 ZANCAROL, of Alexandria, ii, 717
 ZENKER, of Erlangen, i, 137; ii, 72, 470
 ZIEGLER, of Freiburg-i.-B., i, 364
 VON ZIEMSEN, of Munich (*ob.* 1901), i, 816, 881; ii, 23, 124, 135, 345
 ZOPF, i, 19, 20
 ZÜLZER, i, 189, 310

